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KENNETH M. HARGREAVES | LOUIS H. BERMAN

COHEN'S PATHWAYS of the PULP

ELEVENTH EDITION

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COHEN'S PATHWAYS *of the* PULP

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ELEVENTH EDITION

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He has served in leadership roles for various dental organizations, including chair of the International Federation of Endodontic Associations' Research Committee, member on committees of the American Association of Endodontists, European Society of Endodontology, and as scientific reviewer for international endodontic and dental journals. He has also served as president of the Southern California Academy of Endodontists, Israel Endodontic Society, International Association for Dental Research—Israel Division, and chair of the Israel National Board of Diplomates in Endodontics.

Dr. Rotstein has published more than 150 scientific papers and research abstracts in the dental literature as well as chapters in international endodontic textbooks, including *Pathways of the Pulp*, *Ingle's Endodontics*, *Endodontics: Principles and Practice*, *Seltzer and Bender's Dental Pulp*, and *Harty's Endodontics in Clinical Practice*. He has lectured extensively in more than 25 countries throughout 5 continents.

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Stephen Cohen
MA, DDS, FICD, FACD

The field of endodontics would be difficult to imagine without *Pathways of the Pulp*. In speaking with colleagues across North America and around the world, it becomes clear that *Pathways* has had an immense, ubiquitous, and persistent impact on endodontics. This enduring contribution to our specialty is due to the genius of Stephen Cohen, who, together with Richard Burns, developed the most distinguished and perpetually updated evidenced-based textbook in our specialty. Their insight was to form a collaboration of the most renowned experts in our field, with expansion of the authorships for each new edition, and with an unwavering emphasis on the art and science of contemporary endodontic therapy. The result was a textbook that is both comprehensive and nuanced, which has transcended 11 editions and 14 languages since 1976. As each edition of *Pathways* evolved, it changed with the times, updating from unquestionable dogma into what was later considered the novel state of the art. Each edition progressed through the decades of endodontics and was inclusive of the next generation of technologies, philosophies, materials, devices, and instruments. As a result, with Steve as the lead editor since its inception, *Pathways of the Pulp* is considered the most comprehensive and innovative endodontic textbook available, literally defining the field of endodontics.

Stephen is an active educator, having lectured for decades around the world and serving as a Clinical Professor of Endodontics at the Arthur A. Dugoni School of Dentistry of the University of the Pacific. His passion for teaching, coupled with his distinctive authoritative voice and his vast scientific and clinical expertise, generates a highly effective combination for educating students on every facet of the endodontic specialty. His steadfast commitment in his authoring and editing of *Pathways of the Pulp* has propelled this textbook into what it is today.

In short, Dr. Stephen Cohen is a renaissance man, being both a practitioner and a teacher, whose breadth of expertise is leveraged by a passionate focus on detail and clarity. Defined by his unquestionable ethics and pursuit of perfection, Stephen's philosophy of learning, teaching, and practicing endodontics can best be summed up in his own words, as he penned in the Introduction of his last edition of *Pathways*:

"As clinicians we must meet this rich convergence of discovery and invention with an equally rich commitment to continuous learning, exposing ourselves to all the science our field has to offer. This is our duty to our founders, this is our responsibility to our patients, and this is our gift to ourselves."

Steve is a pioneer who has transformed the field of endodontics. For the tenth edition of this textbook, we recognized his legacy by renaming this textbook *Cohen's Pathways of the Pulp*. We reinforce our esteem appreciation of him by dedicating this eleventh edition to our mentor and friend, Dr. Stephen Cohen.

Kenneth M. Hargreaves and
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New to This Edition

EIGHT NEW CHAPTERS

Chapter 2: Radiographic Interpretation covers imaging modalities, diagnostic tasks in endodontics, three-dimensional imaging, cone beam computed tomography, intraoperative or postoperative assessment of endodontic treatment complications, and more!

Chapter 4: Pain Control looks at two overarching topics: local anesthesia for restorative dentistry and endodontics and analgesics and therapeutic recommendations.

Chapter 11: Evaluation of Outcomes covers the reasons for evaluating treatment outcomes, outcome measurements for endodontic treatment, the outcomes of vital pulp therapy procedures, nonsurgical root canal treatment, nonsurgical retreatment, and surgical retreatment.

Chapter 16: Root Resorption looks at the histological features of root resorption, external inflammatory resorption, external cervical resorption, and internal resorption.

Chapter 19: Managing Iatrogenic Endodontic Events looks at treatment scenarios for eight different iatrogenic events: cervicofacial subcutaneous emphysema, sodium hypochlorite accidents, perforations (nonsurgical), inferior alveolar nerve injury (surgical), sinus perforation, instrument separation, apical extrusion of obturation materials, and ledge formation.

Chapter 21: Cracks and Fractures looks at three categories of cracks and fractures: cracked and fractured cusps, cracked and split teeth, and vertical root fractures, emphasizing the early diagnosis of these conditions.

Chapter 23: Vital Pulp Therapy addresses the living pulp, pulpal response to caries, procedures for generating reparative dentin, indications and materials for vital pulp therapy, MTA applications, treatment recommendations, and more!

Chapter 27: Bleaching Procedures provides a review of internal and external bleaching procedures, their impact on pulpal health/endodontic treatment, with presentations of cases and clinical protocols.

NEW CHAPTER ORGANIZATION

Chapters have been reorganized and grouped into three parts: Part I: *The Core Science of Endodontics*, Part II: *The Advanced Science of Endodontics*, and Part III: *Expanded Clinical Topics*. The seven chapters in Part 1 focus on the core clinical concepts for dental students; the chapters in Parts II and III provide the information that advanced students and endodontic residents and clinicians need to know. In addition, seven additional chapters are included in the online version.

The new organization better reflects the chronology of endodontic treatment.

EXPERT CONSULT

New features included on the Expert Consult site include:

- Seven chapters exclusively online:
  - *Chapter 24: Pediatric Endodontics: Endodontic Treatment for the Primary and Young Dentition*
  - *Chapter 25: Endodontic and Periodontic Interrelationships*
  - *Chapter 26: Effects of Age and Systemic Health on Endodontics*
  - *Chapter 27: Bleaching Procedures*
  - *Chapter 28: Understanding and Managing the Fearful Dental Patient*
  - *Chapter 29: Endodontic Records and Legal Responsibilities*
  - *Chapter 30: Key Principles of Endodontic Practice Management*

- Twelve lecture modules consisting of assigned readings, PowerPoint slides, written objectives for each lecture, and suggested examination questions. Topics covered include:
  - Diagnosis
  - Treatment planning
  - Pain control
  - Isolation
  - Cleaning and shaping
  - Obturation
  - Surgery
  - Assessment of outcomes
  - Pulp biology
  - Pathobiology
  - Emergencies
  - Restoration

- New videos and animations

Introduction

ENDODONTICS: A VIEW OF THE FUTURE

The Editors have had the privilege of "standing on the shoulders" of our generous contributors, enabling us to "look over the horizon" to gain a glimpse at our endodontic future. As we advance into the years ahead, we will incorporate even more refined and accurate improvements in pulpal diagnosis, canal cleaning and disinfection, canal obturation, and surgical enhancements.

In looking more clearly toward our impending endeavors, it becomes important to scrutinize the deficiencies of our past and present. Over the past several decades we have gone from arsenic to sodium hypochlorite, from bird droppings to gutta percha, from hand files to motor-driven files, from culturing to one-visit appointments, from two-dimensional to three-dimensional radiography, and from pulp removal to pulpal regeneration. And still, the clinical and academic controversies are pervasive. So, where will the future of our specialty take us?

With patients living longer and with the inescapable comparison of endodontics to endosseous implants, the demand for endodontic excellence has greatly increased. To that end, we suspect that future evidence-based approaches will continue to question the longevity of successful implant retention, intensifying the need for more predictable endodontic outcomes.

Surprisingly, we still base our diagnosis on a presumed and almost subjective pulpal status. Imagine a future in which endodontic diagnosis could be more objective by non-invasively scanning the pulp tissue. Imagine algorithms built into all digital radiography for interpreting and extrapolating disease processes. CBCT has made a huge impact on endodontic diagnosis, but can we enhance these digital captures with a resolution that would approach micro-computed tomography, and with less radiation? Will non-radiation imaging methods such as MRI (magnetic resonance imaging) leave the dental research clinic to provide a novel solution to address these issues? Will it be CT technology or some other form of detection for dramatically enhancing our guidance during surgical and nonsurgical treatment in order to both maximize our precision and minimize tooth structure and associated tissue removal? Considering the differences in color and consistency of the tissues within the pulp chamber, future technology may permit us to better discriminate these differences and enhance our ability for more precision when negotiating the openings to these canals. And as for clinical visualization: will there be digital or electronic enhancements of conventional loupes? Will 3-D visualization and monitor-based observation change the way we visualize and implement our procedures? During our canal cleaning and shaping, we are lucky if we can debride half of the pulpal tissues within all of the canal ramifications; however, we still use an irrigant that is so toxic by a non-selective mechanism, such that when inadvertently extruded beyond the canal system it can cause severe tissue damage. Our future technology should guide us to obtain the complete removal of organic debris within the pulpal spaces while obtaining complete canal disinfection—and without the potential morbidity from toxic non-selective chemicals. We still use files that can inadvertently separate. The resolution may be in a complete transformation in metallurgy or even the implementation of other non-metal cutting materials. Our obturation material is one of the worst filling materials in dentistry. Hopefully, the future evolution of obturation will lead us to a totally leakage-free, non-neurotoxic, and biocompatible substance that will three-dimensionally expand into *all* microscopic canal ramifications and stop when there is no more space to expand to, being limited to when it reaches the periodontal ligament. Will this obturating material be newly regenerated vital pulp?

Clearly, it is evident that our endodontic future lies in out-of-the-box thinking with the next generation of transformations coming with collaborations not just from within the biological sciences, but rather in conjunction with physicists, chemists, engineers, and a multitude of other great innovative minds. The predictability of endodontics must be incontestable, not just with better technology to guide us toward greater success, but also to better elucidate exactly when endodontics *cannot* be successful. Our future needs to focus on predictability, which will only be achieved by reinventing the wheel with disruptive technologies, rather than persisting with variations and modifications of our current convictions.

As a specialty, we have advanced by leaps and bounds since our inception, but we are still in our infancy with a brilliant future ahead of us. Since 1976 and with 11 editions, *Pathways of the Pulp* has always been about the art and science of endodontics. The dedicated contributing authors have generously given their time to meticulously describe what is considered the state of the art of our specialty. We are hopeful that future editions will guide us toward enhanced endodontic outcomes, with the never-ending pursuit of endodontic excellence.

Contents

PART I: THE CORE SCIENCE OF ENDODONTICS, 1

1 **Diagnosis,** *2*
Louis H. Berman and Ilan Rotstein

2 **Radiographic Interpretation,** *33*
Madhu K. Nair, Martin D. Levin, and Umadevi P. Nair

3 **Case Selection and Treatment Planning,** *71*
Paul A. Rosenberg and Matthew Malek

4 **Pain Control,** *90*
Al Reader, John Nusstein, and Asma Khan

5 **Tooth Morphology, Isolation, and Access,** *130*
James L. Gutmann and Bing Fan

6 **Cleaning and Shaping the Root Canal System,** *209*
Ove A. Peters, Christine I. Peters, and Bettina Basrani

7 **Obturation of the Cleaned and Shaped Root Canal System,** *280*
William Johnson, James C. Kulild, and Franklin Tay

PART II: THE ADVANCED SCIENCE OF ENDODONTICS, 323

8 **Nonsurgical Retreatment,** *324*
Robert S. Roda and Bradley H. Gettleman

9 **Periradicular Surgery,** *387*
Bradford R. Johnson and Mohamed I. Fayad

10 **Regenerative Endodontics,** *447*
Anibal Diogenes, Stéphane Simon, and Alan S. Law

11 **Evaluation of Outcomes,** *474*
Yuan-Ling Ng and Kishor Gulabivala

12 **Structure and Functions of the Dentin-Pulp Complex,** *532*
Inge Fristad and Ellen Berggreen

13 **Pulpal Reactions to Caries and Dental Procedures,** *573*
Ashraf Fouad and Linda G. Levin

14 **Microbiology of Endodontic Infections,** *599*
José F. Siqueira, Jr. and Isabela N. Rôças

15 **Pathobiology of Apical Periodontitis,** *630*
Louis M. Lin and George T.-J. Huang

16 **Root Resorption,** *660*
Shanon Patel, Conor Durack, and Domenico Ricucci

17 **Diagnosis of Nonodontogenic Toothache,** *684*
Donna Mattscheck, Alan S. Law, and Donald R. Nixdorf

18 **Management of Endodontic Emergencies,** *706*
Samuel O. Dorn and Gary Shun-Pan Cheung

19 **Managing Iatrogenic Endodontic Events,** *722*
Yoshitsugu Terauchi

PART III: EXPANDED CLINICAL TOPICS, 757

20 **The Role of Endodontics After Dental Traumatic Injuries,** *758*
Martin Trope, Frederic Barnett, Asgeir Sigurdsson, and Noah Chivian

21 **Cracks and Fractures,** *793*
Zvi Metzger, Louis H. Berman, and Aviad Tamse

22 **Restoration of the Endodontically Treated Tooth,** *818*
Didier Dietschi, Serge Bouillaguet, and Avishai Sadan

23 **Vital Pulp Therapy,** *849*
George Bogen, Sergio Kuttler, and Nicholas Chandler

EXPERT CONSULT CHAPTERS

24 **Pediatric Endodontics: Endodontic Treatment for the Primary and Young Permanent Dentition,** *e1*
Paula J. Waterhouse and John M. Whitworth

25 **Endodontic and Periodontal Interrelationships,** *e45*
David G. Kerns and Gerald N. Glickman

26 **Effects of Age and Systemic Health on Endodontics,** *e62*
Carl W. Newton and Jeffrey M. Coil

27 **Bleaching Procedures,** *e96*
Frank Setzer

28 **Understanding and Managing the Fearful Dental Patient,** *e114*
Henrietta L. Logan and Ellen B. Byrne

29 **Endodontic Records and Legal Responsibilities,** *e124*
Edwin J. Zinman

30 **Key Principles of Endodontic Practice Management,** *e191*
Roger P. Levin

Index, *877*

COHEN'S PATHWAYS *of the* PULP

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PART I

The Core Science of Endodontics

CHAPTER 1
Diagnosis

CHAPTER 2
Radiographic Interpretation

CHAPTER 3
Case Selection and Treatment Planning

CHAPTER 4
Pain Control

CHAPTER 5
Tooth Morphology, Isolation, and Access

CHAPTER 6
Cleaning and Shaping the Root Canal System

CHAPTER 7
Obturation of the Cleaned and Shaped Root Canal System

CHAPTER 1

Diagnosis

LOUIS H. BERMAN | ILAN ROTSTEIN

CHAPTER OUTLINE

Art and Science of Diagnosis
Chief Complaint
Medical History
Dental History
Examination and Testing
Extraoral Examination
Intraoral Examination
Pulp Tests
Special Tests
Radiographic Examination and Interpretation
Cracks and Fractures
Perforations
Clinical Classification of Pulpal and Periapical Diseases
Pulpal Disease
Apical (Periapical) Disease
Referred Pain
Summary

ART AND SCIENCE OF DIAGNOSIS

Diagnosis is the art and science of detecting and distinguishing deviations from health and the cause and nature thereof.[6] The purpose of a diagnosis is to determine what problem the patient is having and why the patient is having that problem. Ultimately, this will directly relate to what treatment, if any, will be necessary. No appropriate treatment recommendation can be made until all of the *whys* are answered. Therefore, careful data gathering as well as a planned, methodical, and systematic approach to this investigatory process is crucial.

Gathering objective data and obtaining subjective findings are not enough to formulate an accurate clinical diagnosis. The data must be interpreted and processed to determine what information is significant, and what information might be questionable. The facts need to be collected with an active dialogue between the clinician and the patient, with the clinician asking the right questions and carefully interpreting the answers. In essence, the process of determining the existence of an oral pathosis is the culmination of the art and science of making an accurate diagnosis.

The process of making a diagnosis can be divided into five stages:

1. The patient tells the clinician the reasons for seeking advice.
2. The clinician questions the patient about the symptoms and history that led to the visit.
3. The clinician performs objective clinical tests.
4. The clinician correlates the objective findings with the subjective details and creates a tentative list of differential diagnoses.
5. The clinician formulates a definitive diagnosis.

This information is accumulated by means of an organized and systematic approach that requires considerable clinical judgment. The clinician must be able to approach the problem by crafting what questions to ask the patient and how to ask these pertinent questions. Careful listening is paramount to begin painting the picture that details the patient's complaint. These subjective findings combined with results of diagnostic tests provide the critical information needed to establish the diagnosis.

Neither the art nor the science is effective alone. Establishing a differential diagnosis in endodontics requires a unique blend of knowledge, skills, and ability to interpret and interact with a patient in real time. Questioning, listening, testing, interpreting, and finally answering the ultimate question of *why* will lead to an accurate diagnosis and in turn result in a more successful treatment plan.

Chief Complaint

On arrival for a dental consultation, the patient should complete a thorough registration that includes information pertaining to medical and dental history (Figs. 1-1 and 1-2). This should be signed and dated by the patient, as well as initialed by the clinician as verification that all of the submitted information has been reviewed (see Chapter 29 for more information).

The reasons patients give for consulting with a clinician are often as important as the diagnostic tests performed. Their remarks serve as initial important clues that will help the clinician to formulate a correct diagnosis. Without these direct and unbiased comments, objective findings may lead to an incorrect diagnosis. The clinician may find a dental pathosis, but it may not contribute to the pathologic condition that mediates the patient's chief complaint. Investigating these complaints may indicate that the patient's concerns are related to a medical condition or to recent dental treatment. Certain patients may

TELL US ABOUT YOUR SYMPTOMS

LAST NAME _____ **FIRST NAME** _____

1. Are you experiencing any pain at this time? If not, please go to question 6. Yes _____ No _____
2. If yes, can you locate the tooth that is causing the pain? Yes _____ No _____
3. When did you first notice the symptoms? _____
4. Did your symptoms occur suddenly or gradually? _____
5. Please check the frequency and quality of the discomfort, and the number that most closely reflects the intensity of your pain:

LEVEL OF INTENSITY (On a scale of 1 to 10) 1 = Mild 10 = Severe	FREQUENCY	QUALITY
1___2___3___4___5___6___7___8___9___10___	_____ Constant	_____ Sharp
	_____ Intermittent	_____ Dull
	_____ Momentary	_____ Throbbing
	_____ Occasional	

Is there anything you can do to relieve the pain? Yes _____ No _____

If yes, what? _____

Is there anything you can do to cause the pain to increase? Yes _____ No _____

If yes, what? _____

When eating or drinking, is your tooth sensitive to: Heat _____ Cold _____ Sweets _____

Does your tooth hurt when you bite down or chew? Yes _____ No _____

Does it hurt if you press the gum tissue around this tooth? Yes _____ No _____

Does a change in posture (lying down or bending over) cause your tooth to hurt? Yes _____ No _____

6. Do you grind or clench your teeth? Yes _____ No _____
7. If yes, do you wear a night guard? Yes _____ No _____
8. Has a restoration (filling or crown) been placed on this tooth recently? Yes _____ No _____
9. Prior to this appointment, has root canal therapy been initiated on this tooth? Yes _____ No _____
10. Is there anything else we should know about your teeth, gums, or sinuses that would assist us in our diagnosis? _____

Signed: Patient or Parent _____ **Date** _____

FIG. 1-1 Dental history form that also allows the patient to record pain experience in an organized and descriptive manner.

TELL US ABOUT YOUR HEALTH

LAST NAME _____ **FIRST NAME** _____

How would you rate your health? **Please circle one.** **Excellent Good Fair Poor**

When did you have your last physical exam? _____

If you are under the care of a physician, please give reason(s) for treatment.

_____

Physician's Name, Address, and Telephone Number:

Name _____ **Address** _____

City _____ **State** _____ **Zip** _____ **Telephone** _____

Have you ever had any kind of surgery? **Yes** _____ **No** _____

If yes, what kind? _____ **Date** _____

_____ **Date** _____

Have you ever had any trouble with prolonged bleeding after surgery? **Yes** _____ **No** _____

Do you wear a pacemaker or any other kind of prosthetic device? **Yes** _____ **No** _____

Are you taking any kind of medication or drugs at this time? **Yes** _____ **No** _____

If yes, please give name(s) of the medicine(s) and reason(s) for taking them:

Name _____ **Reason** _____

_____ _____

Have you ever had an unusual reaction to an anesthetic or drug (like penicillin)? **Yes** _____ **No** _____

If yes, please explain: _____

Please circle any past or present illness you have had:

Alcoholism	Blood pressure	Epilepsy	Hepatitis	Kidney or liver	Rheumatic fever
Allergies	Cancer	Glaucoma	Herpes	Mental	Sinusitis
Anemia	Diabetes	Head/Neck injuries	Immunodeficiency	Migraine	Ulcers
Asthma	Drug dependency	Heart disease	Infectious diseases	Respiratory	Venereal disease

Are you allergic to Latex or any other substances or materials? **Yes** _____ **No** _____

If so, please explain _____

If female, are you pregnant? **Yes** _____ **No** _____

Is there any other information that should be known about your health? _____

_____

Signed: Patient or Parent _____ **Date:** _____

FIG. 1-2 Succinct, comprehensive medical history form designed to provide insight into systemic conditions that could produce or affect the patient's symptoms, mandate alterations in treatment modality, or change the treatment plan.

even receive initial emergency treatment for pulpal or periapical symptoms in a general hospital.[93] On occasion, the chief complaint is simply that another clinician correctly or incorrectly advised the patient that he or she had a dental problem, with the patient not necessarily having any symptoms or any objective pathosis. Therefore, the clinician must pay close attention to the actual expressed complaint, determine the chronology of events that led to this complaint, and question the patient about other pertinent issues, including medical and dental history. For future reference and in order to ascertain a correct diagnosis, the patient's chief complaint should be properly documented, using *the patient's own words*.

Medical History

The clinician is responsible for taking a proper medical history from every patient who presents for treatment. Numerous examples of medical history forms are available from a variety of sources, or clinicians may choose to customize their own forms. After the form is completed by the patient, or by the parent or guardian in the case of a minor, the clinician should review the responses with the patient, parent, or guardian and then initial the medical history form to indicate that this review has been done. The patient "of record" should be questioned at each treatment visit to determine whether there have been any changes in the patient's medical history or medications. A more thorough and complete update of the patient's medical history should be taken if the patient has not been seen for over a year.[51,52]

Baseline blood pressure and pulse should be recorded for the patient at each treatment visit. Elevation in blood pressure or a rapid pulse rate may indicate an anxious patient who may require a stress reduction protocol, or it may indicate that the patient has hypertension or other cardiovascular health problems. Referral to a physician or medical facility may be indicated. It is imperative that vital signs be gathered at each treatment visit for any patient with a history of major medical problems. The temperature of patients presenting with subjective fever or any signs or symptoms of a dental infection should be taken.[57,80,105]

The clinician should evaluate a patient's response to the health questionnaire from two perspectives: (1) those medical conditions and current medications that will necessitate altering the manner in which dental care will be provided and (2) those medical conditions that may have oral manifestations or mimic dental pathosis.

Patients with serious medical conditions may require either a modification in the manner in which the dental care will be delivered or a modification in the dental treatment plan (Box 1-1). In addition, the clinician should be aware if the patient has any drug allergies or interactions, allergies to dental products, an artificial joint prosthesis, organ transplants, or is taking medications that may negatively interact with common local anesthetics, analgesics, sedatives, and antibiotics.[80] This may seem overwhelming, but it emphasizes the importance of obtaining a thorough and accurate medical history while considering the various medical conditions and dental treatment modifications that may be necessary before dental treatment is provided.

Several medical conditions have oral manifestations, which must be carefully considered when attempting to arrive at an accurate dental diagnosis. Many of the oral soft tissue changes that occur are more related to the medications used to treat the medical condition rather than to the condition itself. More common examples of medication side effects are stomatitis, xerostomia, petechiae, ecchymoses, lichenoid mucosal lesions, and bleeding of the oral soft tissues.[80]

When developing a dental diagnosis, a clinician must also be aware that some medical conditions can have clinical presentations that mimic oral pathologic lesions.[13,28,32,74,80,102,107,133] For example, tuberculosis involvement of the cervical and submandibular lymph nodes can lead to a misdiagnosis of lymph node enlargement secondary to an odontogenic infection. Lymphomas can involve these same lymph nodes.[80] Immunocompromised patients and patients with uncontrolled diabetes mellitus respond poorly to dental treatment and may exhibit recurring abscesses in the oral cavity that must be differentiated from abscesses of dental origin.[43,76,80,83] Patients with iron deficiency anemia, pernicious anemia, and leukemia frequently exhibit paresthesia of the oral soft tissues. This finding may complicate making a diagnosis when other dental pathosis is present in the same area of the oral cavity. Sickle cell anemia has the complicating factor of bone pain, which mimics odontogenic pain, and loss of trabecular bone pattern on radiographs, which can be confused with radiographic lesions of endodontic origin. Multiple myeloma can result in unexplained mobility of teeth. Radiation therapy to the head and neck region can result in increased sensitivity of the teeth and osteoradionecrosis.[80] Trigeminal neuralgia, referred pain from cardiac angina, and multiple sclerosis can also mimic dental pain (see also Chapter 17). Acute maxillary sinusitis is a common condition that may create diagnostic confusion because it may mimic tooth pain in the maxillary posterior quadrant. In this situation the teeth in the quadrant may be extremely sensitive to cold and percussion, thus mimicking the signs and symptoms of pulpitis. This is certainly not a complete list of all the medical entities that can mimic dental disease, but it should alert the clinician that a medical problem could confuse and complicate

BOX 1-1

Medical Conditions That Warrant Modification of Dental Care or Treatment

Cardiovascular: High- and moderate-risk categories of endocarditis, pathologic heart murmurs, hypertension, unstable angina pectoris, recent myocardial infarction, cardiac arrhythmias, poorly managed congestive heart failure[57,80,105]

Pulmonary: Chronic obstructive pulmonary disease, asthma, tuberculosis[80,129]

Gastrointestinal and renal: End-stage renal disease; hemodialysis; viral hepatitis (types B, C, D, and E); alcoholic liver disease; peptic ulcer disease; inflammatory bowel disease; pseudomembranous colitis[25,34,48,80]

Hematologic: Sexually transmitted diseases, HIV and AIDS, diabetes mellitus, adrenal insufficiency, hyperthyroidism and hypothyroidism, pregnancy, bleeding disorders, cancer and leukemia, osteoarthritis and rheumatoid arthritis, systemic lupus erythematosus[35,43,76,80,83,88,100,135]

Neurologic: Cerebrovascular accident, seizure disorders, anxiety, depression and bipolar disorders, presence or history of drug or alcohol abuse, Alzheimer disease, schizophrenia, eating disorders, neuralgias, multiple sclerosis, Parkinson disease[36,44,80]

the diagnosis of dental pathosis; this issue is discussed in more detail in subsequent chapters.

If, at the completion of a thorough dental examination, the subjective, objective, clinical testing and radiographic findings do not result in a diagnosis with an obvious dental origin, then the clinician must consider that an existing medical problem could be the true source of the pathosis. In such instances, a consultation with the patient's physician is always appropriate.

Dental History

The chronology of events that lead up to the chief complaint is recorded as the *dental history*. This information will help guide the clinician as to which diagnostic tests are to be performed. The history should include any past and present symptoms, as well as any procedures or trauma that might have evoked the chief complaint. Proper documentation is imperative. It may be helpful to use a premade form to record the pertinent information obtained during the dental history interview and diagnostic examination. Often a SOAP format is used, with the history and findings documented under the categories of *S*ubjective, *O*bjective, *A*ppraisal, and *P*lan. There are also built-in features within some practice management software packages that allow digital entries into the patient's electronic file for the diagnostic workup (Figs. 1-3 and 1-4).

History of Present Dental Problem

The dialogue between the patient and the clinician should encompass all of the details pertinent to the events that led to the chief complaint. The clinician should direct the conversation in a manner that produces a clear and concise narrative that chronologically depicts all of the necessary information about the patient's symptoms and the development of these symptoms. To help elucidate this information, the patient is first instructed to fill out a dental history form as a part of the patient's office registration. This information will help the clinician decide which approach to use when asking the patient questions. The interview first determines *what is going on* in an effort to determine *why is it going on* for the purpose of eventually determining *what is necessary to resolve the chief complaint*.

Dental History Interview

After starting the interview and determining the nature of the chief complaint, the clinician continues the conversation by documenting the sequence of events that initiated the request for an evaluation. The dental history is divided into five basic directions of questioning: localization, commencement, intensity, provocation and attenuation, and duration.

Localization. "Can you point to the offending tooth?" Often the patient can point to or tap the offending tooth. This is the most fortunate scenario for the clinician because it helps direct the interview toward the events that might have caused any particular pathosis in this tooth. In addition, localization allows subsequent diagnostic tests to focus more on this particular tooth. When the symptoms are not well localized, the diagnosis is a greater challenge.

Commencement. "When did the symptoms first occur?" A patient who is having symptoms often remembers when these symptoms started. Sometimes the patient will even remember the initiating event: it may have been spontaneous in nature; it may have begun after a dental visit for a restoration; trauma may be the etiology, biting on a hard object may have initially produced the symptoms, or the initiating event may have occurred concurrently with other symptoms (sinusitis, headache, chest pain, etc.). However, the clinician should resist the tendency to make a premature diagnosis based on these circumstances. The clinician should not simply assume "guilt by association" but instead should use this information to enhance the overall diagnostic process.

Intensity. "How intense is the pain?" It often helps to quantify how much pain the patient is actually having. The clinician might ask, "On a scale from 1 to 10, with 10 the most severe, how would you rate your symptoms?" Hypothetically, a patient could present with "an uncomfortable sensitivity to cold" or "an annoying pain when chewing" but might rate this "pain" only as a 2 or a 3. These symptoms certainly contrast with the type of symptoms that prevent a patient from sleeping at night. Often the intensity can be subjectively measured by what is necessary for the diminution of pain—for example, acetaminophen versus a narcotic pain reliever. This intensity level may affect the decision to treat or not to treat with endodontic therapy. Pain is now considered a standard vital sign, and documenting pain intensity (scale of 0 to 10) provides a baseline for comparison after treatment.

Provocation and attenuation. "What produces or reduces the symptoms?" Mastication and locally applied temperature changes account for the majority of initiating factors that cause dental pain. The patient may relate that drinking something cold causes the pain or possibly that chewing or biting is the only stimulus that "makes it hurt." The patient might say that the pain is only reproduced on "release from biting." On occasion, a patient may present to the dental office with a cold drink in hand and state that the symptoms can only be *reduced* by bathing the tooth in cold water. Nonprescription pain relievers may relieve some symptoms, whereas narcotic medication may be required to reduce others (see Chapter 4 for more information). Note that patients who are using narcotic as well as non-narcotic (e.g., ibuprofen) analgesics may respond differently to questions and diagnostic tests, thereby altering the validity of diagnostic results. Thus, it is important to know what drugs patients have taken in the previous 4 to 6 hours. These provoking and relieving factors may help the clinician to determine which diagnostic tests should be performed to establish a more objective diagnosis.

Duration. "Do the symptoms subside shortly, or do they linger after they are provoked?" The difference between a cold sensitivity that subsides in a few seconds and one that subsides in minutes may determine whether a clinician repairs a defective restoration or provides endodontic treatment. The duration of symptoms after a stimulating event should be recorded to establish how long the patient felt the sensation in terms of seconds or minutes. Clinicians often first test control teeth (possibly including a contralateral "normal" tooth) to define a "normal" response for the patient; thus, "lingering" pain is apparent when comparing the duration between the control teeth and the suspected tooth.

With the dental history interview complete, the clinician has a better understanding of the patient's chief complaint and can concentrate on making an objective diagnostic evaluation,

Name: (Last) _____ (First) _____ **Date:** _____ **Tooth:** _____

S. (SUBJECTIVE)
Chief Complaint:
History of Present Illness:

Nature of Pain: None Mild Moderate Severe
Quality: Dull Sharp Throbbing Constant
Onset: Stim Required Intermittent Spontaneous
Location: Localized Diffuse Referred Radiating to:
Duration: Seconds Minutes Hours Constant
Initiated by: Cold Heat Sweet Spontaneous Palpation Mastication Supination Keeps awake at night
Relieved by: Cold Heat OTC-Meds Narc-Meds

O. (OBJECTIVE)

Extraoral:
Facial swelling: Yes No
L Nodes swollen: Yes No

Intraoral:
Soft tissues: WNL
Swelling: Yes No Mild Moderate Severe Location:
Sinus tract: Yes No Closed
Clinical crown: Restn Caries Exposure Fracture

#	Cold	Heat	EPT	Perc	Palp	Mob	Bite Stick	Dis-color	Periodontal Exam								
									MB	B	DB	DL	L	ML	Recessn	Furcation	Bleed - Probing

(Normal: N No Response: 0 Mild: + Moderate: ++ Severe: +++ Lingered: L Delayed: D)

Radiographic Findings:
Alveolar Bone: WNL Apical lucency Lateral lucency Ap / Lat opacity Crestal bone loss
Lamina Dura: WNL Obscure Broken Widened
Roots: WNL Curvature Resorption Perforation Dilaceration Fracture Long Sinus / IAN
Pulp Chamber: WNL Calcification Pulp Stone Exposure Resorption Perforation
Pulp Canal: WNL Calcification Bifurcated Resorption Prior RCT Furcation Involvement Perforation
Crown: WNL Caries Restoration Crown Dens in dente
Sinus Tract: Traces to:

A. (Assessment)
Diagnosis: Pulpal: WNL Rev Pulpitis Irrev Pulpitis Necrosis Prior RCT / Non-healing Pulpless
Periapical: WNL APP CPP APA CPA Cond Osteitis
Etiology: Caries Restoration Prior RCT Iatrogenic Coronal leakage Trauma Perio Elective Resorptn VRF
Prognosis: Good Fair Poor

P. (PLAN)
Endodontic: Caries control RCT ReTx I&D Apico Apexification/genesis Perf / Resorption Repair

Periodontal: S/RP Crown lengthen Root amp Hemisection Extraction

Restorative: Temp Post space B/U P&C Onlay / Crown Bleach

FIG. 1-3 When taking a dental history and *performing* a diagnostic examination, often a premade form can facilitate complete and accurate documentation. (Courtesy Dr. Ravi Koka, San Francisco, CA.)

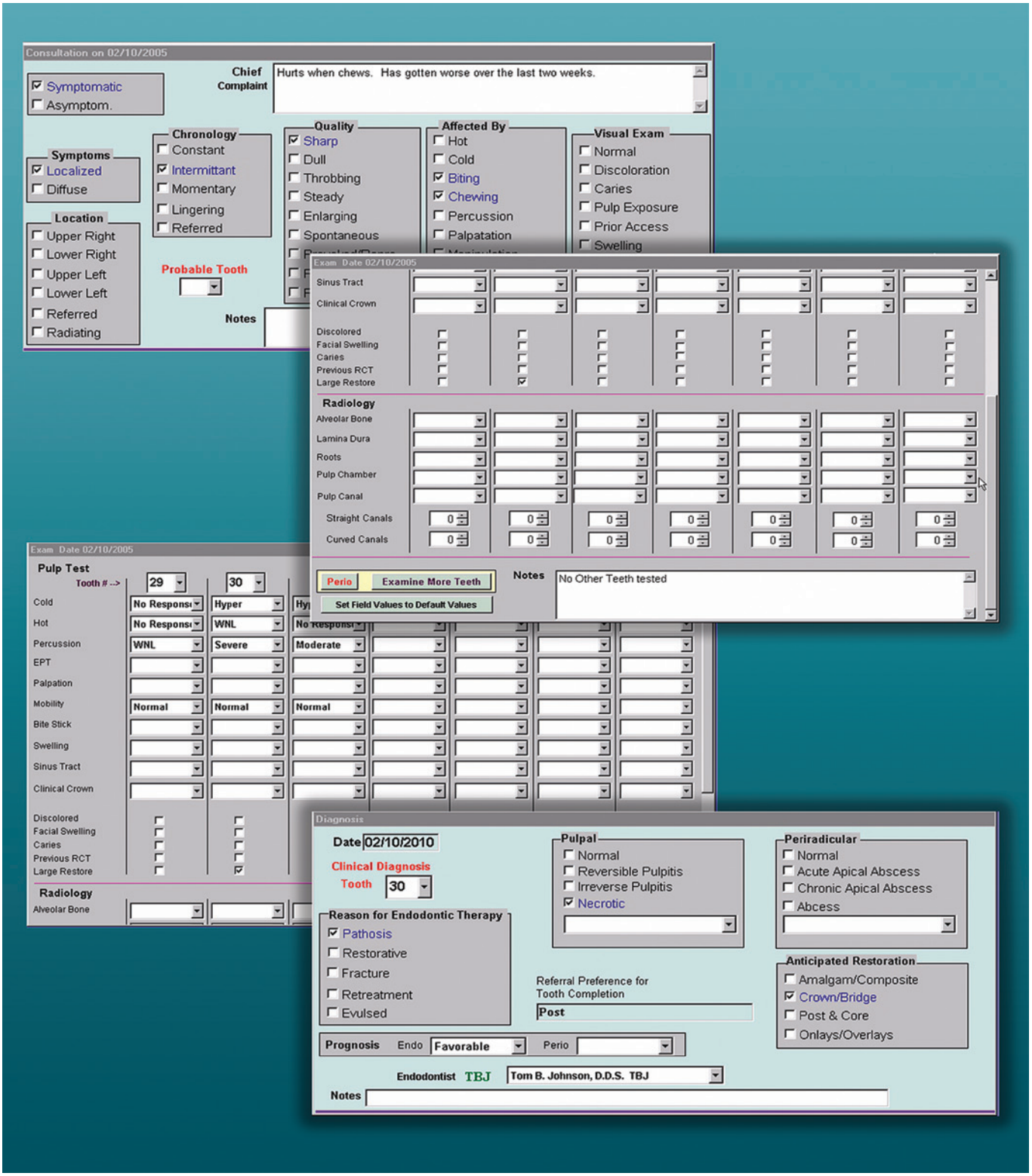


FIG. 1-4 Several practice management software packages have features for charting endodontic diagnoses using user-defined drop-down menus and areas for specific notations. Note that for legal purposes, it is desirable that all recorded documentation have the ability to be locked, or if any modifications are made after 24 hours, the transaction should be recorded with an automated time/date stamp. This is necessary so that the data cannot be fraudulently manipulated. (Courtesy PBS Endo, Cedar Park, TX.)

FIG. 1-5 A, Canine space swelling of the left side of the face extending into and involving the left eye. B, Swelling of the upper lip and the loss of definition of the nasolabial fold on the patient's left side, which indicates an early canine space infection.

although the subjective (and artistic) phase of making a diagnosis is not yet complete and will continue after the more objective testing and scientific phase of the investigatory process.

EXAMINATION AND TESTING

Extraoral Examination

Basic diagnostic protocol suggests that a clinician observe patients as they enter the operatory. Signs of physical limitations may be present, as well as signs of facial asymmetry that result from facial swelling. Visual and palpation examinations of the face and neck are warranted to determine whether swelling is present. Many times a facial swelling can be determined only by palpation when a unilateral "lump or bump" is present. The presence of bilateral swellings may be a normal finding for any given patient; however, it may also be a sign of a systemic disease or the consequence of a developmental event. Palpation allows the clinician to determine whether the swelling is localized or diffuse, firm or fluctuant. These latter findings will play a significant role in determining the appropriate treatment.

Palpation of the cervical and submandibular lymph nodes is an integral part of the examination protocol. If the nodes are found to be firm and tender along with facial swelling and an elevated temperature, there is a high probability that an infection is present. The disease process has moved from a localized area immediately adjacent to the offending tooth to a more widespread systemic involvement.

Extraoral facial swelling of odontogenic origin typically is the result of endodontic etiology because diffuse facial swelling resulting from a periodontal abscess is rare. Swellings of nonodontogenic origin must always be considered in the differential diagnosis, especially if an obvious dental pathosis is not found.[77] This situation is discussed in subsequent chapters.

FIG. 1-6 Buccal space swelling associated with an acute periradicular abscess from the mandibular left second molar.

A subtle visual change such as loss of definition of the nasolabial fold on one side of the nose may be the earliest sign of a canine space infection (Fig. 1-5). Pulpal necrosis and periradicular disease associated with a maxillary canine should be suspected as the source of the problem. Extremely long maxillary central incisors may also be associated with a canine space infection, but most extraoral swellings associated with the maxillary centrals express themselves as a swelling of the upper lip and base of the nose.

If the buccal space becomes involved, the swelling will be extraoral in the area of the posterior cheek (Fig. 1-6). These swellings are generally associated with infections originating from the buccal root apices of the maxillary premolar and molar teeth and the mandibular premolar (Fig. 1-7) and first molar teeth. The mandibular second and third molars may also be involved, but infections associated with these two teeth are just as likely to exit to the lingual where other spaces

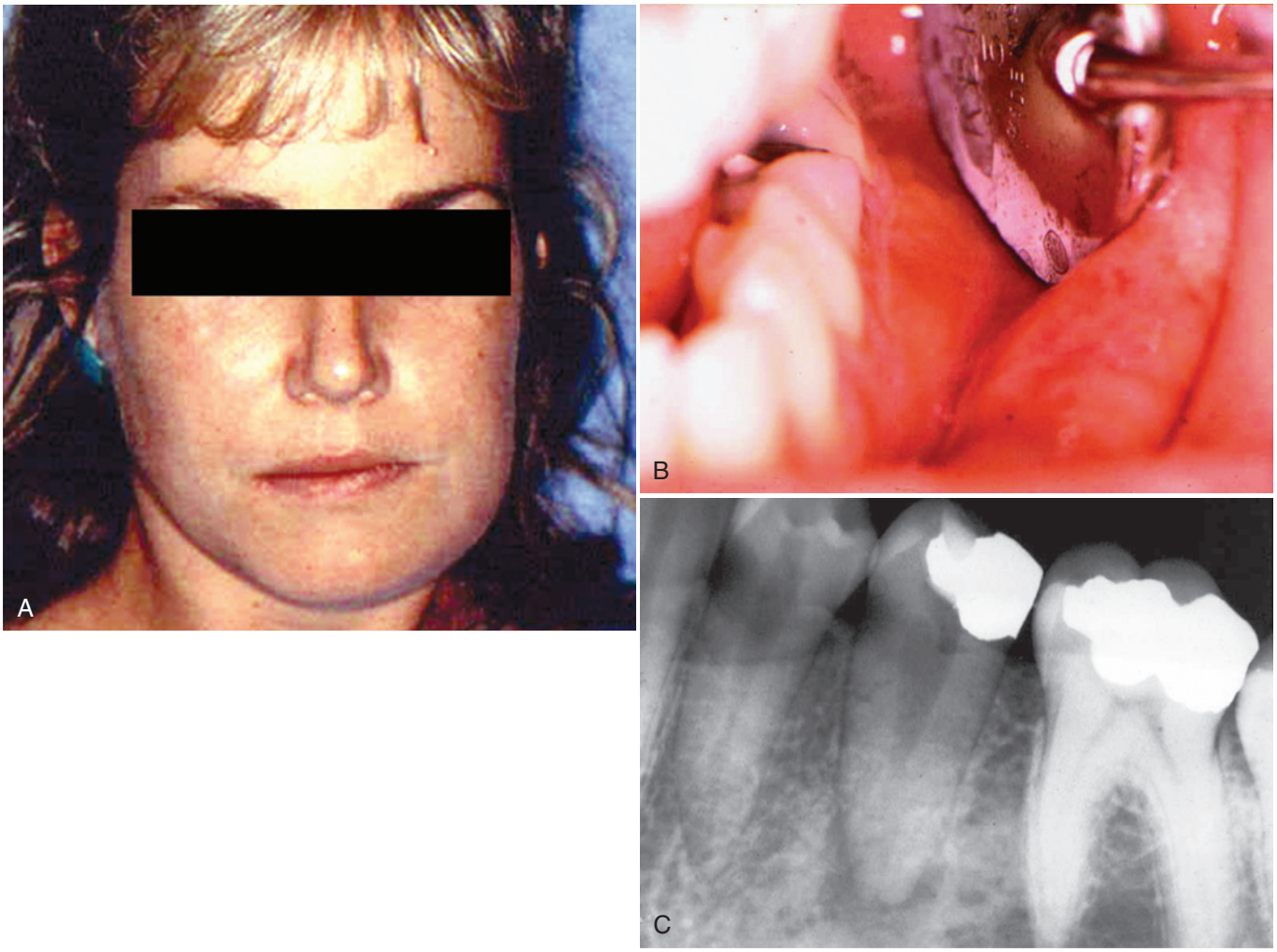


FIG. 1-7 A, Buccal space swelling of the left side of the patient's face. Note the asymmetry of the left side of the face. B, Intraoral view shows swelling present in the left posterior mucobuccal fold. C, This buccal space infection was associated with periradicular disease from the mandibular left second premolar. Note on the radiograph the periradicular radiolucency and large restoration associated with this tooth.

would be involved. For infections associated with these teeth, the root apices of the maxillary teeth must lie superior to the attachment of the buccinator muscle to the maxilla, and the apices of the mandibular teeth must be inferior to the buccinator muscle attachment to the mandible.[77]

Extraoral swelling associated with mandibular incisors will generally exhibit itself in the submental (Fig. 1-8) or submandibular space. Infections associated with any mandibular teeth, which exit the alveolar bone on the lingual and are inferior to the mylohyoid muscle attachment, will be noted as swelling in the submandibular space. Further discussions of fascial space infections may be found in Chapter 14.

Sinus tracts of odontogenic origin may also open through the skin of the face (Figs. 1-9 and 1-10).[2,56,64] These openings in the skin will generally close once the offending tooth is treated and healing occurs. A scar is more likely to be visible on the skin surface in the area of the sinus tract stoma than on the oral mucosal tissues (Fig. 1-10, *C* and *D*). Many patients with extraoral sinus tracts give a history of being treated by general physicians, dermatologists, or plastic surgeons with systemic or topical antibiotics or surgical procedures in

FIG. 1-8 Swelling of the submental space associated with periradicular disease from the mandibular incisors.

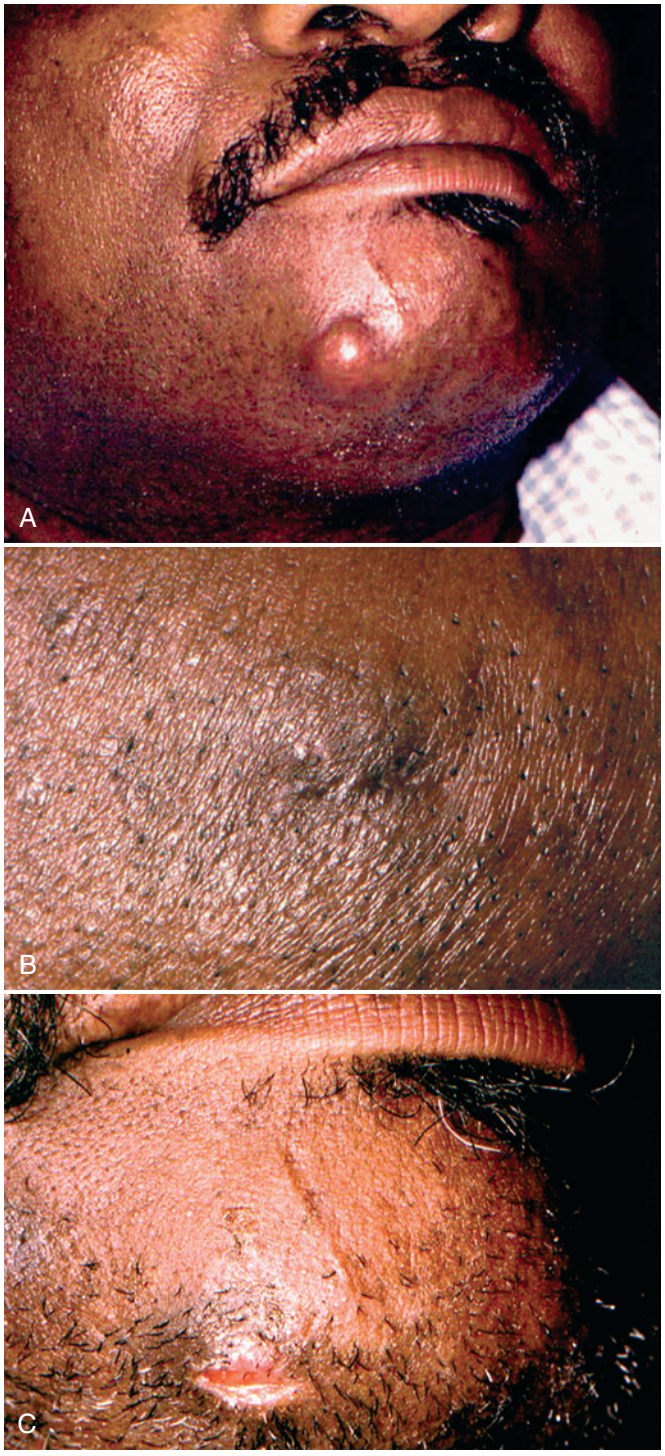


FIG. 1-9 A, Extraoral drainage associated with periradicular disease from the mandibular right canine. Note the parulis on the right anterior side of the face. B, Initial scar associated with the extraoral drainage incision after the parulis was drained and root canal therapy performed on the canine. C, Three-month follow-up shows healing of the incision area. Note the slight inversion of the scar tissue.

attempts to heal the extraoral stoma. In these particular cases, after multiple treatment failures, the patients may finally be referred to a dental clinician to determine whether there is a dental cause. Raising the awareness of physicians to such cases will aid in more accurate diagnosis and faster referral to the dentist or endodontist.

Intraoral Examination

The intraoral examination may give the clinician insight as to which intraoral areas may need a more focused evaluation. Any abnormality should be carefully examined for either prevention or early treatment of associated pathosis.[4,30,75,113,110,126] Swelling, localized lymphadenopathy, or a sinus tract should provoke a more detailed assessment of related and proximal intraoral structures.

Soft Tissue Examination

As with any dental examination, there should be a routine evaluation of the intraoral soft tissues. The gingiva and mucosa should be dried with either a low-pressure air syringe or a 2-by-2-inch gauze pad. By retracting the tongue and cheek, all of the soft tissue should be examined for abnormalities in color or texture. Any raised lesions or ulcerations should be documented and, when necessary, evaluated with a biopsy or referral.[82]

Intraoral Swelling

Intraoral swellings should be visualized and palpated to determine whether they are diffuse or localized and whether they are firm or fluctuant. These swellings may be present in the attached gingiva, alveolar mucosa, mucobuccal fold, palate, or sublingual tissues. Other testing methods are required to determine whether the origin is endodontic, periodontic, or a combination of these two or whether it is of nonodontogenic origin.

Swelling in the anterior part of the palate (Fig. 1-11) is most frequently associated with an infection present at the apex of the maxillary lateral incisor or the palatal root of the maxillary first premolar. More than 50% of the maxillary lateral incisor root apices deviate in the distal or palatal directions. A swelling in the posterior palate (Fig. 1-12) is most likely associated with the palatal root of one of the maxillary molars.[77]

Intraoral swelling present in the mucobuccal fold (Fig. 1-13) can result from an infection associated with the apex of the root of any maxillary tooth that exits the alveolar bone on the facial aspect and is inferior to the muscle attachment present in that area of the maxilla (see also Chapter 14). The same is true with the mandibular teeth if the root apices are superior to the level of the muscle attachments and the infection exits the bone on the facial. Intraoral swelling can also occur in the sublingual space if the infection from the root apex spreads to the lingual and exits the alveolar bone superior to the attachment for the mylohyoid muscle. The tongue will be elevated and the swelling will be bilateral because the sublingual space is contiguous with no midline separation. If the infection exits the alveolar bone to the lingual with mandibular molars and is inferior to the attachment of the mylohyoid muscle, the swelling will be noted in the submandibular space. Severe infections involving the maxillary and mandibular molars can extend into the parapharyngeal space, resulting in intraoral swelling of the tonsillar and pharyngeal areas. This can be life threatening if the patient's airway becomes obstructed.[77,80]

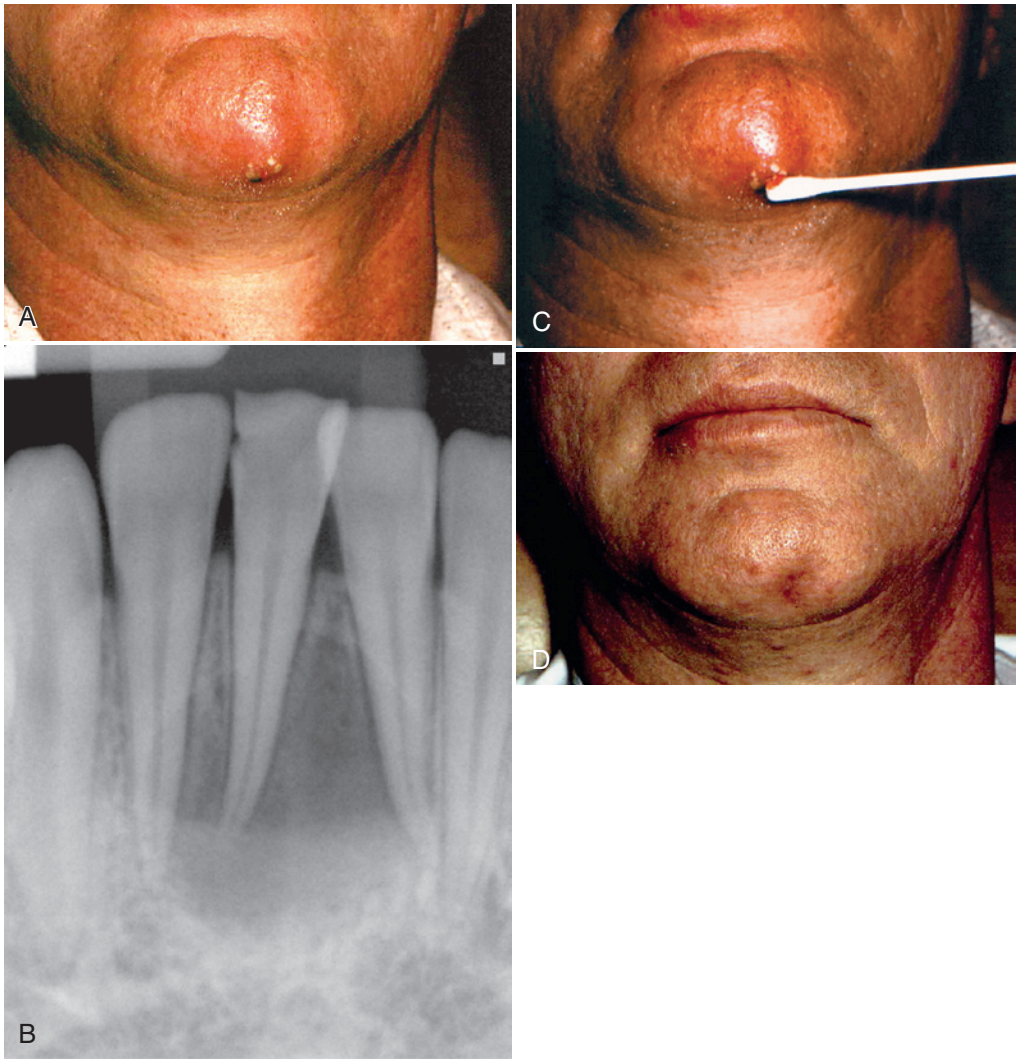


FIG. 1-10 A, Extraoral sinus tract opening onto the skin in the central chin area. B, Radiograph showing large radiolucency associated with the mandibular incisors. C, A culture is obtained from the drainage of the extraoral sinus tract. D, The healed opening of the extraoral sinus tract 1 month after root canal therapy was completed. Note the slight skin concavity in the area of the healed sinus tract.

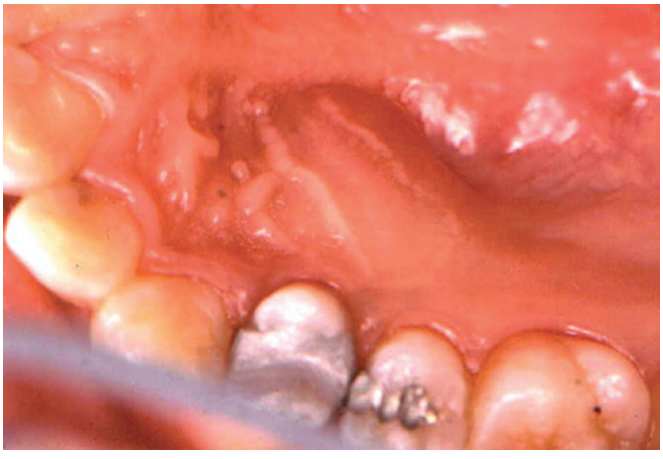


FIG. 1-11 Fluctuant swelling in the anterior palate associated with periradicular disease from the palatal root of the maxillary first premolar.

Intraoral Sinus Tracts

On occasion, a chronic endodontic infection will drain through an intraoral communication to the gingival surface and is known as a *sinus tract*.[12] This pathway, which is sometimes lined with epithelium, extends directly from the source of the infection to a surface opening, or *stoma,* on the attached gingival surface. As previously described, it can also extend extraorally. The term *fistula* is often inappropriately used to describe this type of drainage. The fistula, by definition, is actually an abnormal communication pathway between two internal organs or from one epithelium-lined surface to another epithelium-lined surface.[6]

Histologic studies have found that most sinus tracts are not lined with epithelium throughout their entire length. One study found that only 1 out of the 10 sinus tracts examined were lined with epithelium, whereas the other nine specimens were lined with granulation tissue.[55] Another study, with a larger sample size, found that two thirds of the specimens did not have epithelium extending beyond the level of the surface mucosa rete ridges.[12] The remaining specimens had some

FIG. 1-12 Fluctuant swelling in the posterior palate associated with periradicular disease from the palatal root of the maxillary first molar.

FIG. 1-13 Fluctuant swelling in the mucobuccal fold associated with periradicular disease from the maxillary central incisor.

epithelium that extended from the oral mucosa surface to the periradicular lesion.[12] The presence or absence of an epithelial lining does not seem to prevent closure of the tract as long as the source of the problem is properly diagnosed and adequately treated and the endodontic lesion has healed. Failure of a sinus tract to heal after treatment will necessitate further diagnostic procedures to determine whether other sources of infection are present or whether a misdiagnosis occurred.

In general, a periapical infection that has an associated sinus tract is not painful, although often there is a history of varying magnitudes of discomfort before sinus tract development. Besides providing a conduit for the release of infectious exudate and the subsequent relief of pain, the sinus tract can also provide a useful aid in determining the source of a given infection. Sometimes objective evidence as to the origin of an odontogenic infection is lacking. The stoma of the sinus tract may be located directly adjacent to or at a distant site from the infection. Tracing the sinus tract will provide objectivity in diagnosing the location of the problematic tooth. To trace the sinus tract, a size #25 or #30 gutta-percha cone is threaded into the opening of the sinus tract. Although this may be slightly uncomfortable to the patient, the cone should be inserted until resistance is felt. After a periapical radiograph is exposed, the origin of the sinus tract is determined by following the path taken by the gutta-percha cone (Fig. 1-14). This will direct the clinician to the tooth involved and, more specifically, to the root of the root of the tooth that is the source of the pathosis. Once the causative factors related to the formation of the sinus tract are removed, the stoma and the sinus tract will close within several days.

The stomata of intraoral sinus tracts may open in the alveolar mucosa, in the attached gingiva, or through the furcation or gingival crevice. They may exit through either the facial or the lingual tissues depending on the proximity of the root apices to the cortical bone. If the opening is in the gingival crevice, it is normally present as a narrow defect in one or two isolated areas along the root surface. When a narrow defect is present, the differential diagnosis must include the opening of a periradicular endodontic lesion, a vertical root fracture, or the presence of a developmental groove on the root surface. This type of sinus tract can be differentiated from a primary periodontal lesion because the latter generally presents as a pocket with a broad coronal opening and more generalized alveolar bone loss around the root. Other pulp testing methods may assist in verifying the source of infection.[111,112,121]

Palpation

In the course of the soft tissue examination, the alveolar hard tissues should also be palpated. Emphasis should be placed on detecting any soft tissue swelling or bony expansion, especially noting how it compares with and relates to the adjacent and contralateral tissues. In addition to objective findings, the clinician should question the patient about any areas that feel unusually sensitive during this palpation part of the examination.

A palpation test is performed by applying firm digital pressure to the mucosa covering the roots and apices. The index finger is used to press the mucosa against the underlying cortical bone. This will detect the presence of periradicular abnormalities or specific areas that produce painful response to digital pressure. A positive response to palpation may indicate an active periradicular inflammatory process. This test does not indicate, however, whether the inflammatory process is of endodontic or periodontal origin.

Percussion

Referring back to the patient's chief complaint may indicate the importance of percussion testing for this particular case. If the patient is experiencing acute sensitivity or pain on mastication, this response can typically be duplicated by individually percussing the teeth, which often isolates the symptoms to a particular tooth. Pain to percussion does not indicate that the

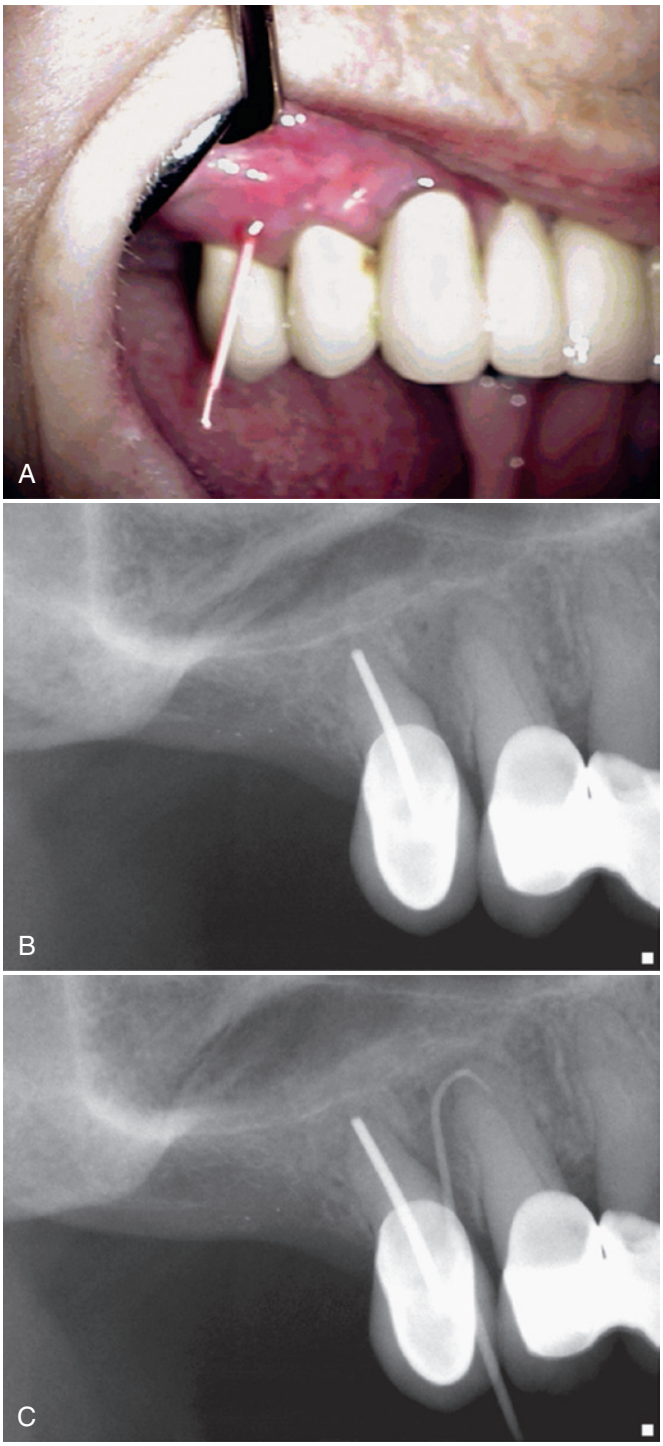


FIG. 1-14 A, To locate the source of an infection, the sinus tract can be traced by threading the stoma with a gutta-percha point. B, Radiograph of the area shows an old root canal in a maxillary second premolar and a questionable radiolucent area associated with the first premolar, with no clear indication of the etiology of the sinus tract. C, After tracing the sinus tract, the gutta-percha is seen to be directed to the source of pathosis, the apex of the maxillary first premolar.

FIG. 1-15 Percussion testing of a tooth, using the back end of a mirror handle.

tooth is vital or nonvital but is rather an indication of inflammation in the periodontal ligament (i.e., symptomatic apical periodontitis). This inflammation may be secondary to physical trauma, occlusal prematurities, periodontal disease, or the extension of pulpal disease into the periodontal ligament space. The indication of where the pain originates is interpreted by the mesencephalic nucleus, receiving its information from proprioceptive nerve receptors. Although subject to debate, the general consensus is that there are relatively few proprioceptors in the dental pulp; however, they are prevalent in the periodontal ligament spaces.[24] This is why it may be difficult for the patient to discriminate the location of dental pain in the earlier stages of pathosis, when only the C fibers are stimulated. Once the disease state extends into the periodontal ligament space, the pain may become more localized for the patient; therefore, the affected tooth will be more identifiable with percussion and mastication testing.

Before percussing any teeth, the clinician should tell the patient what will transpire during this test. Because the presence of acute symptoms may create anxiety and possibly alter the patient's response, properly preparing the patient will lead to more accurate results. The contralateral tooth should first be tested as a control, as should several adjacent teeth that are certain to respond normally. The clinician should advise the patient that the sensation from this tooth is normal and ask to be advised of any tenderness or pain from subsequent teeth.

Percussion is performed by tapping on the incisal or occlusal surfaces of the teeth either with the finger or with a blunt instrument. The testing should initially be done gently, with light pressure being applied digitally with a gloved finger tapping. If the patient cannot detect significant difference between any of the teeth, the test should be repeated using the blunt end of an instrument, like the back end of a mirror handle (Fig. 1-15). The tooth crown is tapped vertically and horizontally. The tooth should first be percussed occlusally, and if the patient discerns no difference, the test should be repeated, percussing the buccal and lingual aspects of the teeth. For any heightened responses, the test should be repeated as necessary to determine that it is accurate and reproducible, and the information should be documented.

Although this test does not disclose the condition of the pulp, it indicates the presence of a periradicular inflammation.

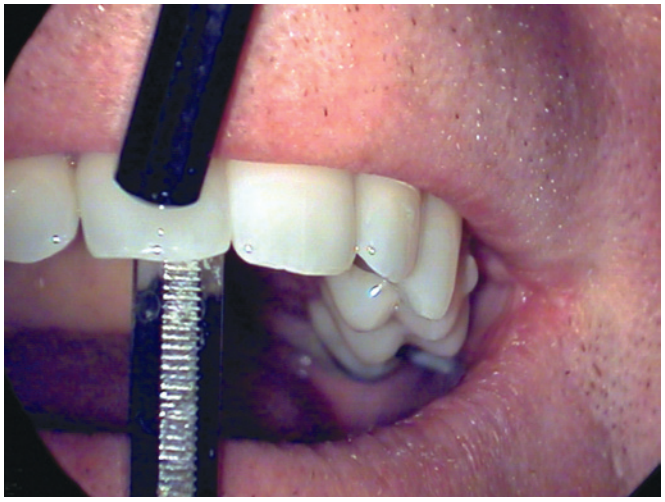


FIG. 1-16 Mobility testing of a tooth, using the back ends of two mirror handles.

BOX 1-2

Recording Tooth Mobility

+1 mobility: The first distinguishable sign of movement greater than normal
+2 mobility: Horizontal tooth movement no greater than 1 mm
+3 mobility: Horizontal tooth movement greater than 1 mm, with or without the visualization of rotation or vertical depressability

BOX 1-3

Recording Furcation Defects

Class I furcation defect: The furcation can be probed but not to a significant depth.
Class II furcation defect: The furcation can be entered into but cannot be probed completely through to the opposite side.
Class III furcation defect: The furcation can be probed completely through to the opposite side.

An abnormal positive response indicates inflammation of the periodontal ligament that may be of either pulpal or periodontal origin. The sensitivity of the proprioceptive fibers in an inflamed periodontal ligament will help identify the location of the pain. This test should be done gently, especially in highly sensitive teeth. It should be repeated several times and compared with control teeth.

Mobility

Like percussion testing, an increase in tooth mobility is not an indication of pulp vitality. It is merely an indication of a compromised periodontal attachment apparatus. This compromise could be the result of acute or chronic physical trauma, occlusal trauma, parafunctional habits, periodontal disease, root fractures, rapid orthodontic movement, or the extension of pulpal disease, specifically an infection, into the periodontal ligament space. Tooth mobility is directly proportional to the integrity of the attachment apparatus or to the extent of inflammation in the periodontal ligament. Often the mobility reverses to normal after the initiating factors are repaired or eliminated. Because determining mobility by simple finger pressure can be visually subjective, the back ends of two mirror handles should be used, one on the buccal aspect and one on the lingual aspect of the tooth (Fig. 1-16). Pressure is applied in a facial-lingual direction as well as in a vertical direction and the tooth mobility is scored (Box 1-2). Any mobility that exceeds +1 should be considered abnormal. However, the teeth should be evaluated on the basis of how mobile they are relative to the adjacent and contralateral teeth.

Periodontal Examination

Periodontal probing is an important part of any intraoral diagnosis. The measurement of periodontal pocket depth is an indication of the depth of the gingival sulcus, which corresponds to the distance between the height of the free gingival margin and the height of the attachment apparatus below. Using a calibrated periodontal probe, the clinician should record the periodontal pocket depths on the mesial, middle, and distal aspects of both the buccal and lingual sides of the tooth, noting the depths in millimeters. The periodontal probe is "stepped" around the long axis of the tooth, progressing in 1-mm increments. Periodontal bone loss that is wide, as determined by a wide span of deep periodontal probings, is generally considered to be of periodontal origin and is typically more generalized in other areas of the mouth. However, isolated areas of vertical bone loss may be of an endodontic origin, specifically from a nonvital tooth whose infection has extended from the periapex to the gingival sulcus. Again, proper pulp testing is imperative, not just for the determination of a diagnosis but also for the development of an accurate prognosis assessment. For example, a periodontal pocket of endodontic origin may resolve after endodontic treatment, but if the tooth was originally vital with an associated deep periodontal pocket, endodontic treatment will not improve the periodontal condition. In addition, as discussed in Chapter 21, a vertical root fracture may often cause a localized narrow periodontal pocket that extends deep down the root surface. Characteristically, the adjacent periodontium is usually within normal limits.

Furcation bone loss can be secondary to periodontal or pulpal disease. The amount of furcation bone loss, as observed both clinically and radiographically, should be documented (Box 1-3). Results of pulp tests (described later) will aid in diagnosis.

Pulp Tests

Pulp testing involves attempting to make a determination of the responsiveness of pulpal sensory neurons.[62,63] The tests involve thermal or electrical stimulation of a tooth in order to obtain a subjective response from the patient (i.e., to determine whether the pulpal nerves are functional), or the tests may involve a more objective approach using devices that detect the integrity of the pulpal vasculature. Unfortunately, the quantitative evaluation of the status of pulp tissue can only be determined histologically, as it has been shown that there is not necessarily a good correlation between the objective clinical signs and symptoms and the pulpal histology.[122,123]

Thermal

Various methods and materials have been used to test the pulp's response to thermal stimuli. The baseline or normal response

to either cold or hot is a patient's report that a sensation is felt but disappears immediately upon removal of the thermal stimulus. Abnormal responses include a lack of response to the stimulus, a lingering or intensification of a painful sensation after the stimulus is removed, or an immediate, excruciatingly painful sensation as soon as the stimulus is placed on the tooth.

Cold testing is the primary pulp testing method used by many clinicians today. It is especially useful for patients presenting with porcelain jacket crowns or porcelain-fused-to-metal crowns where no natural tooth surface (or much metal) is accessible. If a clinician chooses to perform this test with sticks of ice, then the use of a rubber dam is recommended, because melting ice will run onto adjacent teeth and gingiva, yielding potentially false-positive responses.

Frozen carbon dioxide (CO_2), also known as *dry ice* or *carbon dioxide snow*, or *CO_2 stick*, has been found to be reliable in eliciting a positive response if vital pulp tissue is present in the tooth.[46,98,99] One study found that vital teeth would respond to both frozen CO_2 and skin refrigerant, with skin refrigerant producing a slightly quicker response.[66] Frozen carbon dioxide has also been found to be effective in evaluating the pulpal response in teeth with full coverage crowns for which other tests such as electric pulp testing is not possible.[11] For testing purposes, a solid stick of CO_2 is prepared by delivering CO_2 gas into a specially designed plastic cylinder (Fig. 1-17). The resulting CO_2 stick is applied to the facial surface of either the natural tooth structure or crown. Several teeth can be tested with a single CO_2 stick. The teeth should be isolated and the oral soft tissues should be protected with a 2-by-2-inch gauze or cotton roll so the frozen CO_2 will not come into contact with these structures. Because of the extremely cold temperature of the frozen CO_2 (−69°F to −119°F; −56°C to −98°C), burns of the soft tissues can occur. It has been demonstrated on extracted teeth that frozen CO_2 application has resulted in a significantly greater intrapulpal temperature decrease than either skin refrigerant or ice.[11] Also, it appears that the application of CO_2 to teeth does not result in any irreversible damage to the pulp tissues or cause any significant enamel crazing.[61,104]

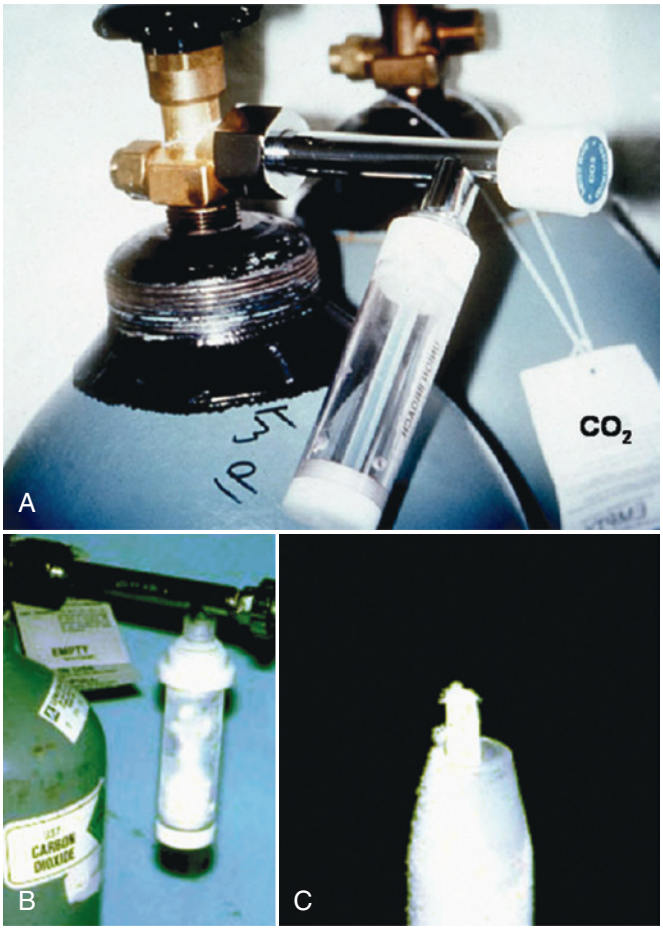


FIG. 1-17 **A,** Carbon dioxide tank with apparatus attached to form solid CO_2 stick/pencil. **B,** CO_2 gas being transformed into a solid stick/pencil. **C,** CO_2 stick/pencil extruded from end of a plastic carrier and ready for use.

The most popular method of performing cold testing is with a refrigerant spray. It is readily available, easy to use, and provides test results that are reproducible, reliable, and equivalent to that of frozen CO_2.[46,66,96,141] One of the current products contains 1,1,1,2-tetrafluoroethane, which has zero ozone depletion potential and is environmentally safe. It has a temperature of −26.2°C.[66] The spray is most effective for testing purposes when it is applied to the tooth on a large #2 cotton pellet (Fig. 1-18). In one study,[65] a significantly lower intrapulpal temperature was achieved when a #2 cotton pellet was dipped or sprayed with the refrigerant compared with the result when a small #4 cotton pellet or cotton applicator was used. The sprayed cotton pellet should be applied to the midfacial area of the tooth or crown. As with any other pulp testing method, adjacent or contralateral "normal" teeth should also be tested to establish a baseline response. It appears that frozen CO_2 and refrigerant spray are superior to other cold testing methods and equivalent or superior to the electric pulp tester for assessing pulp vitality.[11,46] However, one study found that periodontal attachment loss and gingival recession may influence the reported pain response with cold stimuli.[116]

To be most reliable, cold testing should be used in conjunction with an electric pulp tester (described later in this chapter) so that the results from one test will verify the findings of the other test. If a mature, nontraumatized tooth does not respond to both cold testing and electric pulp testing, then the pulp should be considered necrotic.[23,98,141] However, a multirooted tooth, with at least one root containing vital pulp tissue, may respond to a cold test and electric pulp test even if one or more of the roots contain necrotic pulp tissue.[98]

Another thermal testing method involves the use of heat. Heat testing is most useful when a patient's chief complaint is intense dental pain on contact with any hot liquid or food. When a patient is unable to identify which tooth is sensitive, a heat test is appropriate. Starting with the most posterior tooth in that area of the mouth, each tooth is individually isolated with a rubber dam. An irrigating syringe is filled with a liquid (most commonly plain water) that has a temperature similar to that which would cause the painful sensation. The liquid is then expressed from the syringe onto the isolated tooth to determine whether the response is normal or abnormal. The clinician moves forward in the quadrant, isolating each individual tooth until the offending tooth is located. That tooth will exhibit an immediate, intense painful response to the heat. With heat testing, a delayed response may occur, so waiting 10 seconds between each heat test will allow sufficient time for the onset of symptoms. This method can also be used to apply cold water to the entire crown for cases in which cold is the precipitating stimulus.

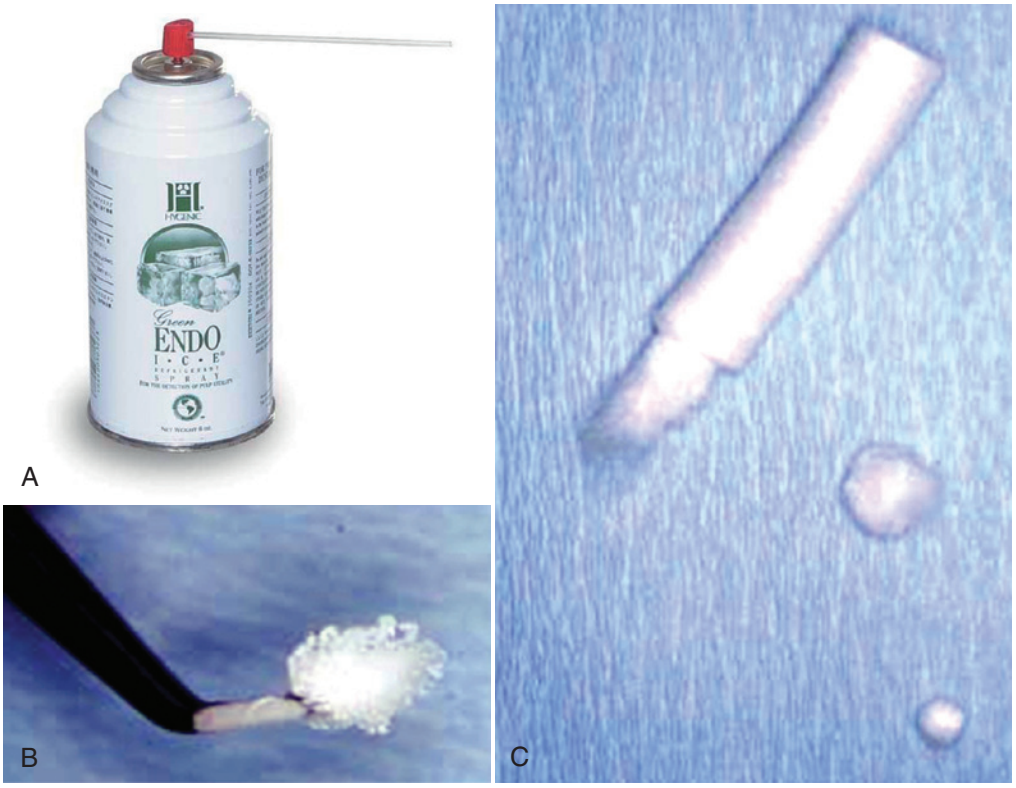


FIG. 1-18 A, Refrigerant spray container. B, A large cotton pellet made of a cotton roll or a ready-made size #2 (large) cotton pellet can be used to apply the refrigerant spray to the tooth surface. The small #4 cotton pellet does not provide as much surface area as the #2 cotton pellet, and therefore should not be used to deliver the refrigerant to the tooth surface. C, A large cotton pellet sprayed with the refrigerant and ready to be applied to the tooth surface. (*A,* Courtesy Coltène/Whaledent, Cuyahoga Falls, OH.)

Another method for heat testing is to apply heated gutta-percha or compound stick to the surface of the tooth. If this method is used, a light layer of lubricant should be placed onto the tooth surface before applying the heated material to prevent the hot gutta-percha or compound from adhering to the dry tooth surface. Heat can also be generated by the friction created when a dry rubber-polishing wheel is run at a high speed against the dry surface of a tooth. However, this latter method is seldom used today and is not recommended. Another approach is the use of electronic heat-testing instruments.[20]

If the heat test confirms the results of other pulp testing procedures, emergency care can then be provided. Often a tooth that is sensitive to heat may also be responsible for some spontaneous pain. The patient may present with cold liquids in hand just to minimize the pain (Fig. 1-19). In such cases, the application of cold to a specific tooth may eliminate the pain and greatly assist in the diagnosis. Typically, a tooth that responds to heat and then is relieved by cold is found to be necrotic.

FIG. 1-19 Irreversible pulpitis associated with the mandibular right second molar. Patient has found that the only way to alleviate the pain is to place a jar filled with ice water against the right side of his face.

Electric

Assessment of pulp neural responses (*vitality*) can also be accomplished by electric pulp testing.[79] Electric pulp testers of different designs and manufacturers have been used for this purpose. Electric pulp testers should be an integral part of any dental practice. It should be noted that the vitality of the pulp is determined by the intactness and health of the vascular supply, not by the status of the pulpal nerve fibers. Even though

BOX 1-4

Potential Common Interpretation Errors of Responses Obtained from Electric Pulp Testing

False-Positive Responses
Partial pulp necrosis
Patient's high anxiety
Ineffective tooth isolation
Contact with metal restorations

False-Negative Responses
Calcific obliterations in the root canals
Recently traumatized teeth
Immature apex
Drugs that increase patient's threshold for pain
Poor contact of pulp tester to tooth

advances are being made with regard to determining the vitality of the pulp on the basis of the blood supply, this technology has not been perfected enough at this time to be used on a routine basis in a clinical setting.

The electric pulp tester has some limitations in providing predictable information about the vitality of the pulp. The response of the pulp to electric testing does not reflect the histologic health or disease status of the pulp.[122,123] A response by the pulp to the electric current only denotes that some viable nerve fibers are present in the pulp and are capable of responding. Numeric readings on the pulp tester have significance only if the number differs significantly from the readings obtained from a control tooth tested on the same patient with the electrode positioned at a similar area on both teeth. However, in most cases, the response is scored as either present or absent. Studies[122,123] have shown that electric pulp test results are most accurate when no response is obtained to any amount of electric current. This lack of response has been found most frequently when a necrotic pulp is present. In addition, false-positive and false-negative responses can occur (Box 1-4), and the clinician must take it into account when formulating the final diagnosis.

The electric pulp tester will not work unless the probe can be placed in contact with or be bridged to the natural tooth structure.[95] With the advent of universal precautions for infection control, the use of rubber gloves prevents the clinician from completing the circuit.[7] Some pulp testers may require the patient to place a finger, or fingers, on the tester probe to complete the electric circuit; however, the use of lip clips is an alternative to having patients hold the tester. Proper use of the electric pulp tester requires the evaluated teeth to be carefully isolated and dried. A control tooth of similar tooth type and location in the arch should be tested first in order to establish a baseline response and to inform the patient as to what a "normal" sensation is. The suspected tooth should be tested at least twice to confirm the results. The tip of the testing probe that will be placed in contact with the tooth structure must be coated with a water- or petroleum-based medium.[86] The most commonly used medium is toothpaste. The coated probe tip is placed in the incisal third of the facial or buccal area of the tooth to be tested.[15] Once the probe is in contact with the tooth, the patient is asked to touch or grasp the tester probe, unless a lip clip is used (Fig. 1-20, *A*). This completes the circuit and initiates the delivery of electric current to the tooth.

FIG. 1-20 A, Electric pulp tester with probe. The probe tip will be coated with a conducive medium such as toothpaste and placed in contact with the tooth surface. The patient will activate the unit by placing a finger on the metal shaft of the probe. B, View of the electric pulp tester control panel; the knob on the front right of the unit controls the rate at which the electric current is delivered to the tooth. The plastic panel on the left front displays the digital numerical reading obtained from the pulp test. The digital scale runs from 0 to 80. (Courtesy SybronEndo, Orange, CA.)

The patient is instructed to remove his or her finger(s) from the probe when a "tingling" or "warming" sensation is felt in the tooth. The readings from the pulp tester are recorded (Fig. 1-20, *B*) and will be evaluated once all the appropriate teeth have been tested by the electric pulp tester and the other pulp testing methods.

If a complete coverage crown or extensive restoration is present, a bridging technique can be attempted to deliver the electric current to any exposed natural tooth structure.[95] The tip of an endodontic explorer is coated with toothpaste or other appropriate medium and placed in contact with the natural tooth structure. The tip of the electric pulp tester probe is coated with a small amount of toothpaste and placed in contact with the side of the explorer. The patient completes the circuit and the testing proceeds as described previously. If no natural tooth structure is available, then an alternative pulp testing method, such as cold, should be used.

One study compared the ability of thermal and electric pulp testing methods to register the presence of vital pulp tissue.[99] The *sensitivity,* which is the ability of a test to identify teeth that are diseased, was 0.83 for the cold test, 0.86 for heat test,

and 0.72 for the electric test. This means the cold test correctly identified 83% of the teeth that had a necrotic pulp, whereas heat tests were correct 86% of the time and electric pulp tests were correct only 72% of the time. This same study evaluated the *specificity* of these three tests. Specificity relates to the ability of a test to identify teeth without disease. Ninety-three percent of teeth with healthy pulps were correctly identified by both the cold and electric pulp tests, whereas only 41% of the teeth with healthy pulps were identified correctly by the heat test. From the results of the testing, it was found that the cold test had an accuracy of 86%, the electric pulp test 81%, and the heat test 71%.

Some studies have indicated there might not be a significant difference between pulp testing results obtained by electric pulp tester and those obtained by the thermal methods.[46,98,99] Cold tests, however, have been shown to be more reliable than electric pulp tests in younger patients with less developed root apices.[5,42,98] This is the reason to verify the results obtained by one testing method and compare them with results obtained by other methods. Until such time that the testing methods used to assess the vascular supply of the pulp become less time consuming and technique sensitive, thermal and electric pulp testing will continue to be the primary methods for determining pulp vitality.

Laser Doppler Flowmetry

Laser Doppler flowmetry (LDF) is a method used to assess blood flow in microvascular systems. Attempts are being made to adapt this technology to assess pulpal blood flow. A diode is used to project an infrared light beam through the crown and pulp chamber of a tooth. The infrared light beam is scattered as it passes through the pulp tissue. The Doppler principle states that the light beam's frequency will shift when hitting moving red blood cells but will remain unshifted as it passes through static tissue. The average Doppler frequency shift will measure the velocity at which the red blood cells are moving.[114]

Several studies[40,60,69,84,114,115,117] have found LDF to be an accurate, reliable, and reproducible method of assessing pulpal blood flow. One of the great advantages of pulp testing with devices such as the LDF is that the collected data are based on objective findings rather than subjective patient responses. As is discussed in Chapter 20, certain luxation injuries will cause inaccuracies in the results of electric and thermal pulp testing. LDF has been shown to be a great indicator for pulpal vitality in these cases.[130] This technology, however, is not being used routinely in the dental practice.

Pulse Oximetry

The pulse oximeter is another noninvasive device (Fig. 1-21). Widely used in medicine, it is designed to measure the oxygen concentration in the blood and the pulse rate. A pulse oximeter works by transmitting two wavelengths of light, red and infrared, through a translucent portion of a patient's body (e.g., a finger, earlobe, or tooth). Some of the light is absorbed as it passes through the tissue; the amount absorbed depends on the ratio of oxygenated to deoxygenated hemoglobin in the blood. On the opposite side of the targeted tissue, a sensor detects the absorbed light. On the basis of the difference between the light emitted and the light received, a microprocessor calculates the pulse rate and oxygen concentration in the blood.[118] The transmission of light to the sensor requires that there be no obstruction from restorations, which can sometimes limit the usefulness of pulse oximetry to test pulp vitality.

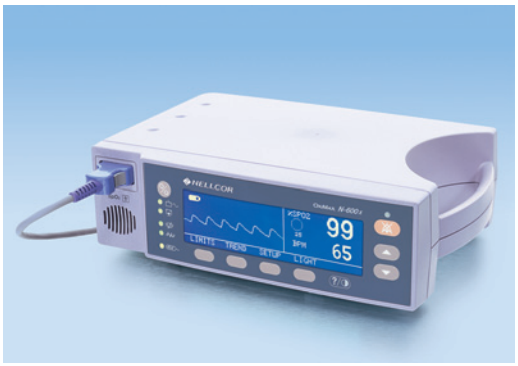


FIG. 1-21 Nellcor OxiMax N-600x pulse oximeter. (Courtesy Nellcor Puritan Bennett, Boulder, CO; now part of Covidien.)

Custom-made sensors have been developed and were found to be more accurate than electric and thermal pulp tests.[31,54] This sensor has been especially useful in evaluating teeth that have been subjected to traumatic injuries, as such teeth tend to present, especially in the short term, with questionable vitality using conventional pulp testing methods.[8,31,53]

Studies regarding the ability of pulse oximetry to diagnose pulp vitality draw various conclusions. Several studies have found pulse oximetry to be a reliable method for assessing pulp vitality.[69,70,118,125,140] Others have stated that in its present form the pulse oximeter may not be predictable in diagnosing pulp vitality.[140] Most of the problems appear to be related to the currently available technology. Some investigators have concluded that the devices used for pulp testing are too cumbersome and complicated to be used on a routine basis in a dental practice.[68,118,140]

Special Tests

Bite Test

Percussion and bite tests are indicated when a patient presents with pain while biting. On occasion, the patient may not know which tooth is sensitive to biting pressure, and percussion and bite tests may help to localize the tooth involved. The tooth may be sensitive to biting when the pulpal pathosis has extended into the periodontal ligament space, creating a *symptomatic apical periodontitis,* or the sensitivity may be present secondary to a crack in the tooth. The clinician can often differentiate between periradicular periodontitis and a cracked tooth or fractured cusp. If periradicular periodontitis is present, the tooth will respond with pain to percussion and biting tests regardless of where the pressure is applied to the coronal part of the tooth. A cracked tooth or fractured cusp will typically elicit pain only when the percussion or bite test is applied in a certain direction to one cusp or section of the tooth.[22,108]

For the bite test to be meaningful, a device should be used that will allow the clinician to apply pressure to individual cusps or areas of the tooth. A variety of devices have been used for bite tests, including cotton tip applicators, toothpicks, orangewood sticks, and rubber polishing wheels. There are several devices specifically designed to perform this test. The Tooth Slooth (Professional Results, Laguna Niguel, CA) (Fig. 1-22) and FracFinder (Hu-Friedy, Oakbrook, IL) are just two of the commercially available devices used for the bite test. As with all pulp tests, adjacent and contralateral teeth should

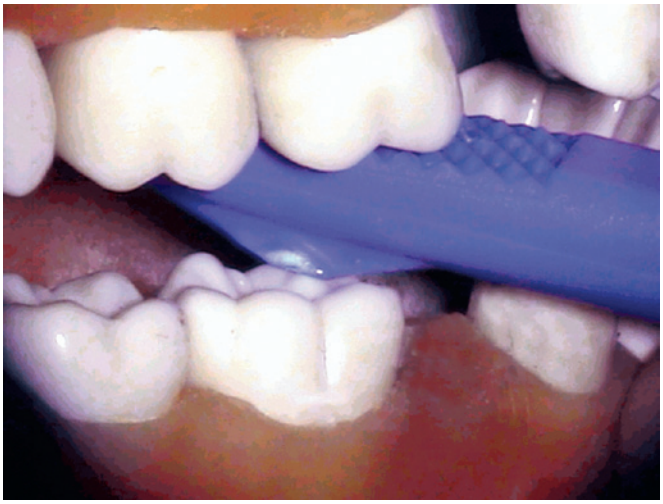


FIG. 1-22 To determine which tooth, or tooth part, is sensitive to mastication, having the patient bite on a specially designed bite stick is often helpful.

be used as controls so that the patient is aware of the "normal" response to these tests. The small cupped-out area on these instruments is placed in contact with the cusp to be tested. The patient is then asked to apply biting pressure with the opposing teeth to the flat surface on the opposite side of the device. The biting pressure should be applied slowly until full closure is achieved. The firm pressure should be applied for a few seconds; the patient is then asked to release the pressure quickly. Each individual cusp on a tooth can be tested in a like manner. The clinician should note whether the pain is elicited during the pressure phase or on quick release of the pressure. A common finding with a fractured cusp or cracked tooth is the frequent presence of pain upon release of biting pressure.

Test Cavity

The test cavity method for assessing pulp vitality is not routinely used since, by definition, it is an invasive irreversible test. This method is used only when all other test methods are deemed impossible or the results of the other tests are inconclusive. An example of a situation in which this method can be used is when the tooth suspected of having pulpal disease has a full coverage crown. If no sound tooth structure is available to use a bridging technique with the electric pulp tester and cold test results are inconclusive, a small class I cavity preparation is made through the occlusal surface of the crown. This is accomplished with a high-speed #1 or #2 round bur with proper air and water coolant. The patient is not anesthetized while this procedure is performed, and the patient is asked to respond if any painful sensation is felt during the drilling procedure. If the patient feels pain once the bur contacts sound dentin, the procedure is terminated and the class I cavity preparation is restored. This sensation signifies only that there is some viable nerve tissue remaining in the pulp, not that the pulp is totally healthy. If the patient fails to feel any sensation when the bur reaches the dentin, this is a good indication that the pulp is necrotic and root canal therapy is indicated.

Staining and Transillumination

To determine the presence of a crack in the surface of a tooth, the application of a stain to the area is often of great assistance. It may be necessary to remove the restoration in the tooth to better visualize a crack or fracture. Methylene blue dye, when painted on the tooth surface with a cotton tip applicator, will penetrate into cracked areas. The excess dye may be removed with a moist application of 70% isopropyl alcohol. The dye will indicate the possible location of the crack.

Transillumination using a bright fiberoptic light probe to the surface of the tooth may be very helpful (Fig. 1-23). Directing a high-intensity light directly on the exterior surface of the tooth at the cementum-enamel junction (CEJ) may reveal the extent of the fracture. Teeth with fractures block transilluminated light. The part of the tooth that is proximal to the light source will absorb this light and glow, whereas the area beyond this fracture will not have light transmitted to it and will show as gray by comparison.[101] Although the presence of a fracture may be evident using dyes and transillumination, the depth of the fracture cannot always be determined.

Selective Anesthesia

When symptoms are not localized or referred, the diagnosis may be challenging. Sometimes the patient may not even be able to specify whether the symptoms are emanating from the maxillary or mandibular arch. In these instances, when pulp testing is inconclusive, *selective anesthesia* may be helpful.

If the patient cannot determine which arch the pain is coming from, then the clinician should first selectively anesthetize the maxillary arch. This should be accomplished by using a periodontal ligament (intraligamentary) injection. The injection is administered to the most posterior tooth in the quadrant of the arch that may be suspected, starting from the distal sulcus. The anesthesia is subsequently administered in an anterior direction, one tooth at a time, until the pain is eliminated. If the pain is not eliminated after an appropriate period of time, then the clinician should similarly repeat this technique on the mandibular teeth below. It should be understood that periodontal ligament injections may anesthetize an adjacent tooth and thus are more useful for identifying the arch rather than the specific tooth.

Radiographic Examination and Interpretation

Intraoral Radiographs

The radiographic interpretation of a potential endodontic pathosis is an integral part of endodontic diagnosis and prognosis assessment. Few diagnostic tests provide as much useful information as dental radiography. For this reason, the clinician is sometimes tempted to prematurely make a definitive diagnosis based solely on radiographic interpretation. However, the image should be used only as one sign, providing important clues in the diagnostic investigation. When not coupled with a proper history and clinical examination and testing, the radiograph alone can lead to a misinterpretation of normality and pathosis (Fig. 1-24). Because treatment planning will ultimately be based on the diagnosis, the potential for inappropriate treatment may frequently exist if the radiograph alone is used for making final diagnosis. The clinician should not subject the patient to unnecessary multiple radiation exposures; two pretreatment images from different angulations are often sufficient. Under extenuating circumstances, however, especially when the diagnosis is difficult, additional exposures may be necessary to determine the presence of multiple roots,

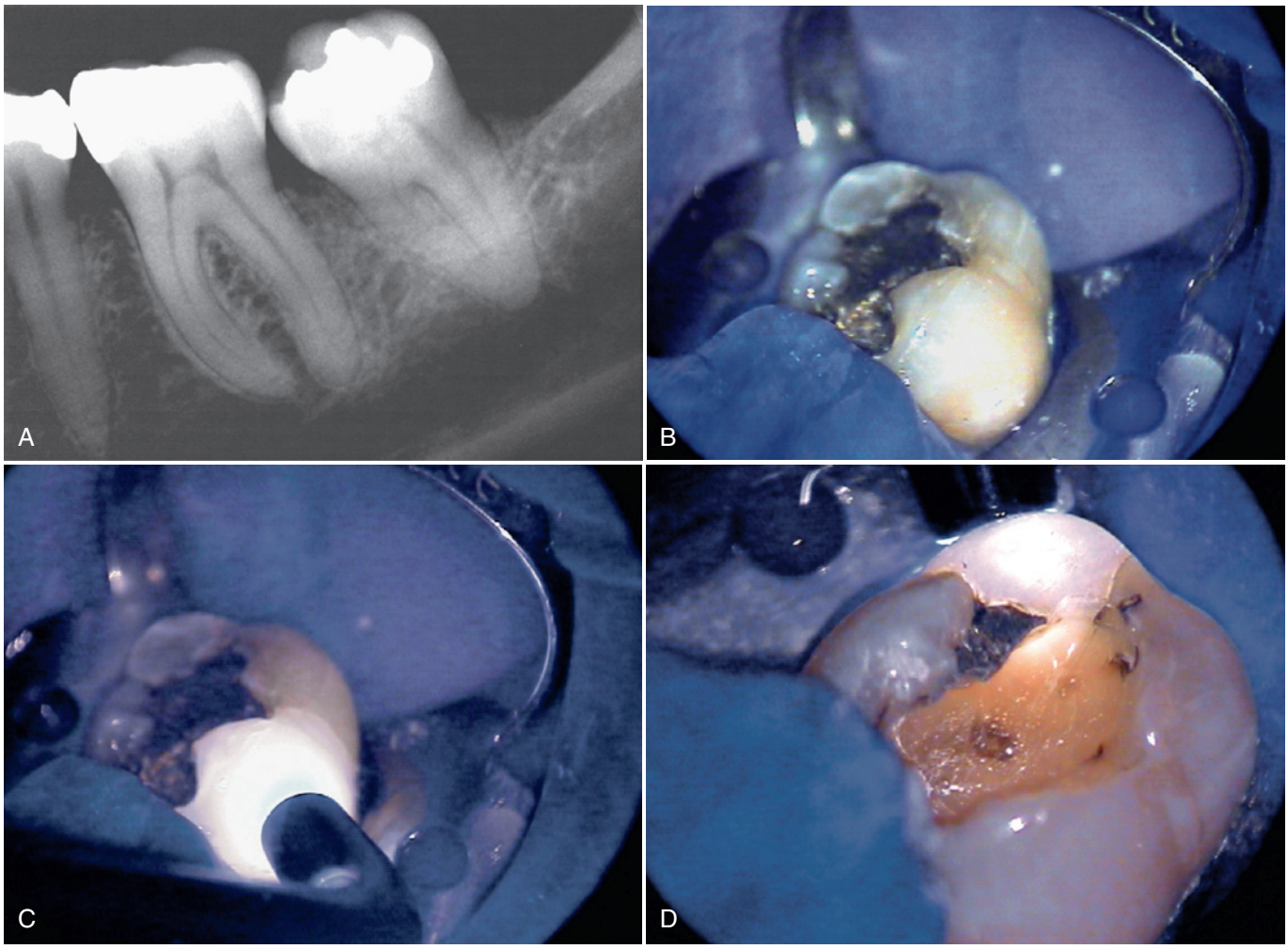


FIG. 1-23 Sometimes there is no clear indication of why a tooth is symptomatic. This radiograph shows a mandibular second molar with a moderately deep restoration **(A)**; the pulp tests nonvital. Without any transillumination, a fracture cannot be detected **(B)**. However, by placing a high-intensity light source on the tooth surface, a root fracture can be observed on the buccal surface **(C)** and the distal-lingual surface **(D)**.

multiple canals, resorptive defects, caries, restoration defects, root fractures, and the extent of root maturation and apical development.

The radiographic appearance of endodontic pathosis can sometimes be highly subjective. In a study by Goldman and colleagues, there was only 50% agreement among interpreters for the radiographic presence of pathosis.[49] When the cases were reevaluated several months later, the same evaluators agreed with their own original diagnosis less than 85% of the time.[50] This further emphasizes the necessity for additional objective diagnostic tests, as well as the importance of obtaining and comparing older radiographs.

For standard two-dimensional radiography, clinicians basically project x-radiation through an object and capture the image on a recording medium, either x-ray film or a digital sensor. Much like casting a shadow from a light source, the image appearance may vary greatly depending on how the radiographic source is directed. Thus, the three-dimensional interpretation of the resulting two-dimensional image requires not only knowledge of normality and pathosis but also advanced knowledge of how the radiograph was exposed. By virtue of "casting a shadow," the anatomic features that are closest to the film (or sensor) will move the least when there is a change in the horizontal or vertical angulation of the radiation source (Fig. 1-25). This may be helpful in determining the existence of additional roots, the location of pathosis, and the unmasking of anatomic structures. Changes in the horizontal or vertical angulation may help elucidate valuable anatomic and pathologic information; it also has the potential to hide important information. An incorrect vertical angulation may cause the buccal roots of a maxillary molar to be masked by the zygomatic arch. An incorrect horizontal angulation may cause roots to overlap with the roots of adjacent teeth, or it may incorrectly create the appearance of a one-rooted tooth, when two roots are actually present.

In general, when endodontic pathosis appears radiographically, it appears as bone loss in the area of the periapex. The pathosis may present merely as a widening or break in the lamina dura—the most consistent radiographic finding when a tooth is nonvital[67]—or it may present as a radiolucent area at the apex of the root or in the alveolar bone adjacent to the exit of a lateral or furcation accessory canal. On occasion there may be no radiographic change at all, even in the presence of a disease process in the alveolar bone.

FIG. 1-24 Radiograph showing what appears to be a mandibular lateral incisor associated with periapical lesion of a nonvital tooth. Although pulp necrosis can be suspected, the tooth tested vital. In this case, the appearance of apical bone loss is secondary to a cementoma.

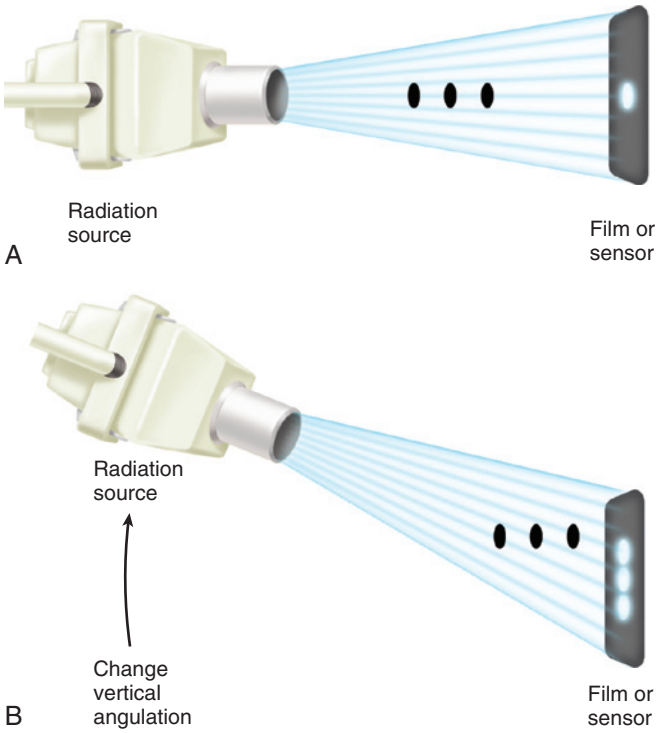


FIG. 1-25 Radiographic images are only two-dimensional, and often it is difficult to discriminate the relative location of overlapping objects. A, When the source of radiation is directly perpendicular to overlapping objects, the image is captured without much separation of the objects. However, when the radiation source is at an angle to offset the overlapping objects, the image is captured with the objects being viewed as separated. B, The object that is closest to the film (or sensor) will move the least, with the object closest to the radiation source appearing farthest away.

Two-dimensional dental radiography has two basic shortcomings: the lack of early detection of pathosis in the cancellous bone, because of the density of the cortical plates, and the influence of the superimposition of anatomic structures. Variability in the radiographic expression of an osseous pathosis has much to do with the relative location of the root of the tooth and how it is oriented with respect to the cortical and cancellous bone. Radiographic changes from bone loss will not be detected if the loss is only in cancellous bone.[16] However, the radiographic evidence of pathosis will be observed once this bone loss extends to the junction of the cortical and cancellous bone. In addition, certain teeth are more prone to exhibit radiographic changes than others, depending on their anatomic location.[17] The radiographic appearance of endodontic pathosis is correlated with the relationship of the periapex of the tooth and its juxtaposition to the cortical-cancellous bone junction. The apices of most anterior and premolar teeth are located close to the cortical-cancellous bone junction. Therefore, periapical pathosis from these teeth is exhibited sooner on the radiograph. By comparison, the distal roots of mandibular first molars and both roots of mandibular second molars are generally positioned more centrally within the cancellous bone, as are maxillary molars, especially the palatal roots. Periapical lesions from these roots must expand more before they reach the cortical-cancellous bone junction and are recognized as radiographic pathosis. For these reasons, it is important not to exclude the possibility of pulpal pathosis in situations in which there are no radiographic changes.

Many factors can influence the quality of the radiographic interpretation, including the ability of the person exposing the radiograph, the quality of the radiographic film, the quality of the exposure source, the quality of the film processing, and the skill with which the film is viewed. Controlling all of these variables can be a difficult challenge but is paramount for obtaining an accurate radiographic interpretation.

Digital Radiography

Digital radiography has been available since the late 1980s and has recently been refined with better hardware and more user-friendly software. It has the ability to capture, view, magnify, enhance, and store radiographic images in an easily reproducible format that does not degrade over time. Significant advantages of digital radiographs over conventional radiographs include lower radiation doses, instant viewing, convenient manipulation, efficient transmission of an image via the Internet, simple duplication; and easy archiving.

Digital radiography uses no x-ray film and requires no chemical processing. Instead, a *sensor* is used to capture the image created by the radiation source. This sensor is either directly or wirelessly attached to a local computer, which interprets this signal and, using specialized software, translates the signal into a two-dimensional digital image that can be displayed, enhanced, and analyzed. The image is stored in the patient's file, typically in a dedicated network server, and can be recalled as needed. Further information about digital radiography may be found in Chapter 2.

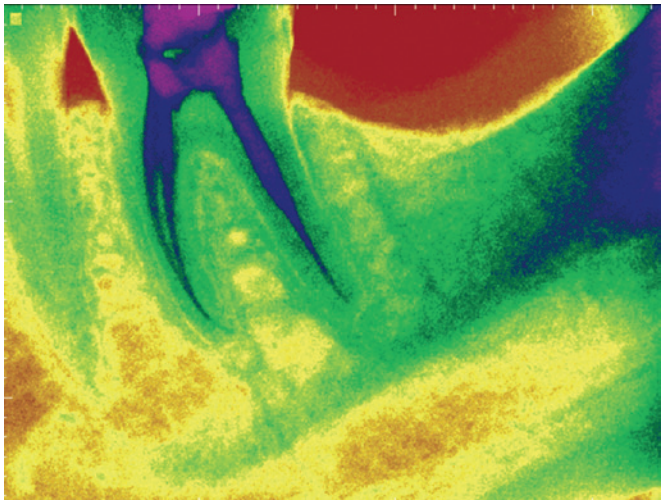


FIG. 1-26 Digital radiography has an advantage over conventional film in that the image can be enhanced and colorized, a useful tool for patient education.

The viewing of a digital radiographic image on a high-resolution monitor allows for rapid and easy interpretation for both the clinician and the patient. The image appears almost instantly, with no potential for image distortion from improper chemical processing. The clinician can magnify different areas on the radiograph and then digitally enhance the image in order to better visualize certain anatomic structures; in some cases the image can even be colorized, a useful tool for patient education (Fig. 1-26).

In the past, x-ray film has had a slightly better resolution than most digital radiography images, at about 16 line pairs per millimeter (lp/mm).[87] Some sensor manufacturers, however, now claim to offer resolutions beyond that of conventional film. Under the best of circumstances, the human eye can see only about 10 lp/mm, which is the lowest resolution for most dental digital radiography systems. Digital sensors are much more sensitive to radiation than conventional x-ray film and thus require 50% to 90% less radiation in order to acquire an image, an important feature for generating greater patient acceptance of dental radiographs.

The diagnostic quality of this expensive technology has been shown to be comparable to, but not necessarily superior to, perfectly exposed and perfectly processed conventional film-based radiography.[39,73,97] Furthermore, it was found that the interpretation of a digital radiograph can be subjective, similar to that of the conventional film.[134] Factors that appear to have the most impact on the interpretation of the image are the years of experience of the examiner and familiarity of the operator with the given digital system.[134]

Cone-Beam Computerized Tomography

Limitations in conventional two-dimensional radiography promulgated a need for three-dimensional imaging, known as *cone-beam computerized tomography* (CBCT) (also known as *cone-beam volumetric tomography* [CBVT]) or as *cone-beam volumetric imaging* [CBVI]. Although a form of this technology has existed since the early 1980s,[106] specific devices for dental use first appeared almost two decades later.[90] Most of these machines are similar to a dental panoramic radiographic device, whereby the patient stands or sits as a cone-shaped

FIG. 1-27 Cone-beam volumetric tomography, using the 3D Accuitomo 80. (Courtesy J. Morita USA, Irvine, CA.)

FIG. 1-28 Cone-beam volumetric tomography has the ability to capture, store, and present radiographic images in various horizontal and vertical planes. (Courtesy J. Morita USA, Irvine, CA.)

radiographic beam is directed to the target area with a reciprocating capturing sensor on the opposite side (Fig. 1-27). The resulting information is digitally reconstructed and interpreted to create an interface whereby the clinician can three-dimensionally interpret "slices" of the patient's tissues in a multitude of planes (Figs. 1-28 and 1-29).[37,33] The survey of the scans can be interpreted immediately after the scan. Various software applications have been used to enable the images to be sent to other clinicians. This is accomplished either in printed format or with portable and transferable software that can be used interactively by another clinician.

In general, many dental applications only require a limited field of vision, confining the study to the maxilla and mandible.

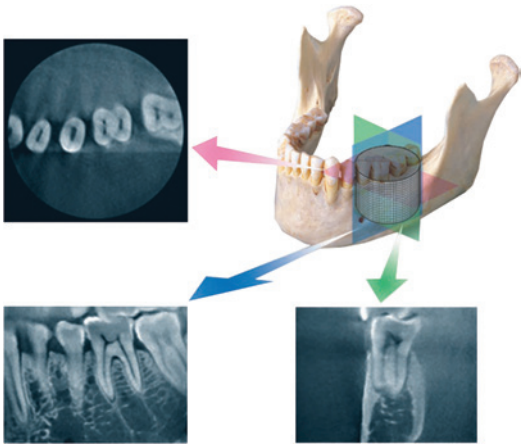


FIG. 1-29 Cone-beam volumetric tomography has the advantage of being able to detect pathosis in the bone or associated with the teeth without the obstruction of anatomic structures. The planes of vision may be axial, sagittal, or coronal. (Courtesy J. Morita USA, Irvine, CA.)

However, many devices have the ability to provide a full field of vision for viewing more regional structures. Clinicians should thoroughly understand the ethical and medical-legal ramifications of doing scans with full fields of view. Incidental nondental findings have been seen from these scans, such as intracranial aneurysms, that when undetected could be life threatening.[91]

The radiation source of CBCT is different from that of conventional two-dimensional dental imaging in that the radiation beam created is conical in shape. Also, conventional digital dental radiography is captured and interpreted as *pixels*, a series of dots that collectively produces an image of the scanned structure. For CBCT, the image is instead captured as a series of three-dimensional pixels, known as *voxels*. Combining these voxels gives a three-dimensional image that can be "sliced" into various planes, allowing for specific evaluations never before possible without a necropsy (Fig. 1-30). One of the advantages of using a device that has a limited field of vision is that the voxel size can be less than half that of a device using a full field of vision, thereby increasing the resolution of the resulting image and providing for a more accurate interpretation of anatomic structures and pathologic conditions. The development of limited field of vision devices has also contributed to decreasing the costs of these relatively expensive machines, making them more practical for dental office use.[41]

Compared with two-dimensional radiographs, CBCT can clearly visualize the interior of the cancellous bone without the superimposition of the cortical bone. Studies show that CBCT is much more predictable and efficient in demonstrating anatomic landmarks, bone density, bone loss, periapical lesions, root fractures, root perforations and root resorptions.[1,21,26,27,38,47,71,78,81,85,92,94,128,131,142]

The superimposition of anatomic structures can also mask the interpretation of alveolar defects. Specifically, the maxillary sinus, zygoma, incisive canal and foramen, nasal bone, orbit, mandibular oblique ridge, mental foramen, mandibular mentalis, sublingual salivary glands, tori, and the overlap of adjacent roots may either obscure bone loss or mimic bone loss, making an accurate interpretation of conventional radiography sometimes difficult or impossible. Several studies have

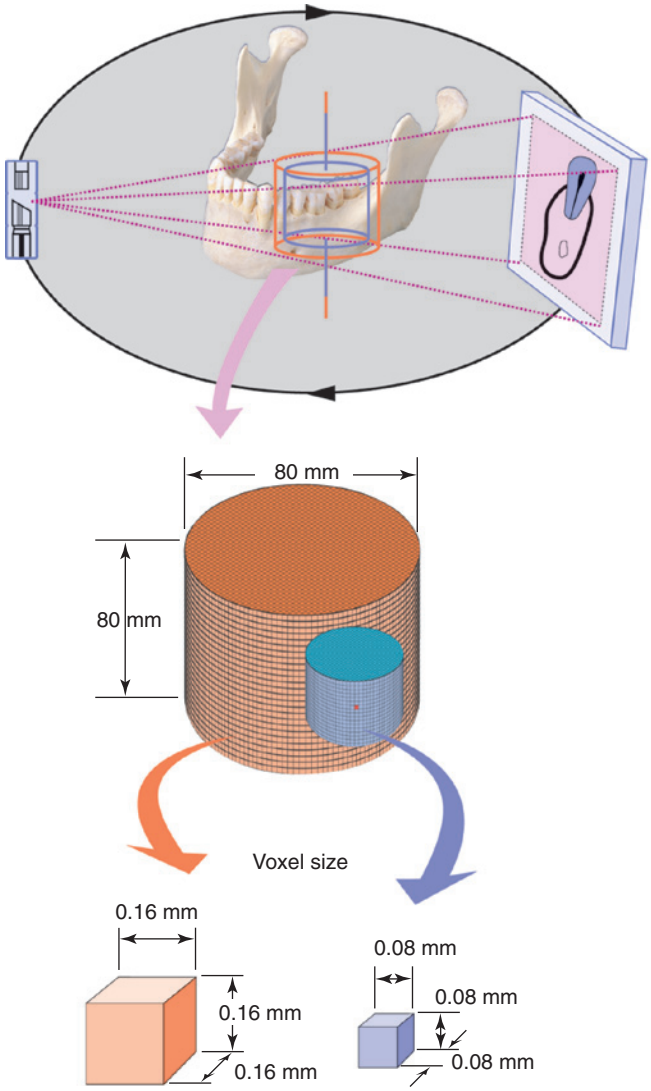


FIG. 1-30 The radiation source in cone-beam volumetric tomography is conical. The receiving sensor captures the image as "voxels," or three-dimensional pixels of information, allowing digital interpretation.

demonstrated the advantages of CBCT in the differential diagnosis of such structures from pathologic conditions.[21,29,71,137]

Cone-beam computerized tomography should not be seen as a replacement for conventional dental radiography, but rather as a diagnostic adjunct. The advantage of conventional dental radiography is that it can visualize most of the structures in one image. CBCT can show great detail in many planes of vision but can also leave out important details if the "slice" is not in the area of existing pathosis (Fig. 1-31). There is a promising future for the use of CBCT for endodontic diagnosis and treatment. It has already proven invaluable in the detection of dental and nondental pathoses (Fig. 1-32). For a further review of CBCT and radiography, see Chapter 2.

Magnetic Resonance Imaging (MRI)

MRI has also been suggested for dental diagnosis. It may offer simultaneous three-dimensional hard- and soft-tissue imaging of teeth without ionizing radiation.[58] The use of MRI in endodontics is still limited.

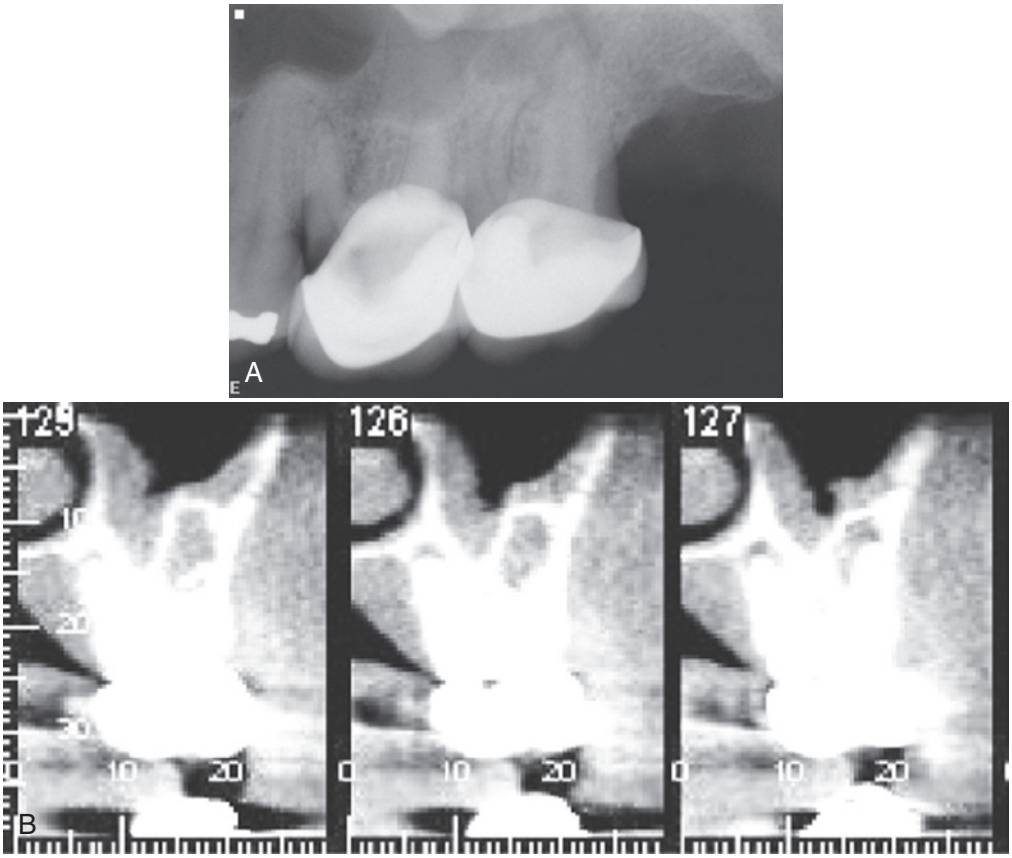


FIG. 1-31 **A,** This standard two-dimensional radiographic image reveals recurrent caries under the mesial margin of the maxillary first molar. However, the patient localized pain to mastication on the maxillary second molar. **B,** Cone-beam volumetric tomography revealed an apical radiolucency associated with the maxillary second molar. The bone loss was obscured on the two-dimensional radiograph by the maxillary sinus, zygoma, and cortical bone.

Cracks and Fractures

The wide variety of types of cracks and fractures in teeth and their associated signs and symptoms often make their diagnosis difficult. The extensiveness of the crack or fracture line may directly alter the prognosis assessment for a given tooth and should be examined before treatment decision making. Certain types of cracks may be as innocent as a superficial enamel craze line, or they may be as prominent as a fractured cusp. The crack may progress into the root system to involve the pulp, or it may split the entire tooth into two separate segments. The crack may be oblique, extending cervically, such that once the coronal segment is removed the tooth may or may not be restorable. Any of these situations may present with mild, moderate, or severe symptoms or possibly no symptoms at all.

Crack Types

There have been many suggestions in the literature of was to classify cracks in teeth. By defining the type of crack present, an assessment of the prognosis may be determined and treatment alternatives can be planned (see Chapter 21). Unfortunately, it is often extremely difficult to determine how extensive a crack is until the tooth is extracted.

Cracks in teeth can be divided into three basic categories:

- Craze lines
- Fractures (also referred to as *cracks*)
- Split tooth/roots

Craze lines are merely cracks in the enamel that do not extend into the dentin and either occur naturally or develop after trauma. They are more prevalent in adult teeth and usually occur more in the posterior teeth. If light is transilluminated through the crown of such a tooth, these craze lines may show up as fine lines in the enamel with light being able to transmit through them, indicating that the crack is only superficial. The use of optical coherence tomography (OCT) has also been suggested for detection of enamel cracks.[59] Craze lines typically will not manifest with symptoms. No treatment is necessary for craze lines unless they create a cosmetic issue.

Fractures extend deeper into the dentin than superficial craze lines and primarily extend mesially to distally, involving the marginal ridges. Dyes and transillumination are helpful for visualizing potential root fractures.

Symptoms from a fractured tooth range from none to severe pain. A fracture in the tooth does not necessarily dictate that the tooth has split into two pieces; however, left alone or especially with provocations such as occlusal prematurities, the fracture may progress into a split root. A fractured tooth may be treated by a simple restoration, endodontics (nonsurgical or surgical), or even extraction, depending on the extent and orientation of the fracture, the degree of symptoms, and whether the symptoms can be eliminated. This makes the clinical management of fractured teeth difficult and sometimes unpredictable.

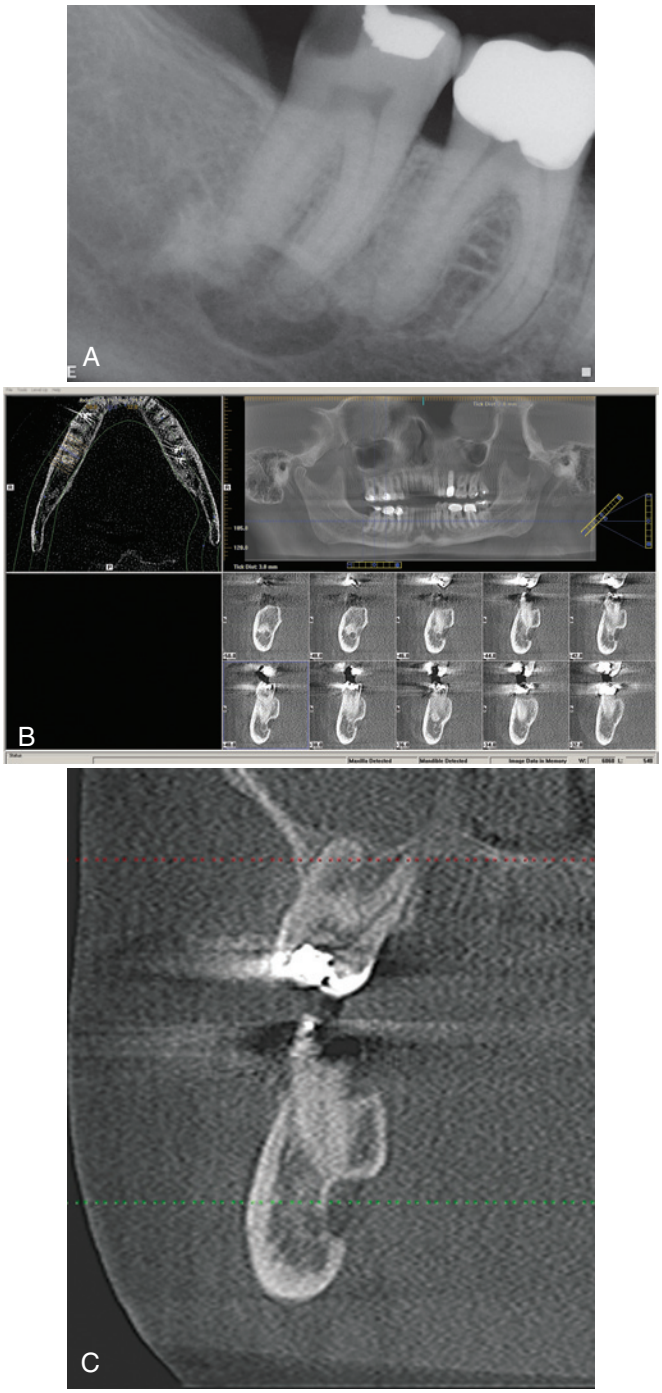


FIG. 1-32 A, Periapical radiograph showing a large apical radiolucency associated with the mandibular second molar. Apical pathosis should be ruled out. B, Cone-beam volumetric tomography revealed salivary indentation of the mandible in the area apical and lingual to the mandibular second molar, consistent with a Stafne defect. C, Enlargement of coronal section in the area of the mandibular second molar and the Stafne defect located on the lingual aspect of the mandible.

A definitive combination of factors, signs, and symptoms that, when collectively observed, allows the clinician to conclude the existence of a specific disease state is termed a *syndrome*. However, given the multitude of signs and symptoms that fractured roots can present with, it is often difficult to achieve an objective definitive diagnosis. For this reason, the terminology of *cracked tooth syndrome*[22,108] should be avoided.[6] The subjective and objective factors seen in cases of fractured teeth will generally be diverse; therefore, a tentative diagnosis of a fractured tooth will most likely be more of a prediction. Once this prediction is made, the patient must be properly informed as to any potential decrease in prognosis of the pending dental treatment. Because treatment options for repairing fractured teeth have only a limited degree of success, early detection and prevention, and proper informed consent, are crucial.[9,10,72,119,120,124,132]

Split tooth/roots occur when a fracture extends from one surface of the tooth to another surface of the tooth, with the tooth separating into two segments. If the split is more oblique, it is possible that once the smaller separated segment is removed, the tooth might still be restorable—for example, a fractured cusp. However, if the split extends below the osseous level, the tooth may not be restorable and endodontic treatment may not result in a favorable prognosis.

Proper prognosis assessment is imperative before any dental treatment but is often difficult in cases of cracked teeth. Because of the questionable long-term success from treating cases of suspected or known fractures, the clinician should be cautious in making the decision to continue with treatment and should avoid endodontic treatment in cases of a definitive diagnosis of split roots.

Vertical Root Fractures

One of the more common reasons for recurrent endodontic pathosis is the *vertical root fracture,* a severe crack in the tooth that extends longitudinally down the long axis of the root (Figs. 1-33 and 1-34). Often it extends through the pulp and to the periodontium. It tends to be more centrally located within the tooth, as opposed to being more oblique, and typically traverses through the marginal ridges. These fractures may be present before endodontic treatment, secondary to endodontic treatment, or may develop after endodontic treatment has been completed. Because diagnosing these vertical root fractures may be difficult, they often go unrecognized. Therefore, diagnosing the existence and extent of a vertical root fracture is imperative before any restorative or endodontic treatment is done, as it can dramatically affect the overall success of treatment.

A patient who consents to endodontic treatment must be informed if the tooth has a questionable prognosis. The clinician must be able to interpret the subjective and objective findings that suggest a vertical root fracture or split tooth, be able to make a prediction as to the eventual potential of healing, and convey this information to the patient. A more detailed discussion on vertical root fractures is described in Chapter 21.

Perforations

Root perforations are clinical complications that may lead to treatment failure. When root perforation occurs, communications between the root canal system and either periradicular tissues or the oral cavity may reduce the prognosis of treatment. Root perforations may result from extensive carious lesions, resorption, or operator error occurring during root canal instrumentation or post preparation.

The treatment prognosis of root perforations depends on the size, location, time of diagnosis and treatment, degree of periodontal damage, as well as the sealing ability and biocompatibility of the repair material.[45] It has been recognized that

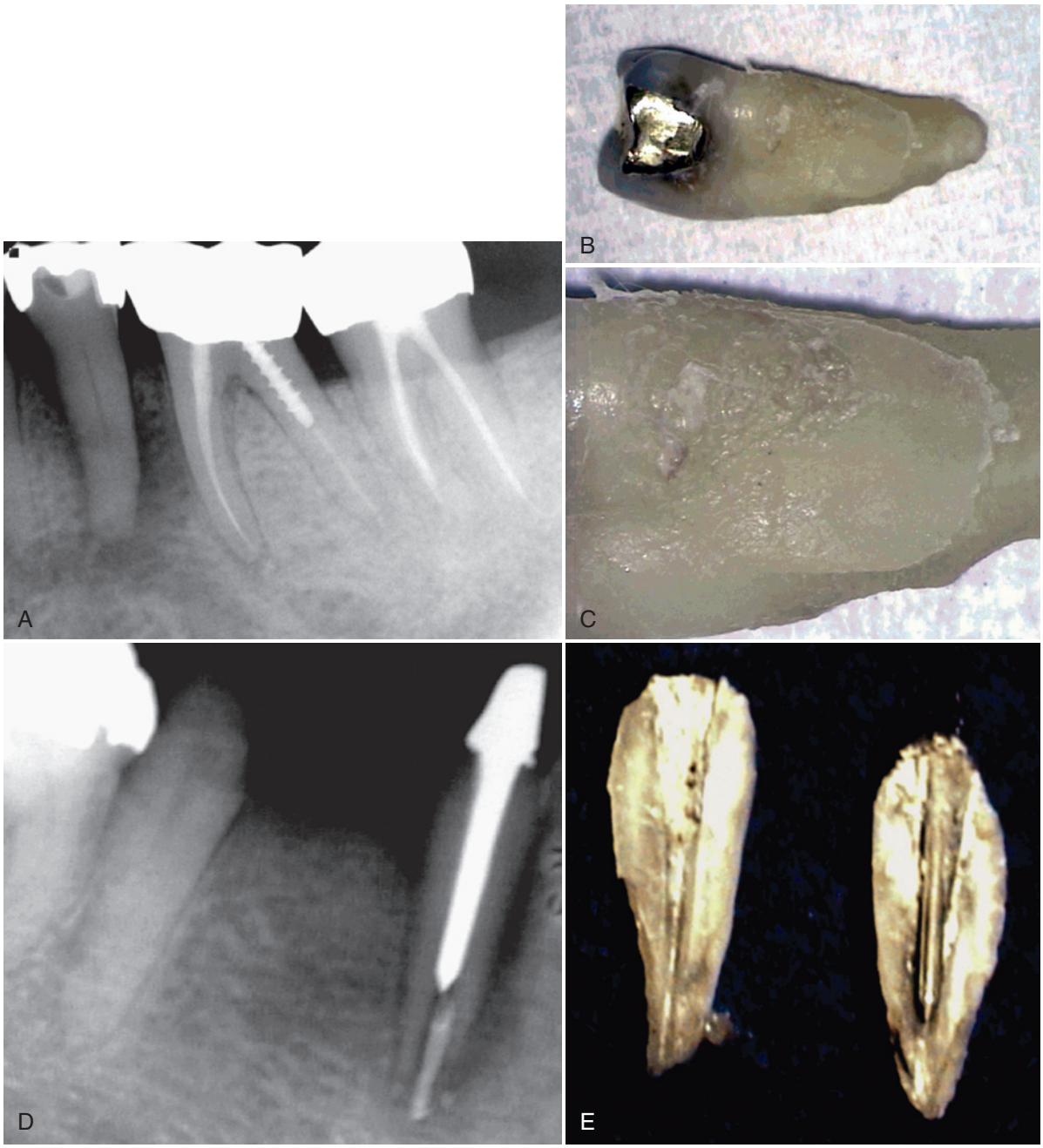


FIG. 1-33 Poorly fitting intracoronal restorations can place stresses within the tooth that can cause a vertical root fracture. **A,** This radiograph of a mandibular second premolar (with a gold inlay) reveals extensive periapical and periradicular bone loss, especially on the distal aspect. **B,** The tooth pulp tested nonvital, and there was an associated 12-mm-deep, narrow, isolated periodontal pocket on the buccal aspect of the tooth. After the tooth was extracted, the distal aspect was examined. **C,** On magnification (×16) the distal aspect of the root revealed an oblique vertical root fracture. Similarly, the placement of an ill-fitting post may exert intraradicular stresses on a root that can cause a fracture to occur vertically. **D,** This radiograph depicts a symmetrical space between the obturation and the canal wall, suggesting a vertical root fracture. **E,** After the tooth is extracted, the root fracture can be easily observed.

treatment success depends mainly on immediate sealing of the perforation and appropriate infection control. Among the materials that are commonly used to seal root perforations are mineral trioxide aggregate (MTA), Super EBA, intermediate restorative material (IRM), glass ionomer cements, and composites. The topic of perforations is further discussed in Chapter 19.

CLINICAL CLASSIFICATION OF PULPAL AND PERIAPICAL DISEASES

Many attempts have been made over the years to develop classifications of pulpal and periapical disease. However, studies have shown that making a correlation between clinical signs and symptoms and the histopathology of a given clinical

FIG. 1-34 Physical trauma from sports-related injuries or seizure-induced trauma, if directed accordingly, may cause a vertical root fracture in a tooth. This fracture occurred in a 7-year-old child secondary to trauma from a grand mal seizure.

condition is challenging.[122,123] Therefore, *clinical* classifications have been developed in order to formulate treatment plan options. In the most general terms, the objective and subjective findings are used to classify the suspected pathosis, with the assigned designations merely representing the presence of healthy or diseased tissue.

The terminology and classifications that follow are based on those suggested by the American Association of Endodontists in 2012.[6]

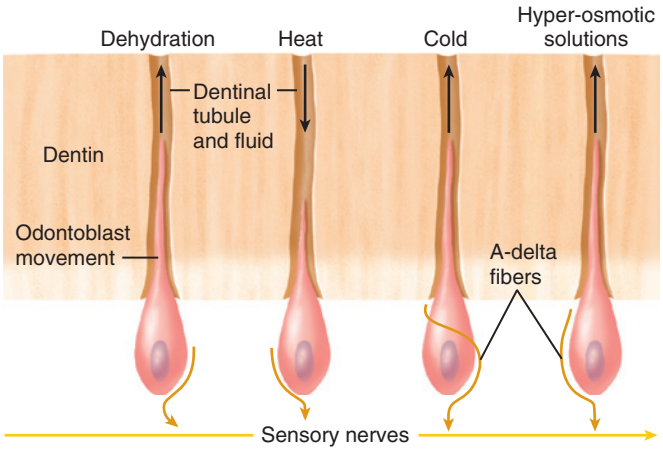
Pulpal Disease

Normal Pulp

This is a clinical diagnostic category in which the pulp is symptom-free and normally responsive to pulp testing.[6] Teeth with normal pulp do not usually exhibit any spontaneous symptoms. The symptoms produced from pulp tests are mild, do not cause the patient distress, and result in a transient sensation that resolves in seconds. Radiographically, there may be varying degrees of pulpal calcification but no evidence of resorption, caries, or mechanical pulp exposure. No endodontic treatment is indicated for these teeth.

Pulpitis

This is a clinical and histologic term denoting inflammation of the dental pulp, clinically described as reversible or irreversible and histologically described as acute, chronic, or hyperplastic.[6]



Dentin tubule fluid movement

FIG. 1-35 Dentinal tubules are filled with fluid that, when stimulated, will cause sensation. Temperature changes, air, and osmotic changes can provoke the odontoblastic process to induce the stimulation of underlying A-delta fibers.

Reversible Pulpitis

This is a clinical diagnosis based on subjective and objective findings indicating that the inflammation should resolve and the pulp return to normal.[6] When the pulp within the tooth is irritated so that the stimulation is uncomfortable to the patient but reverses quickly after irritation, it is classified as *reversible pulpitis*. Causative factors include caries, exposed dentin, recent dental treatment, and defective restorations. Conservative removal of the irritant will resolve the symptoms. Confusion can occur when there is exposed dentin, without evidence of pulp pathosis, which can sometimes respond with sharp, quickly reversible pain when subjected to thermal, evaporative, tactile, mechanical, osmotic, or chemical stimuli. This is known as *dentin* (or *dentinal*) *sensitivity* (or *hypersensitivity*). Exposed dentin in the cervical area of the tooth accounts for most of the cases diagnosed as dentin sensitivity.[103]

As described in Chapter 12, fluid movement within dentinal tubules can stimulate the odontoblasts and associated fast-conducting A-delta nerve fibers in the pulp, which in turn produce sharp, quickly reversible dental pain (Fig. 1-35). The more open these tubules are (e.g., from a newly exposed preparation, dentin decalcification, periodontal scaling, tooth-bleaching materials, or coronal tooth fractures), the more the tubule fluid will move and, subsequently, the more the tooth will display dentin sensitivity when stimulated. When making a diagnosis, it is important to discriminate this dentin sensitivity sensation from that of reversible pulpitis, which would be secondary to caries, trauma, or new or defective restorations. Detailed questioning about recent dental treatment and a thorough clinical and radiographic examination will help to separate dentin sensitivity from other pulpal pathosis, as the treatment modalities for each are completely different.[18]

Irreversible Pulpitis

As the disease state of the pulp progresses, the inflammatory condition of the pulp can change to *irreversible pulpitis*. At this stage, treatment to remove the diseased pulp will be necessary. This condition can be divided into the subcategories of *symptomatic* and *asymptomatic* irreversible pulpitis.

Symptomatic Irreversible Pulpitis

This is a clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing.[6] Teeth that are classified as having *symptomatic irreversible pulpitis* exhibit intermittent or spontaneous pain. Rapid exposure to dramatic temperature changes (especially to cold stimuli) will elicit heightened and prolonged episodes of pain even after the thermal stimulus has been removed. The pain in these cases may be sharp or dull, localized, diffuse, or referred. Typically, there are minimal or no changes in the radiographic appearance of the periradicular bone. With advanced irreversible pulpitis, a thickening of the periodontal ligament may become apparent on the radiograph, and there may be some evidence of pulpal irritation by virtue of extensive pulp chamber or root canal space calcification. Deep restorations, caries, pulp exposure, or any other direct or indirect insult to the pulp, recently or historically, may be present. It may be seen radiographically or clinically or may be suggested from a complete dental history. Patients who present with symptomatic anterior teeth for which there are no obvious etiologic factors should be also questioned regarding past general anesthesia or endotracheal intubation procedures.[3,127,138] In addition, patients should be questioned about a history of orthodontic treatment. Typically, when symptomatic irreversible pulpitis remains untreated, the pulp will eventually become necrotic.[109,139]

Asymptomatic Irreversible Pulpitis

This is a clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing.[6] The patient, however, does not complain of any symptoms. On occasion, deep caries will not produce any symptoms, even though clinically or radiographically the caries may extend well into the pulp. Left untreated, the tooth may become symptomatic or the pulp will become necrotic. In cases of *asymptomatic irreversible pulpitis,* endodontic treatment should be performed as soon as possible so that symptomatic irreversible pulpitis or necrosis does not develop and cause the patient severe pain and distress.

Pulp Necrosis

This is a clinical diagnostic category indicating death of the dental pulp. The pulp is usually nonresponsive to pulp testing.[6] When pulpal *necrosis* (or *nonvital pulp*) occurs, the pulpal blood supply is nonexistent and the pulpal nerves are nonfunctional. It is the only clinical classification that directly attempts to describe the histologic status of the pulp (or lack thereof). This condition is subsequent to symptomatic or asymptomatic irreversible pulpitis. After the pulp becomes completely necrotic, the tooth will typically become asymptomatic until such time when there is an extension of the disease process into the periradicular tissues. With pulp necrosis, the tooth will usually not respond to electric pulp tests or to cold stimulation. However, if heat is applied for an extended period of time, the tooth may respond to this stimulus. This response could possibly be related to remnants of fluid or gases in the pulp canal space expanding and extending into the periapical tissues.

Pulpal necrosis may be partial or complete and it may not involve all of the canals in a multirooted tooth. For this reason, the tooth may present with confusing symptoms. Pulp testing over one root may give no response, whereas over another root it may give a positive response. The tooth may also exhibit symptoms of symptomatic irreversible pulpitis. Pulp necrosis, in the absence of restorations, caries, or luxation injuries, is likely caused by a longitudinal fracture extending from the occlusal surface and into the pulp.[19]

After the pulp becomes necrotic, bacterial growth can be sustained within the canal. When this infection (or its bacterial byproducts) extends into the periodontal ligament space, the tooth may become symptomatic to percussion or exhibit spontaneous pain. Radiographic changes may occur, ranging from a thickening of the periodontal ligament space to the appearance of a periapical radiolucent lesion. The tooth may become hypersensitive to heat, even to the warmth of the oral cavity, and is often relieved by applications of cold. As previously discussed, this may be helpful in attempting to localize a necrotic tooth (i.e., by the application of cold one tooth at a time) when the pain is referred or not well localized.

Previously Treated

This is a clinical diagnostic category indicating that the tooth has been endodontically treated and the canals are obturated with various filling materials other than intracanal medicaments.[6] In this situation, the tooth may or may not present with signs or symptoms but will require additional nonsurgical or surgical endodontic procedures to retain the tooth. In most such situations, there will no longer be any vital or necrotic pulp tissue present to respond to pulp testing procedures.

Previously Initiated Therapy

This is a clinical diagnostic category indicating that the tooth has been previously treated by partial endodontic therapy (e.g., pulpotomy, pulpectomy).[6] In most instances, the partial endodontic therapy was performed as an emergency procedure for symptomatic or asymptomatic irreversible pulpitis cases. In other situations, these procedures may have been performed as part of vital pulp therapy procedures, traumatic tooth injuries, apexification, or apexogenesis therapy. At the time these cases present for root canal therapy it would not be possible to make an accurate pulpal diagnosis because all, or part, of the pulp tissue has already been removed.

Apical (Periapical) Disease

Normal Apical Tissues

This classification is the standard against which all of the other apical disease processes are compared. In this category the patient is asymptomatic and the tooth responds normally to percussion and palpation testing. The radiograph reveals an intact lamina dura and periodontal ligament space around all the root apices.

Periodontitis

This classification refers to an inflammation of the periodontium.[6] When located in the periapical tissues it is referred to as apical periodontitis. Apical periodontitis can be subclassified to symptomatic apical periodontitis and asymptomatic apical periodontitis.

Symptomatic Apical Periodontitis

This condition is defined as an inflammation, usually of the apical periodontium, producing clinical symptoms including a painful response to biting or percussion or palpation. It might or might not be associated with an apical radiolucent area.[6]

This tooth may or may not respond to pulp vitality tests, and the radiograph or image of the tooth will typically exhibit at least a widened periodontal ligament space and may or may not show an apical radiolucency associated with one or all of the roots.

Asymptomatic Apical Periodontitis

This condition is defined as inflammation and destruction of apical periodontium that is of pulpal origin, appears as an apical radiolucent area, and does not produce clinical symptoms.[6] This tooth does not usually respond to pulp vitality tests, and the radiograph or image of the tooth will exhibit an apical radiolucency. The tooth is generally not sensitive to biting pressure but may "feel different" to the patient on percussion. Manifestation of persistent apical periodontitis may vary among patients.[89]

Acute Apical Abscess

This condition is defined as an inflammatory reaction to pulpal infection and necrosis characterized by *rapid onset*, spontaneous pain, tenderness of the tooth to pressure, pus formation, and swelling of associated tissues.[6] A tooth with an *acute apical abscess* will be acutely painful to biting pressure, percussion, and palpation. This tooth will not respond to any pulp vitality tests and will exhibit varying degrees of mobility. The radiograph or image can exhibit anything from a widened periodontal ligament space to an apical radiolucency. Swelling will be present intraorally and the facial tissues adjacent to the tooth will almost always present with some degree of swelling. The patient will frequently be febrile, and the cervical and submandibular lymph nodes may exhibit tenderness to palpation.

Chronic Apical Abscess

This condition is defined as an inflammatory reaction to pulpal infection and necrosis characterized by *gradual onset*, little or no discomfort, and the intermittent discharge of pus through an associated sinus tract.[6] In general, a tooth with a *chronic apical abscess* will not present with clinical symptoms. The tooth will not respond to pulp vitality tests, and the radiograph or image will exhibit an apical radiolucency. Usually the tooth is not sensitive to biting pressure but can "feel different" to the patient on percussion. This entity is distinguished from asymptomatic apical periodontitis because it will exhibit intermittent drainage through an associated sinus tract.

REFERRED PAIN

The perception of pain in one part of the body that is distant from the actual source of the pain is known as *referred pain*. Whereas pain of nonodontogenic origin can refer pain to the teeth, teeth may also refer pain to other teeth as well as to other anatomic areas of the head and neck (see Chapters 4 and 17). This may create a diagnostic challenge, in that the patient may insist that the pain is from a certain tooth or even from an ear when, in fact, it is originating from a distant tooth with pulpal pathosis. Using electronic pulp testers, investigators found that patients could localize *which* tooth was being stimulated only 37.2% of the time and could narrow the location to three teeth only 79.5% of the time, illustrating that patients may have a difficult time discriminating the exact location of pulpal pain.[44]

Referred pain from a tooth is usually provoked by an intense stimulation of pulpal C fibers, the slow conducting nerves that when stimulated cause an intense, slow, dull pain. Anterior teeth seldom refer pain to other teeth or to opposite arches, whereas posterior teeth may refer pain to the opposite arch or to the periauricular area but seldom to the anterior teeth.[14] Mandibular posterior teeth tend to transmit referred pain to the periauricular area more often than maxillary posterior teeth. One study showed that when second molars were stimulated with an electric pulp tester, patients could discriminate accurately which arch the sensation was coming from only 85% of the time, compared with an accuracy level of 95% with first molars and 100% with anterior teeth.[136] The investigators also pointed out that when patients first feel the sensation of pain, they are more likely to accurately discriminate the origin of the pain. With higher levels of discomfort, patients have less ability to accurately determine the source of the pain. Therefore, in cases of diffuse or referred pain, the history of where the patient first felt the pain may be significant.

Because referred pain can complicate a dental diagnosis, the clinician must be sure to make an accurate diagnosis to protect the patient from unnecessary dental or medical treatment. If after all the testing procedures are complete and it is determined that the pain is not of odontogenic origin, then the patient should be referred to an orofacial pain clinic for further testing. For further information on pain of nonodontogenic origin, see Chapter 17.

SUMMARY

Endodontics is a multifaceted specialty, with much emphasis on how cases are clinically treated. Clinicians have increased their ability to more accurately perform endodontic procedures by way of increased visualization using the operating microscope, precise apical foramen detection using electronic apex locators, enhanced imaging techniques using digital radiography, and more. Practices have incorporated more refined canal cleaning and shaping techniques by using ultrasonics and rotary-driven nickel titanium files facilitated with computer-assisted electronic handpieces. Many other advancements have also been introduced with the objective of achieving an optimal result during endodontic treatment. However, these advancements are useless if an incorrect diagnosis is made. Before the clinician ever considers performing any endodontic treatment, the following questions must be answered:

- Is the existing problem of dental origin?
- Are the pulpal tissues within the tooth pathologically involved?
- Why is the pulpal pathosis present?
- What is the prognosis?
- What is the appropriate form of treatment?

Testing, questioning, and reasoning are combined to achieve an accurate diagnosis and to ultimately form an appropriate treatment plan. The art and science of making this diagnosis are the first steps that must be taken before initiating any endodontic treatment.

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CHAPTER 2

Radiographic Interpretation

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CHAPTER OUTLINE

Radiographic Interpretation
Imaging Modalities
Image Characteristics and Processing
Digital Imaging and Communications in Medicine (DICOM)
Diagnostic Tasks in Endodontics
Working Length Determination
Diagnosis and Healing
Three-Dimensional Imaging
Principles of Cone Beam Computed Tomography
Voxels and Voxel Sizes
Field of View
Imaging Tasks Improved or Simplified by Cone Beam Volumetric Computed Tomography
Differential Diagnosis
Diagnosis of Endodontic Treatment Failures
Evaluation of Anatomy and Complex Morphology
Intraoperative or Postoperative Assessment of Endodontic Treatment Complications
Materials Extending Beyond the Root Canal
Fractured Instruments
Calcified Canals
Perforations
Dentoalveolar Trauma
Internal and External Root Resorption
Presurgical Visualization
Outcomes Assessment
Implant Site Assessments
Image Perception and Viewing Environment
Future of CBCT
Conclusions

RADIOGRAPHIC INTERPRETATION

Interpretation of information captured by radiographic imaging modalities is central to the diagnostic process. It is very important to capture a diagnostically useful image using appropriate exposure parameters and view it with interactive manipulation of brightness and contrast or window/level (for cone beam computed tomography [CBCT] studies) in an optimal environment to adequately evaluate anatomy and diagnose pathoses. Accurate interpretation of root and canal morphology, determination of radiographic canal length, diagnosis of radicular and periradicular disease (Fig. 2-1), and postsurgical and long-term evaluation of the outcome of endodontic treatment are some of the routine diagnostic imaging tasks in endodontics.[181] Systematic and methodical interpretation processes must be followed for all images. Recognition of anatomy, anatomic variants, and pathologic conditions or deviations from normal is important. Various imaging modalities exist in radiology. Some use ionizing radiation, whereas others use ultrasonic waves (ultrasonography, or US) or powerful external magnetic fields (magnetic resonance imaging, or MRI). Interventional and noninterventional imaging modalities are also available. Imaging modalities using ionizing radiation are most frequently used in endodontic diagnoses. The different image capture modalities include conventional intraoral film and the more modern digital receptors.

Imaging Modalities

Digital radiography using electronic sensors or photostimulable phosphor (PSP) plates is widely used in endodontics. The advantages of using digital sensors over film are many. Significant advantages include noteworthy dose reduction (especially in comparison with D-speed film used with round collimation); almost instantaneous generation of high-resolution digital images with resolution approaching or equaling that of film for specific diagnostic tasks; the ability to postprocess images for enhanced diagnostic outcomes; elimination of variables associated with wet processing of conventional film; ease of transmission and of archiving and retrieving images from databases or picture archiving and communication systems (PACS); facilitation of use of an all-electronic patient record[123,188]; reduced exposure of personnel to hazardous chemicals; and reduced environmental impact.

Digital imaging modalities in endodontics use different image capture technologies, which include a charge-coupled device (CCD), a complementary metal oxide semiconductor (CMOS), or a PSP (also sometimes referred to as an indirect acquisition modality). Film images also can be digitized using a flatbed scanner or CCD/CMOS-based cameras mounted on a camera stand, with images captured using a frame grabber from a mounted, lighted platform.

CCD-based solid-state sensors were used extensively in endodontics initially. However, the earlier generation sensors had

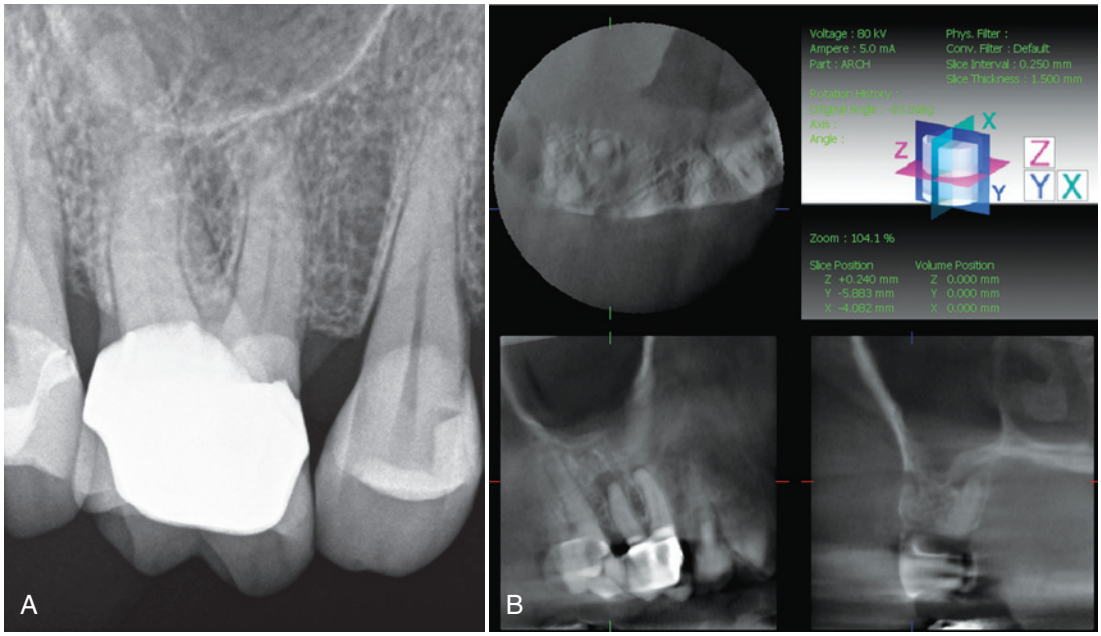

FIG. 2-1 A, A well-angulated periapical radiograph of the maxillary right first molar taken during a diagnostic appointment for endodontic evaluation of the maxillary right quadrant. At first glance, there is little radiographic evidence of significant or periradicular change. B, Contemporaneous CBCT image of same tooth gives an entirely different perspective; periapical changes are visible on all three roots in all three anatomic planes of section. (*B taken* with J. Morita Veraviewepocs 3D [J. Morita, Osaka, Japan]).

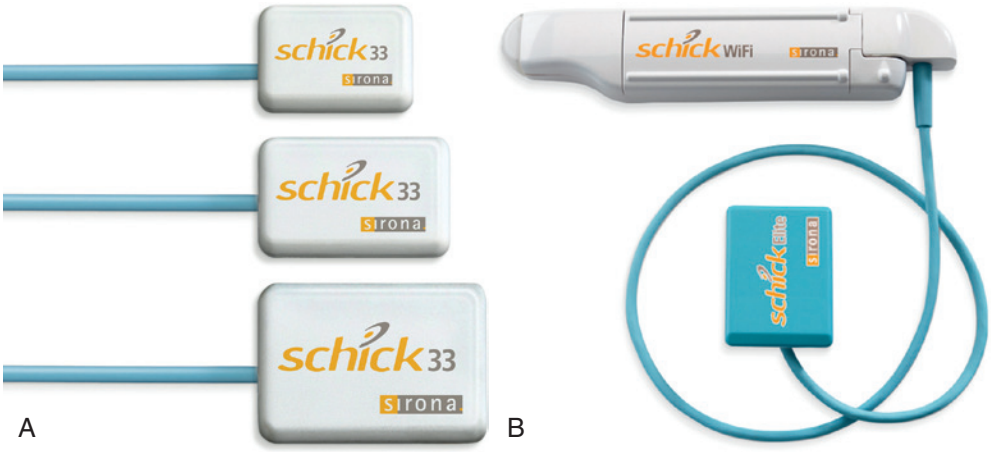

FIG. 2-2 A and B, High-resolution complementary metal oxide semiconductor (CMOS) sensors are available from many manufacturers. Note that Figure B shows wireless CMOS sensors transmit images to the chairside workstation by 2.4 GHz radio frequency. (Courtesy SIRONA DENTAL SYSTEMS, Long Island City, NY.)

a smaller active area and limited x-ray absorption and conversion efficiency, in addition to being bulky. Sensors use an array of radiation-sensitive elements that generate electric charges proportionate to the amount of incident radiation. To reduce the amount of radiation needed to capture an image, a light-sensitive array was developed that uses a scintillation layer laid on top of the CCD chip or added with a fiberoptic coupling. The generated charge is read out in a "bucket brigade" fashion and transferred to an analog-to-digital converter in the frame grabber assembly of the workstation. The digital information is processed, and an image is formed. CMOS-based sensors, on the other hand, have an active transistor at each element location. The area available for signal generation is relatively less, and there is a fixed pattern noise. These sensors are less expensive to manufacture and have been shown to be equally useful for specific diagnostic tasks.[4] Unlike the CCD, the CMOS chip requires very little electrical energy; therefore, no external power supply is needed to support USB utilization, and wireless applications are feasible. Wireless sensors are available (Fig. 2-2). However, radiofrequency interference may be a problem with these sensors. The current WiFi sensor is less bulky and has a wire attached to it that enables transmission via 802.11 b/g standard. It uses a lithium-ion polymer battery that can last for approximately 100 exposures.

Yet another type of sensor uses PSPs for image capture. PSP technology is also referred to as *computed radiography* (CR).[94,169]

Unlike the CCD and CMOS sensors, PSP sensors are wireless. The phosphor is activated by a process called *doping*, which enables charges to be generated and stored when exposed to radiation. A latent image is stored in the sensor, and a PSP reader with a laser beam of specific wavelength is used to read out the image. Previously captured images can be erased by exposing the PSP sensor to white light. PSP plates can be damaged easily by scratching, but they are not as expensive as CCD or CMOS sensors. Incomplete erasure of the image can lead to ghost images when the plate is reused, and delayed processing can result in a decrease in image clarity.[2] PSP-based sensors are used in high-volume scenarios. Spatial resolution is lower with this type of sensor, but it has a wider dynamic range. These sensors can tolerate a wider range of exposures to produce a diagnostically useful image.

Radiation dose continues to be a concern with all imaging studies. The lowest possible dose must be delivered for each study. Most dental offices would not be in compliance with the latest recommendations of the National Council for Radiation Protection (NCRP) on reducing the radiation dose from intraoral radiographs (Box 2-1). Two terms have been specifically defined in the NCRP's report. The terms *shall* and *shall not* indicate that adherence to the recommendation would be in compliance with the standards of radiation safety. The terms *should* and *should not* indicate prudent practice and acknowledge that exceptions may be made in certain circumstances. In addition, the report establishes nine new recommendations for image processing of conventional film.

A strong argument can be made for clinicians to switch to a direct digital radiography (DDR) system to avoid all the drastic changes necessary to ensure compliance with the new recommendations. Even though restriction of an intraoral dental x-ray beam is mandated by federal law to a circle no greater than 7 cm, rectangular collimation has been proven to significantly reduce the radiation dose to the patient.

The American Dental Association (ADA) Council on Scientific Affairs has made the following statement:

> Tissue area exposed to the primary x-ray beam should not exceed the minimum coverage consistent with meeting diagnostic requirements and clinical feasibility. For periapical and bitewing radiography, rectangular collimation should be used whenever possible because a round field beam used with a rectangular image receptor produces … unnecessary radiation exposure to the patient.[1]

BOX 2-1

Recommendations of the National Council on Radiation Protection

1. Dentists must examine their patients before ordering or prescribing x-ray images (this is not a new guideline).
2. The use of leaded aprons on patients shall not be required if all other recommendations in this report are rigorously followed (read full Report #145).
3. Thyroid shielding shall be used for children and should be provided for adults when it will not interfere with the examination (e.g., panoramic imaging).
4. Rectangular collimation of the beam, which has been recommended for years, shall be routinely used for periapical radiographs. Each dimension of the beam, measured in the plane of the image receptor, should not exceed the dimension of the image receptor by more than 2% of the source-to-image receptor distance. Similar collimation should be used, when feasible, for bitewing radiographs.
5. Image receptors of speeds slower than ANSI speed Group E films shall not be used for intraoral radiography. Faster receptors should be evaluated and adopted if found acceptable. For extraoral radiography, high-speed (400 or greater) rare earth screen-film systems or digital-imaging systems of equivalent or greater speed shall be used.
6. Dental radiographic films shall be developed according to the film manufacturer's instructions using the time-temperature method. In practical application, this means that sight development (reading wet x-ray films at the time of the procedure) shall not be used.
7. Radiographic techniques for digital imaging shall be adjusted for the minimum patient dose required to produce a signal-to-noise ratio sufficient to provide image quality to meet the purpose of the examination.
8. Clinicians designing new offices or remodeling existing locations will need shield protection to be provided by a qualified expert.

Modified from the National Council on Radiation Protection and Measurements: Radiation protection in dentistry, Report #145, Bethesda, Md, 2003. Available at: www.ncrppublications.org/Reports/145.

Image Characteristics and Processing

Spatial resolution achieved with current generation digital sensors is equally good or better than that of conventional intraoral radiographic film. Intraoral film has a resolution of 16 line pairs per millimeter (lp/mm) as measured using a resolution tool, and it increases to 20 to 24 lp/mm with magnification. *Spatial resolution* is defined as the ability to display two objects that are close to each other as two separate entities. *Contrast resolution* is defined as the ability to differentiate between areas on the image based on density. Most diagnostic tasks in endodontics require a high-contrast resolution.[121] However, image quality is not just a function of spatial resolution. The choice of appropriate exposure parameters, sensor properties, the image processing used, and viewing conditions and modalities directly affect diagnostic accuracy.

Postprocessing of images may be carried out to alter image characteristics. Radiographs need not be reexposed if image quality is not adequate. Diagnostic information can be teased out of the image if appropriate image processing is used. However, the original image must be acquired with optimal exposure parameters to accomplish meaningful image processing.[178] Suboptimally exposed images cannot be processed to yield diagnostic information, which may lead to a reduction in the diagnostic accuracy of the image. Image enhancement must be task specific. Signal-to-noise ratio (SNR) must be optimized to extract necessary information from the image. The bit-depth of images also has a direct relationship to image quality. It indicates the number of shades of gray that the sensor can capture for display. For example, an 8-bit image can depict 256 shades of gray. Most sensors are 12 or 14 bits in depth, capturing 4,096 or 65,536 shades of gray, respectively. If the sensor captures several thousand shades of gray, the image can be manipulated through enhancement techniques to display those shades of gray that best depict the anatomy of interest. The human visual system is limited in the number of shades of gray that can be read at any point in time. Therefore, image

enhancement is a must for all images, so as to delineate signals of interest through manipulation of the grayscale. Most endodontic tasks require a high contrast and thus a shorter grayscale.

Digital radiographs can be saved in different file formats. Several file formats are available: DICOM (*D*igital *I*maging and *C*ommunications in *M*edicine); tiff (*t*agged *i*mage *f*ile *f*ormat); jpeg (*j*oint *p*hotographic *e*xperts *g*roup); gif (*g*raphics *i*nterchange *f*ormat); BMP (Windows' bitmap image file); PNG (*p*ortable *n*etwork *g*raphics); and so on. There also are several proprietary formats. "Lossy" and "lossless" compression schemes can be used for saving images, although lossless compression is preferred.[58]

Digital Imaging and Communications in Medicine (DICOM)

DICOM is a set of international standards established in 1985 by the American College of Radiology (ACR) and the National Electrical Manufacturers Association (NEMA)[46,175] to address the issue of vendor-independent data formats and data transfers for digital medical images.[77] The ADA has promoted the interoperability of dental images through the efforts of its Working Group 12.1.26. DICOM serves as a standard for the transferal of radiologic images and other medical information between computers, allowing digital communication between systems from various manufacturers and across different platforms (e.g., Apple iOS or Microsoft Windows).[78] The DICOM standard provides for several hundred attribute fields in the record header, which contains information about the image (e.g., pixel density, dimensions, and number of bits per pixel), in addition to relevant patient data and medical information. Although earlier versions did not specify the exact order and definition of the header fields, each vendor is required to publish a DICOM conformance statement, which gives the location of pertinent data. The big hurdle is to support medical and dental consultations between two or more locations with different imaging software. With DICOM in place, dental clinicians can change vendors and maintain database interoperability. Most software vendors are striving to achieve full DICOM compliance, and some have achieved at least partial compliance. However, proprietary DICOM images are still produced in different systems, with the capability to export in universal DICOM format as needed. Diagnostic images are best saved as DICOM files to preserve image fidelity or as tiff files with no compression. Diagnosis suffers when images undergo lossy compression.[50,103,180]

Based on the DICOM model, the ADA Standards Committee on Dental Informatics has identified four basic goals for electronic standards in dentistry: (1) interoperability, (2) electronic health record design, (3) clinical workstation architecture, and (4) electronic dissemination of dental information.[7] The dental profession must continue to promote DICOM compatibility so that proprietary software and file types do not hinder communication and risk making data obsolete.

DIAGNOSTIC TASKS IN ENDODONTICS

Working Length Determination

Digital imaging systems perform as well as intraoral film or better for working length determination.[121] No significant difference was noted between measurements made on digital images.[99] Older studies compared the early generation digital sensors with limited bit-depth to D-speed films, and the films showed better performance. It is important to analyze the type of sensor used, software, processing, video card and monitor, and viewing conditions to determine whether the sensor is good for a specific diagnostic task. Calibration improves the diagnostic accuracy.[108] Likewise, the use of optimal processing parameters improves image quality to the extent of making a significant difference in the diagnostic outcome. For instance, density plot analysis was shown to help with endodontic file measurements.[146] The major advantage of direct digital radiography (CCD, CMOS) is that the dose is significantly less compared with that required for film. The use of DDR, therefore, is justified when its performance is comparable to that of film with no statistically significant differences.[101]

The three types of measurement generally available with digital imaging software are (1) linear measurement, the distance between two points in millimeters (Fig. 2-3); (2) angle measurement, the angle between two lines; and (3) area measurement, the area of the image or a segment of the image. Because magnification and distortion errors play a significant role in the accuracy of two-dimensional (2D) radiographic measurement, both film and digital systems are subject to parallax error. However, a study that compared endodontic file length images of human teeth taken with a custom jig suggested that "measurement error was significantly less for the digital images than the film-based images."[49] This was true even though, as the authors pointed out, the measurement differences may not have been clinically significant. Sophisticated calibration algorithms are under development, and accurate measurement of parallel images should be more feasible in the future.[30]

Diagnosis and Healing

Image enhancement of direct and indirect digital radiographs based on the diagnostic task at hand has been shown to increase diagnostic accuracy compared to film-based images, which cannot be enhanced.[2,190] Posttreatment endodontic evaluation of healing of apical radiolucent areas is a challenge. Early changes indicating healing and bone fill are difficult to detect on conventional or digital radiographs. However, bone fill can be detected using more sensitive techniques, such as digital subtraction radiography, in which two images, separated in time but acquired with the exact same projection geometry and technique factors, can be subtracted from one another to tease out subtle changes in the periodontium and surrounding bone. Subtraction techniques are difficult to carry out in routine clinical practice because they are technique sensitive and can yield incorrect information if not performed accurately. Several studies have shown the usefulness of subtraction radiography using digital sensors.[117,129,194]

Three-Dimensional Imaging

Computed tomography (CT) was introduced by Sir Godfrey Hounsfield in the 1970s. *Tomography* refers to "slice imaging," in which thin slices of the anatomy of interest are captured and synthesized manually or using an algorithm. CT makes use of automated reconstruction. Medical-grade CT used a translate-rotate image acquisition scheme as the technology developed, but the modality always resulted in higher radiation dose delivery because of redundancy of data capture, in addition to longer scan times with the potential for motion artifact.

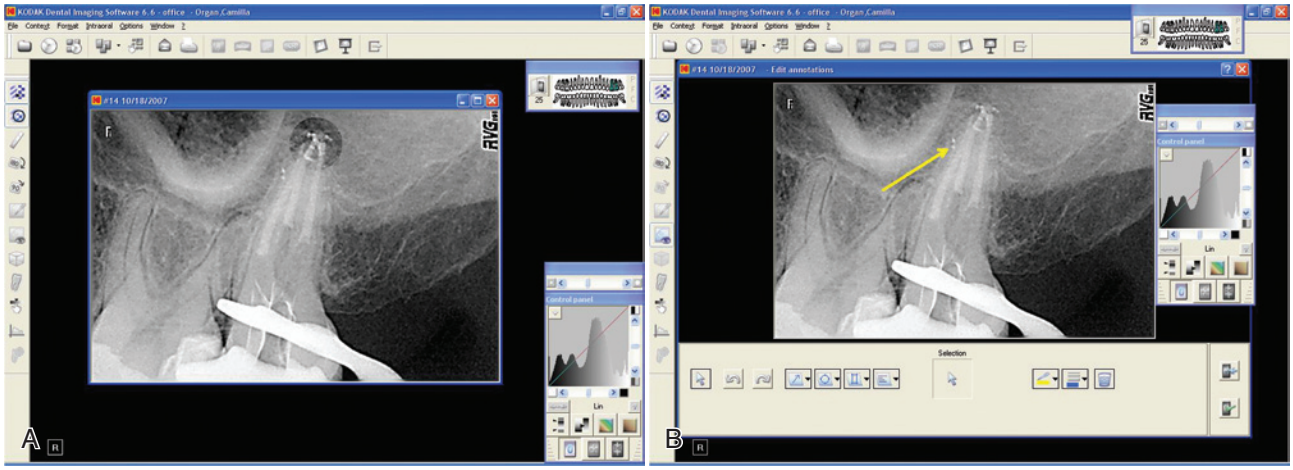

FIG. 2-3 A, Certain regions of interest (ROI) can be highlighted with a preset contrast tool that can be moved around the image. B, Preprogramed filters that enhance sharpness and contrast can be selected to optimize the image acquired. (Courtesy Carestream Dental LLC, Atlanta, GA.)

Multiple detectors and x-ray sources were used in later generations of CT units to reduce scanning times. The increased radiation dose, artifacts from metallic restorations, cost of scanning, long acquisition times, and lack of adequate dental-specific software have been drawbacks limiting the use of the technology in dentistry until recently. The advent of cone beam volumetric computed tomography (CBVCT) introduced a faster, low-dose, low-cost, high-contrast imaging modality that could capture information in three dimensions using a limited field of view.

CBVCT, or cone beam CT (CBCT), is a relatively new diagnostic imaging modality that has been recently added to the endodontic imaging armamentarium. This modality uses a cone beam instead of a fan-shaped beam in multidetector computed tomography (MDCT), acquiring images of the entire volume as it rotates around the anatomy of interest. Compared with MDCT images, CBCT offers relatively high-resolution, isotropic images, allowing effective evaluation of root canal morphology and other subtle changes within the root canal system. Even though the resolution is not as high as that of conventional radiographs (18 microns), the availability of three-dimensional (3D) information, the relatively higher resolution, and a significantly lower dose compared to MDCT make CBCT the imaging modality of choice in challenging situations demanding localization and characterization of root canals.

The adoption of advanced imaging modalities such as CBCT for select diagnostic tasks is becoming popular with clinicians performing endodontic procedures. Two-dimensional gray-scale images, whether conventional film based or digital, cannot accurately depict the full 3D representation of the teeth and supporting structures. In fact, traditional images are poor representations of even the pulpal anatomy. They grossly underestimate canal structure and often cannot accurately visualize periapical changes, especially where there is thick cortical bone, as in the presence of anatomic obstructions (Fig. 2-4, *A*). CBCT, however, allows the clinician to view the tooth and pulpal structures in thin slices in all three anatomic planes: axial, sagittal, and coronal. This capability alone allows visualization of periapical pathoses and root morphology previously impossible to assess (Fig. 2-4, *B* and *C*). Several tools available in CBCT, such as the ability to change the vertical or horizontal angulation of the image in real time, in addition to thin-slice, grayscale data of varying thicknesses, will never be available for conventional or even digital radiography. Furthermore, the use of CBCT data to view the region of interest in three anatomic planes of section at very low x-ray doses has never been as easy or accessible as it is today.

Microcomputed tomography (micro-CT) has also been evaluated in endodontic imaging.[87,144,145] Comparison of the effects of biomechanical preparation on the canal volume of reconstructed root canals in extracted teeth using micro-CT data was shown to assist with characterization of morphologic changes associated with these techniques.[145] Peters et al.[144] used micro-CT to evaluate the relative performance of nickel-titanium (Ni-Ti) instruments after the shaping of root canals of varying preoperative canal geometry (for examples, see Chapter 6). A study to examine the potential and accuracy of micro-CT for imaging of filled root canals showed it to be a highly accurate and nondestructive method for the evaluation of root canal fillings and its constituents. The qualitative and quantitative correlations between histologic and micro-CT examination of root canal fillings were high.[87] However, it is important to note that micro-CT remains a research tool and cannot be used for human imaging in vivo.

This chapter discusses the principles, applications, imaging attributes, image artifacts, and potential liability of adopting CBCT technology for endodontic procedures. Given this information, the student of endodontics will begin to realize the significant advantages, limitations, and diagnostic and treatment planning capabilities of this radiographic imaging modality.

PRINCIPLES OF CONE BEAM COMPUTED TOMOGRAPHY

Three important parameters of cone beam imaging are described in the following sections:

- Voxel size
- Field of view (FOV)
- Slice thickness/measurement accuracy

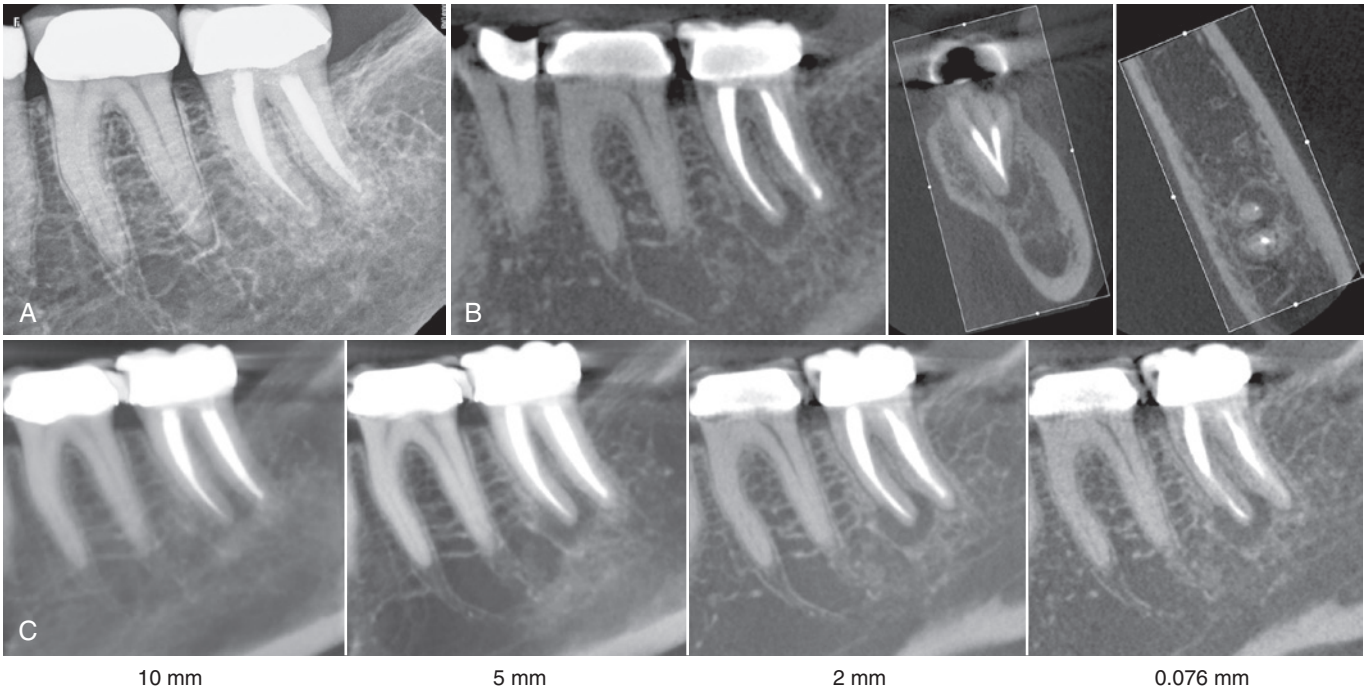

FIG. 2-4 This case demonstrates the difficulty in assessing lesions in the mandibular posterior region when there is a dense cortex. This well-angulated periapical radiograph **(A)** does not clearly show any radiolucencies associated with the mandibular left second molar, for which the patient has presented for evaluation and possible retreatment. The CBCT corrected sagittal, coronal, and axial reconstructed multiplanar views (**B**, *left to right,* sagittal, coronal, and axial views) show previous endodontic treatment, with a 6-mm diameter radiolucency with a well-defined, mildly corticated border, centered over a point on the buccal aspect of the root, 2 mm coronal to the apex; these are features consistent with an apical rarefying osteitis. The ray sum images of the sagittal view, where the image is "thinned" by decreasing the number of adjacent voxels using postprocessing software, simulates a curvilinear projection, showing diminishing superimposition (**C**, *left to right,* image layer of 10 mm, 5 mm, 2 mm, and 0.076 mm). (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

Voxels and Voxel Sizes

Voxels are cuboidal elements that constitute a 3D volume, unlike pixels, which are 2D. Data are acquired and represented in three dimensions using voxels. Unlike with medical computed tomography (MDCT), cone beam units acquire x-ray information using low kV and low mA exposure parameters in a single pass from 180 to 360 degrees of rotation around the anatomy of interest. Medical scanners use higher voltages of 120 kV or more and current of about 400 mA. Several units used in maxillofacial imaging use significantly lower exposure parameters (Figs. 2-5 to 2-7). The x-ray dose for all cone beam units is significantly lower than the dose received from a MDCT unit. Image attributes are also different in that volumes are reconstructed from isotropic voxels; that is, the images are constructed from volumetric detector elements that are cubical in nature and have the same dimensions of length, width, and depth. These voxel sizes can be as small as 0.076 to 0.6 mm.[118] By comparison, MDCT slice data are 0.5 mm to 1 cm thick. Fig. 2-8 illustrates the difference between a pixel and a voxel, the difference between an anisotropic pixel of MDCT and an isotropic pixel (voxel) of CBCT, and how the pixel data are acquired from both modalities.

The patient is positioned on a gantry in an MDCT unit, and images are acquired multiple slices at a time, which

FIG. 2-5 i-CAT unit. (Courtesy Imaging Sciences International, Hatfield, Pa.)

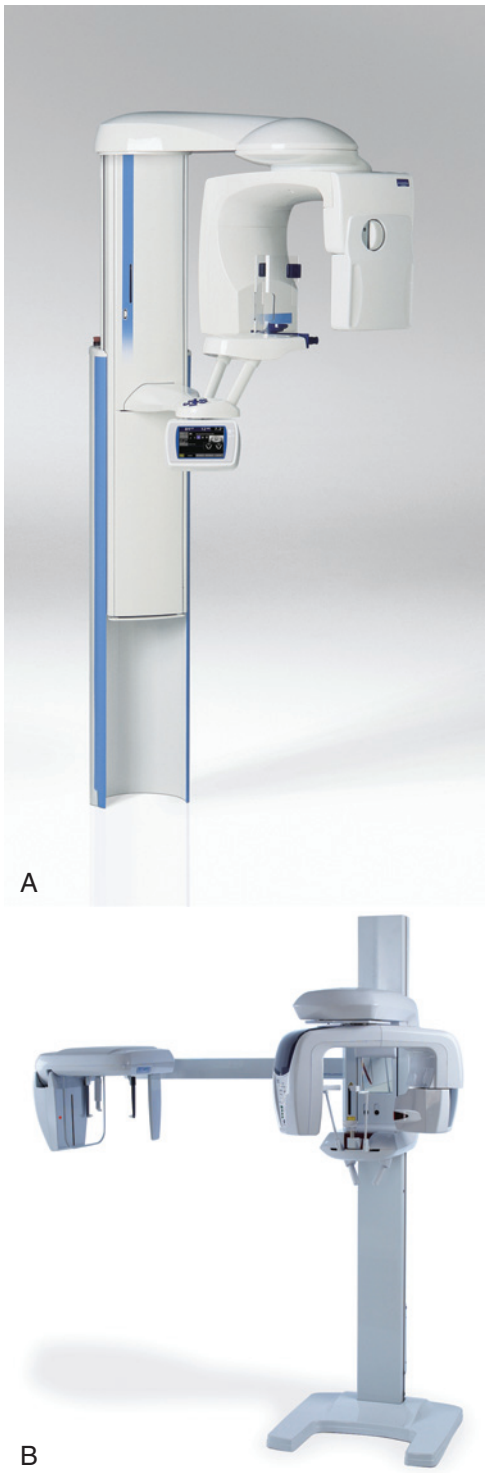

FIG. 2-6 **A**, Planmeca ProMax 3D. **B**, J. Morita Veraviewepocs 3D. (**A** courtesy Planmeca Oy, Helsinki, Finland; **B** courtesy J. Morita Corp, Osaka, Japan.)

prolongs the acquisition time. The number of slices acquired is a direct function of the sensor array configuration. Spiral CT uses continuous translator motion of the gantry as images are acquired, thus shortening the acquisition time. This results in significantly higher absorbed x-ray doses for the patient. A typical CBCT examination would expose the patient to only about 20 to 500 μSv in a single study, whereas a typical medical examination of the head would approach 2100 μSv[2] because the image data are gathered one section at a time. Therefore, soft tissue imaging is better with MDCT because the signal intensity is higher. However, this is not a requirement for dental diagnostic tasks because hard tissue visualization is more important. Consequently, CBCT data have a much higher resolution than MDCT data for hard tissue visualization because of the smaller voxel sizes that medical-grade scanners are incapable of achieving at a significantly lower dose. Increased noise is observed as a result of volumetric acquisition, but the SNR is maintained at a desirable level that facilitates adequate diagnosis based on hard tissue signals.

Field of View

The field of view (FOV) (Figs. 2-9 and 2-10) ranges from as small as a portion of a dental arch to an area as large as the entire head. The selection of the FOV depends on several factors. Among the most important are the following:

- Diagnostic task
- Type of patient
- Spatial resolution requirements

Diagnostic Task

The diagnostic task is the single most important determinant of the FOV in any imaging study. Based on the outcome of the clinical assessment, history, and evaluation of previous and other available imaging studies, a segment of the jaw or a larger area may need to be imaged using an appropriate FOV. If systemic conditions or generalized disorders are suspected, a larger FOV is sometimes required. For most endodontic purposes, a limited FOV can be used, if no signs or symptoms of systemic conditions are reported or noted. Under no circumstances should a screening study be done using a large FOV in the absence of signs and symptoms justifying the procedure. Several multifunctional cone beam units are available that allow the clinician to acquire several image types. Image quality has a direct impact on the diagnostic outcome; therefore, the choice of an FOV should be made carefully. Figure 2-11 illustrates the advantages of using multiple image types for an endodontic case.

Additional benefits of CBCT imaging software include allowing the clinician to format the volume to generate an image that looks like a panoramic radiograph. Conventional panoramic machines, although not commonly used by endodontists, use the focal trough, or zone of sharpness, to position patients so as to minimize distortion along multiple axes. All inherent problems associated with panoramic imaging, including distortion, magnification, blurring, ghost shadows, and other artifacts, can be expected on the resulting image if patient positioning is not accurate. With CBCT, such artifacts are not generated, resulting in a distortion-free panoramic reconstruction (Fig. 2-12). However, it must be noted that CBCTs should not be generated in patients requiring a panoramic radiograph alone, because of dose concerns.

Newer hybrid units, such as the CS 9300 3D Extraoral Imaging System (Carestream Dental, Atlanta, Georgia), have a wide range of FOV choices for a variety of diagnostic tasks, in addition to a conventional panoramic imaging option

FIG. 2-7 A, CS 9000 3D and CS 8100 Extraoral imaging systems. B, Morita Accu-i-tomo 170. (A Courtesy Carestream Dental LLC, Atlanta, GA; B Courtesy J Morita, Irvine, CA.)

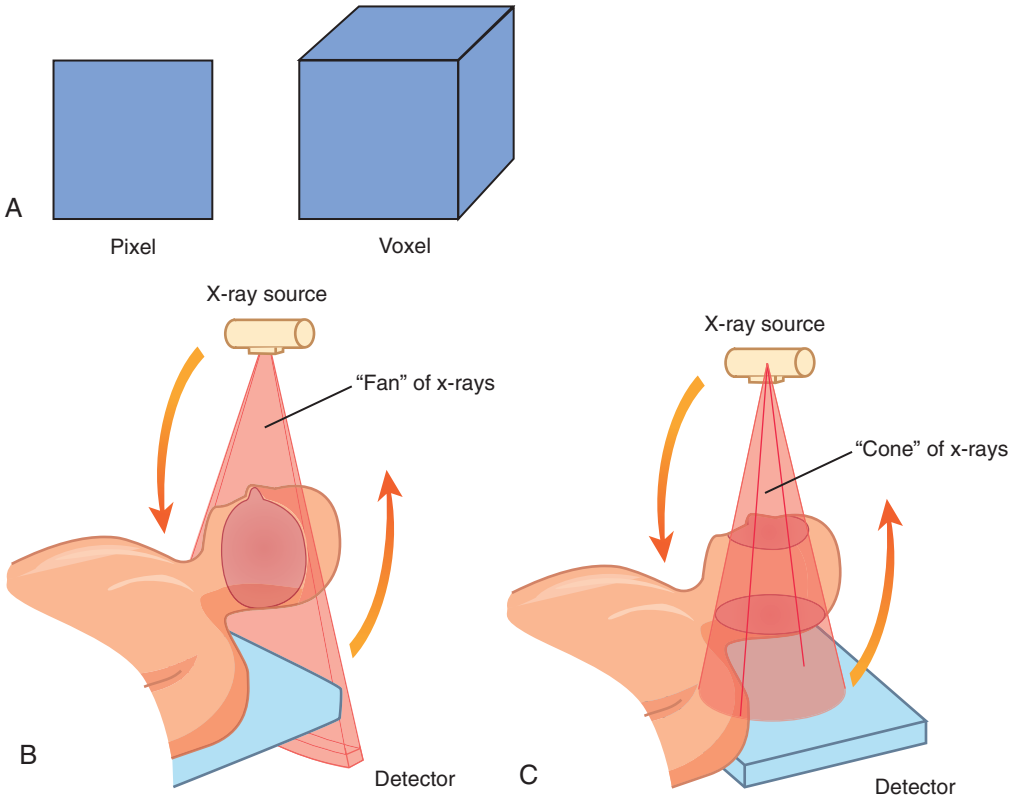

FIG. 2-8 *Left,* Drawing represents a *pixel* (picture element), the image capture and display element of any traditional digital image displayed on the computer. Shades of gray or color are displayed in these pixels to represent a 2D image. *Right,* Drawing represents a *voxel* (volume element). Voxels in CBCT are isometric and have the same dimension or length on all sides. They are very small (from 0.076 to 0.60 microns) and are the capture elements for cone beam imaging devices. Principles of conventional fan beam and cone beam computed tomography are presented in (B) and (C), respectively. (B and C from Babbush CA: *Dental implants: the art and science,* ed 2, St Louis, 2011, Elsevier/Saunders.)

(Fig. 2-13). The CS 9000 unit offers the lowest voxel size of 76 microns, whereas the CS 9300 can resolve down to 90 microns, with a range extending to 500 microns for larger FOV studies. Likewise, the Morita 3D Accuitomo 80 (J. Morita USA, Irvine, California) generates isotropic voxels of 80 microns. Although not necessary for use in every case, this technology, when appropriate, improves visualization and ultimately leads to better care in select situations. A record of exposure and doses must be maintained for each patient.

Type of Patient

Patient size and thus the amount of regional anatomy captured in the study also help determine the FOV. The smallest possible FOV must be chosen for the task at hand. Just because a

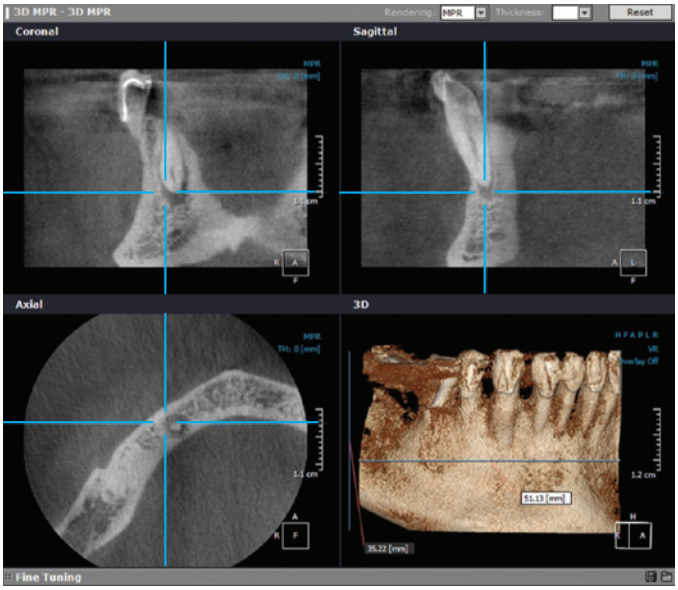

FIG. 2-9 Multiplanar and 3D color reconstructed views of the mandibular quadrant taken on a CBCT machine with a volume size of 37 × 50 mm. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

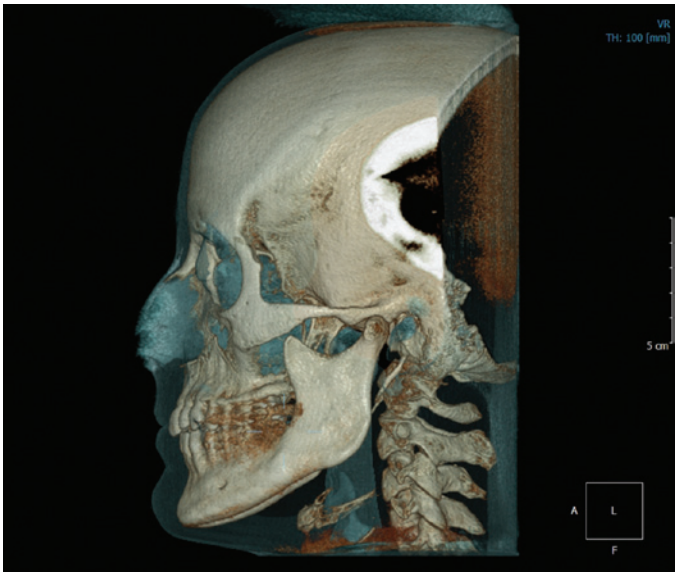

FIG. 2-10 Image of entire head (17 × 23 cm) from a large FoV machine. (Image acquired with i-CAT unit [Imaging Sciences International, Hatfield, Pa].)

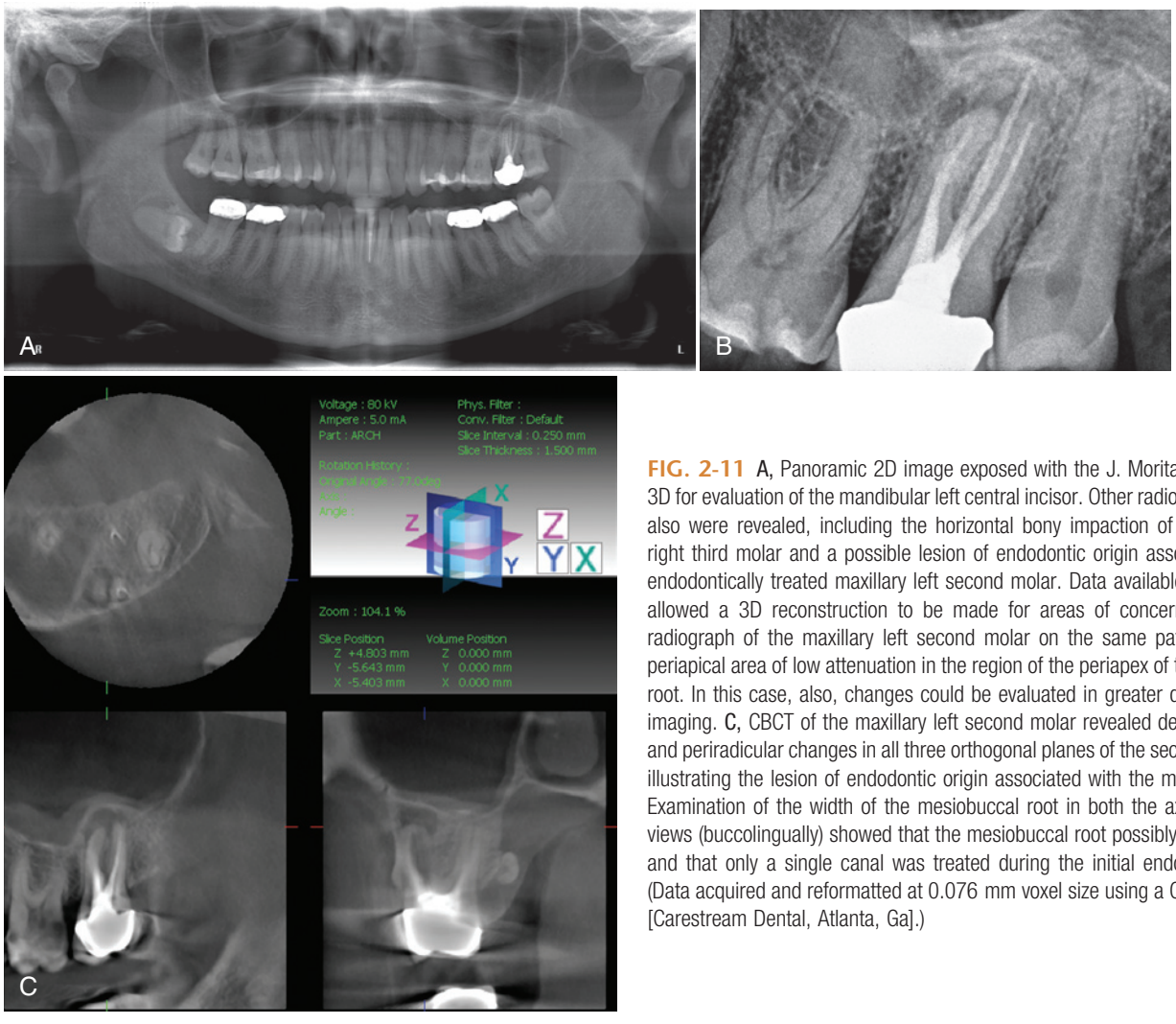

FIG. 2-11 A, Panoramic 2D image exposed with the J. Morita Veraviewepocs 3D for evaluation of the mandibular left central incisor. Other radiographic findings also were revealed, including the horizontal bony impaction of the mandibular right third molar and a possible lesion of endodontic origin associated with the endodontically treated maxillary left second molar. Data available from the scan allowed a 3D reconstruction to be made for areas of concern. B, Periapical radiograph of the maxillary left second molar on the same patient revealed a periapical area of low attenuation in the region of the periapex of the mesiobuccal root. In this case, also, changes could be evaluated in greater detail with CBCT imaging. C, CBCT of the maxillary left second molar revealed detailed periapical and periradicular changes in all three orthogonal planes of the section, specifically illustrating the lesion of endodontic origin associated with the mesiobuccal root. Examination of the width of the mesiobuccal root in both the axial and coronal views (buccolingually) showed that the mesiobuccal root possibly had two canals and that only a single canal was treated during the initial endodontic therapy. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

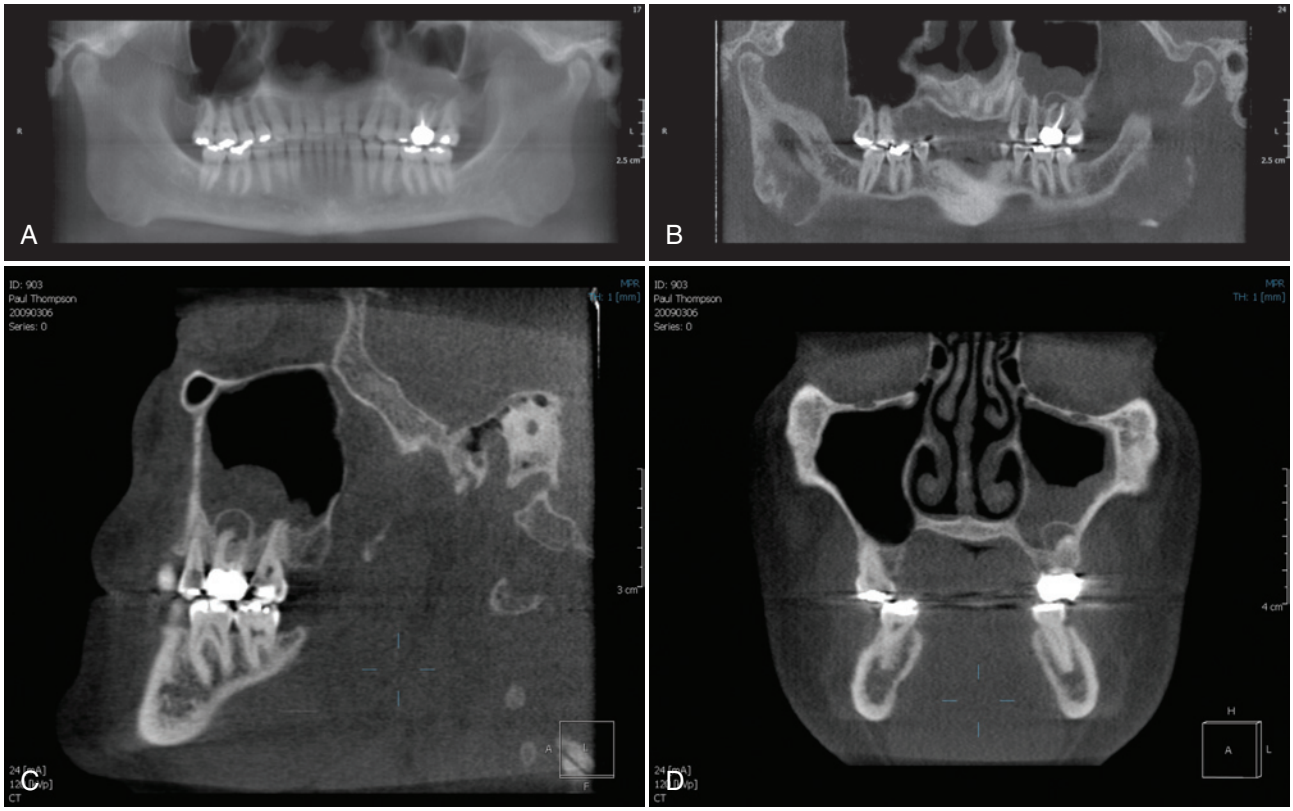

FIG. 2-12 A, This reconstructed panoramic image from CBCT data approximates the view one would see with a conventional panoramic radiograph. It is somewhat difficult to see the lesion on the maxillary left first molar. B, This thin-slice pseudopanoramic image shows the lesion more precisely because the thin slice (0.10 mm) removes most of the anatomic superimposition. These slices of the maxillary left first molar show the lesion in sagittal (C) and coronal (D) views, confirming the features seen in the pseudopanoramic view.

FIG. 2-13 CS 9300 3D Extraoral Imaging System. (Courtesy Carestream Dental LLC, Atlanta, GA.)

clinician owns a cone beam machine does *not* mean that every patient should be exposed to a cone beam study.[119] If previous studies are available, they need to be evaluated first in a recall patient. Use of imaging in children must be minimized. Cone beam machines with smaller FOVs can somewhat limit the radiation dose to critical organs and tissues of the head and neck in these cases.

Spatial Resolution Requirements

All endodontic imaging procedures require high spatial resolution. Assessment of canal structure, canal length, and lesions of endodontic origin (LEOs)[160] showing apical change, in addition to an understanding of possible revision cases, are important tasks requiring minute detail. If CBCT is used, the data acquisition should be performed at the smallest voxel size: the smaller the voxel size, the higher the spatial resolution. Many of the larger stand-alone cone beam machines, such as the i-CAT (Imaging Sciences International, Irvine, California), default to a 0.4 mm voxel size. This voxel size is inadequate for high spatial detail. However, these units often have a voxel size selection option that allows smaller voxel sizes to be used during the image acquisition. The absolute maximum voxel size for endodontic imaging should be 0.2 mm.[35] Units typically use voxel sizes of 0.076 to 0.16 mm for their native image capture (Fig. 2-14).

FIG. 2-14 A, Axial slice from data obtained with a 0.4 mm voxel size. Compare the trabecular pattern and outline of the mental foramina to the same location in an axial slice from data obtained with a 0.16 mm voxel size (B).

IMAGING TASKS IMPROVED OR SIMPLIFIED BY CONE BEAM VOLUMETRIC COMPUTED TOMOGRAPHY

The Executive Opinion of the American Academy of Oral and Maxillofacial Radiology and, later, the position paper on the use of CBCT in endodontics jointly developed by the American Association of Endodontists (AAE) and the American Academy of Oral and Maxillofacial Radiology (AAOMR), list indications for potential use in selected cases, including evaluation of the anatomy and complex morphology, differential diagnosis of complex pathoses with certain qualifiers, intraoperative or postoperative assessment of endodontic treatment, dentoalveolar trauma, resorption, presurgical case planning, and dental implant case planning.[34] Use of CBCT must be determined on a case-by-case basis only. These indications do not in any way mandate the use of CBCT for every case that falls into one of the preceding categories. For endodontic treatment and assessments, there are at least five primary imaging tasks in which CBCT scans have a distinct advantage over traditional 2D radiographs. These tasks include evaluation of the following factors:

1. Differential diagnosis
   a. Lesions of endodontic origin
   b. Lesions of nonendodontic origin
   c. Diagnosis of endodontic treatment failures
   d. Vertical root fractures
2. Evaluation of anatomy and complex morphology
   a. Anomalies
   b. Root canal system morphology
3. Intraoperative or postoperative assessment of endodontic treatment complications
   a. Overextended root canal obturation material
   b. Separated endodontic instruments
   c. Calcified canal identification
   d. Localization of perforation
4. Dentoalveolar trauma
5. Internal and external root resorption
6. Presurgical case planning
7. Dental implant case planning
8. Assessment of endodontic treatment outcomes

Differential Diagnosis

Lesions of Endodontic Origin

Clinical endodontic diagnosis relies on subjective and objective information collected during patient examinations. Diagnosis of the pulpal status of the teeth can sometimes be challenging if adequate radiographic information is unavailable. It is fundamental to understand that lesions of endodontic origin arise secondary to pulpal breakdown products and form adjacent to canal portals of exit.[155,161] These radiolucent lesions, formed as a result of loss of bone mineralization, can and do form three dimensionally anywhere along the root surface anatomy.[154] A 30% to 40% mineral content loss is needed for these lesions to be visualized on conventional radiographs.[116] Furthermore, the thickness of the cortical plate covering the lesion may significantly affect the radiographic appearance of the lesion on a conventional image.[192] In a comparative investigation of the use of CBCT and periapical (PA) radiography in the evaluation of the periodontal ligament (PDL), Pope et al.[148a] showed that necrotic teeth examined with CBCT had widened PDLs, but healthy, vital teeth showed significant variation. They called for further investigation to determine whether health and disease can be appropriately judged by the use of CBCT in epidemiologic investigations.

Digital subtraction radiography (DSR) has been observed to increase diagnostic capability; observers identified incipient periapical lesions in more than 70% of the cases.[116] Before the advent of CBCT, clinicians were unable to routinely visualize the presence, specific location, and extensiveness of periapical bone loss using conventional radiography.[107] This was especially true in areas with superimposition of anatomic structures. Visual obstruction from anatomic features, such as buccal bone and the malar process over the apices of maxillary roots, simply "disappears" when the examiner can scroll through the slices of the bone from facial to palatal in 0.1 mm sections while also changing axial orientations. CBCT showed significantly higher rates of detection of

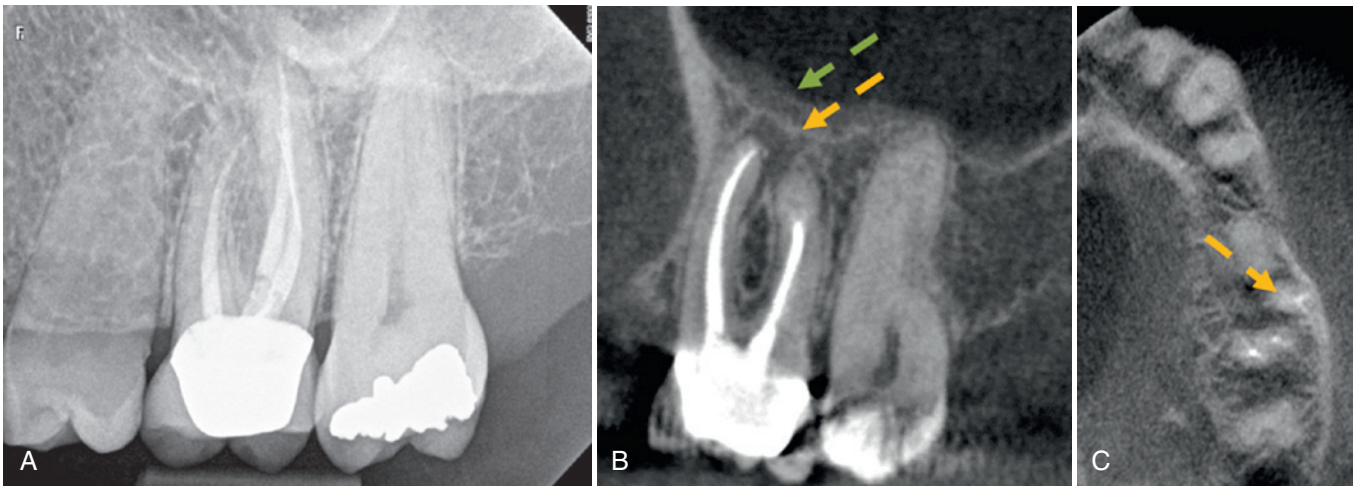

FIG. 2-15 A 62-year-old female was referred for evaluation and possible treatment for chronic continuous dentoalveoar pain (CCDAP) in the maxillary left region. The patient could alleviate this condition by placing a moist cotton roll in the adjacent vestibule to prevent the buccal mucosa from contacting the alveolus supporting the maxillary teeth in this area. This condition began after a history of local anesthetic administration, persisted for 1 year, and led to three successive new crowns and endodontic treatment on the "offending tooth," in an effort to remedy the condition without improvement. The response to endodontic tests, TMJ, and myofascial evaluations were normal. Application of topical xylocaine resulted in cessation of pain for 15 minutes. A PA radiograph **(A)** showed a root-treated, maxillary left first molar with no apparent radiographic lesion. A limited FOV CBCT of the maxillary left posterior was exposed. The corrected sagittal view **(B)** showed an approximately 4 mm, well-defined oval, mildly corticated area of low attenuation (radiolucent) centered over the apex of the mesiobuccal root and extending to the junction of the middle and apical third of the mesiobuccal root (yellow arrow). There was a mild mucositis (green arrow). There was a previously untreated mesioaccessory canal **(C)** and a mild mucositis. A diagnosis of neuropathic pain and a chronic apical periodontitis was made. Daily application of topical ketamine, gabapentin, and clonidine was prescribed. Endodontic revision of the maxillary left first molar was performed 3 months after the patient was stabilized with the topical medications. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D [Carestream Dental, LLC, Atlanta, GA].)

periapical lesions in maxillary molars and premolars compared to PA radiography.[109]

Lesions of Nonendodontic Origin

Differential diagnosis of periapical pathology is crucial to endodontic treatment planning. Substantial evidence in the literature points to a significant chance that lesions of the tooth-supporting structures are nonendodontic in origin, such as periapical cemento-osseous dysplasia; central giant cell granulomas; simple bone cysts; odontogenic cysts, tumors, or malignancies; and neuropathic pain (Fig. 2-15).*

Neuropathic orofacial pain or atypical odontalgia (AO), also known as *chronic continuous dentoalveolar pain* (CCDAP)[131a] and *persistent idiopathic facial pain* (PDAP),[80a] is related to a tooth, teeth, or pain at an extraction site where no clinical or radiographic pathosis is evident. Two systematic reviews of AO showed the incidence of persistent pain of more than 6 months' duration after nonsurgical and surgical endodontic treatment, excluding local inflammatory causes, was 3.4%.[130a] The pathophysiology of this pain is uncertain, but it is hypothesized to involve deafferentation of peripheral sensory neurons in predisposed patients. The diagnosis of AO is challenging and depends on the patient history and clinical examination findings, in addition to the absence of radiographic findings. In some cases the symptoms from AP and AO are closely related. Pigg et al.[146a] conducted a study of 20 patients with AO. All of the patients had at least one tooth in the region of discomfort that had undergone invasive treatment; 21 of 30 teeth had undergone endodontic treatment. These researchers found that 60% had no periapical lesions, and among those who did, CBCT showed 17% more periapical lesions than conventional radiography. This study demonstrated that CBCT may be a useful supplement to 2D radiography (see Fig. 2-15).

The 3D radiographic appearance of a periapical lesion provides additional information about the lesion's relationship to the tooth and other anatomic structures (e.g., the vascular bundle) and about the aggressiveness of the lesion. This information, along with pulp sensitivity testing, is useful for adequate treatment planning and management of these conditions.

Diagnosis of Endodontic Treatment Failures

Failure of previous endodontic therapy can be attributed to various factors, such as procedural errors, missed canals, or persistent periapical pathosis. Knowledge of the cause of failure is pertinent to the treatment of these cases because it allows the cause to be adequately rectified. With the advent of CBCT, in select cases of retreatment in which the cause of failure is otherwise undetectable, adequate information may be collected to apply to the treatment plan (Fig. 2-16). The technology is most useful in detecting uninstrumented and unfilled

*References 28, 54, 56, 81, 130, 143, 151, and 152.

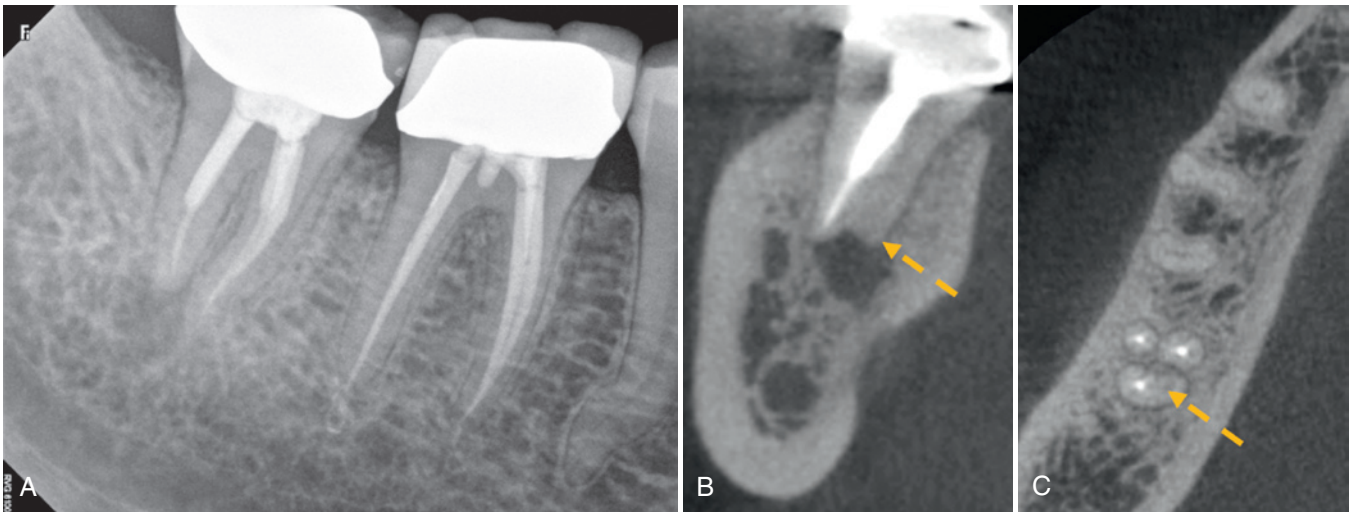

FIG. 2-16 This 38-year-old female patient presented for evaluation and treatment of a symptomatic mandibular right second molar, which had been endodontically treated more than 10 years previously. This tooth was sensitive to percussion and biting forces; periodontal findings were normal. Microscopic examination of the exposed dentin was negative for a vertical fracture. The PA radiograph **(A)** showed the previous endodontic treatment; a post present in the distal canal; and an approximately 5 mm diameter, unilocular, diffuse area of low density centered at the periapex of the distal root. Contemporaneous CBCT sagittal **(B)** and axial **(C)** images revealed a previously untreated distobuccal canal. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

canals, extension of the root canal filling, and the presence and extent of periradicular bone loss. The sensitivity of CBCT and PA radiographs for diagnosing strip perforations in root-filled teeth has been shown to be low.[51] Radiopaque filling materials in the root canals of endodontically treated teeth can produce streak artifacts, which can mimic fracture lines or perforations.[166,198]

Vertical Root Fractures

Vertical root fractures (VRFs) that run along the long axis of a tooth are often difficult to diagnose clinically. The prevalence of VRF in endodontically treated teeth has been reported to range from 8.8% to 13.4%.[61,174,196] These fractures typically run in the buccolingual direction and are confined to the roots, making it difficult to visualize the fracture. Visualizing the fracture on a conventional radiograph is possible when the x-ray beam is parallel to the plane of the fracture.[153] Challenges in diagnosis with regard to the extent and exact location of the fracture often lead to unwarranted extraction of teeth. Since the introduction of CBCT to dentistry, various reports of the application of the technology to detect vertical root fractures have been published. The reported sensitivity for detection of VRFs has ranged from 18.8% to 100%[115]; by comparison, conventional radiographs have a reported sensitivity of approximately 37%[48,75] (Fig. 2-17). CBCT has been used to visualize VRFs in controlled clinical studies in which clinical diagnosis was difficult.[48] Vertical root fractures were successfully detected at a spatial resolution ranging from 76 to 140 microns. However, only a limited number of units provide such high resolution. A comparison of various CBCT units for the detection of VRFs demonstrated that the units with flat panel detectors (FPDs) were superior to the image intensifier tube/charge-coupled device (IIT/CCD)–based detectors; the smaller FOV and the ability to view axial slices also improved detection of VRFs.[76] Continued improvement of sensor technology, including the use of FPDs, has resulted in enhanced resolution. Voxel dimensions are smaller in these units. Detection of vertical root fractures with thickness ranging from 0.2 to 0.4 mm was found to be more accurate with CBCT than with digital radiography.[133,138] The presence of root canal filling in the teeth lowers the specificity of CBCT in detecting vertical root fractures[75,76,95]; this has been attributed to the radiopaque material causing streak artifacts that mimic fracture lines.[198]

Evaluation of Anatomy and Complex Morphology

The precise location and visualization of dental anomalies, root morphology, and canal anatomy are vastly improved with CBCT data. Root curvature, additional roots, and anomalies within the canals themselves (e.g., obstructions, narrowing, bifurcation) are made more apparent when all three anatomic planes of section are available for review, especially with the capability of narrowing the slice thickness to as little as 0.076 mm. Visual obstruction from anatomic features such as buccal bone and the malar process over the apices of maxillary roots simply will "disappear" when you can scroll through the slices of the bone from facial to palatal in 0.076 mm sections while also changing axial orientations (Fig. 2-18).

Dental Anomalies

The use of CBCT technology has been reported in the diagnosis and treatment planning of various dental anomalies (e.g., dens invaginatus) that often have complex morphologic presentations.[131] The prevalence of dens invaginatus was as high as 6.8% in the adolescent Swedish population studied.[14] The complex nature of the anomaly presents a diagnostic challenge

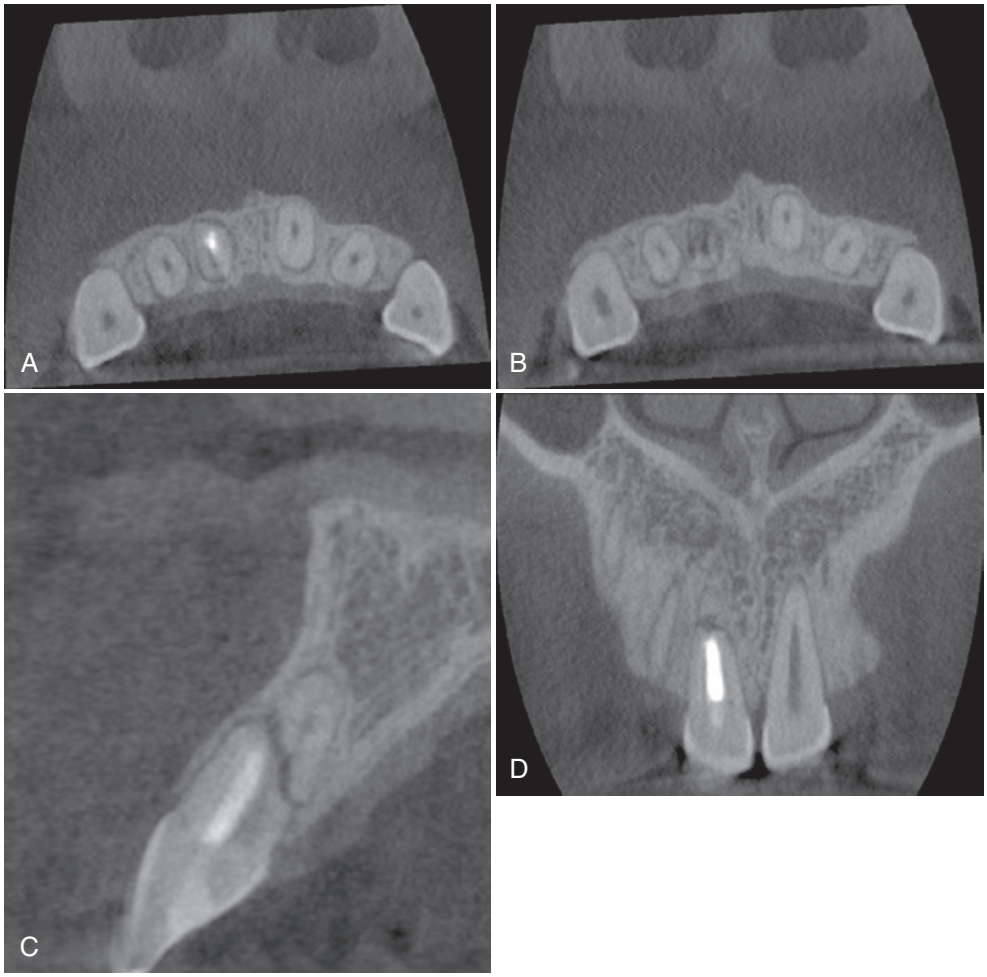

FIG. 2-17 Root fracture in an endodontically obturated maxillary right central incisor. **A**, Axial view with artifacts from a highly attenuating (opaque) obturant. **B**, View without artifacts from obturant. **C**, Oblique parasagittal view. **D**, Paracoronal view. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

when conventional radiographs are used.[137] In case reports in which CBCT was used for diagnosis and treatment planning, treatment options included conservative endodontic treatment of the invagination, surgical treatment of the periapical pathology, and complete revascularization of the dens after removal of the invagination (Fig. 2-19).[124,185]

Root Canal System Morphology

As the adage goes, nature seldom makes a straight line and never makes two of the same. This statement is dramatically illustrated in the evaluation of root canal system morphology. With ever-present unusual and atypical root shapes and numbers, there is sometimes a need to look further than what a clinician can see or imagine with 2D radiography (Fig. 2-20). Variations in root canal morphology have been studied using various in vitro techniques.[57,147,182,183] The results of these studies point to the fact that there is significant variation in the root canal morphology among various ethnic population groups.[3,72,73,128,187] CBCT has been reported to be comparable to canal staining and clearing techniques for identification of the root canal morphology,[125] and CBCT studies report variation in the root canal morphology among various ethnic groups.[126,173,197,199]

INTRAOPERATIVE OR POSTOPERATIVE ASSESSMENT OF ENDODONTIC TREATMENT COMPLICATIONS

Materials Extending Beyond the Root Canal

CBCT scans provide the opportunity to map endodontic treatment complications through the examination of 3D representations of the teeth and supporting structures in different planes. Few high-level studies related to the effects of endodontic treatment complications have been published in the endodontic literature.[176] However, it is generally recognized that overfilling of the root canal, causing damage to vital structures such as the inferior alveolar neurovascular bundle (IAN) (Fig. 2-21) or the maxillary sinus, can cause significant morbidity.[24,25,60,64]

Endodontic therapy undertaken in close proximity to the IAN should receive special attention because direct trauma, mechanical compression, chemical neurotoxicity, and an increase in temperature greater than 10° C may cause irreversible damage.[51,68,71,179] Scolozzi et al.[162] reported that sensory disturbances can include pain, anesthesia, paresthesia,

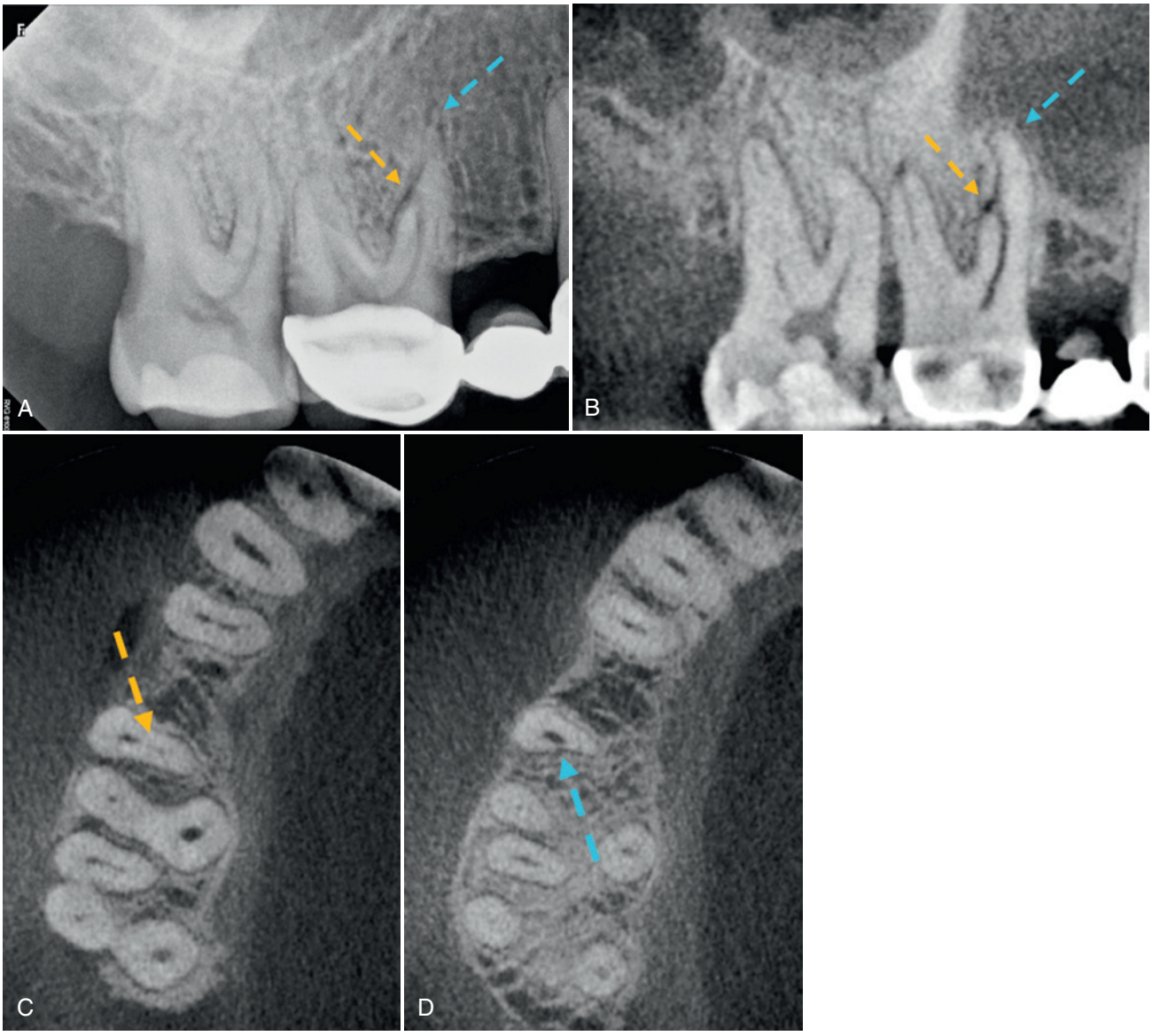

FIG. 2-18 This patient was referred for evaluation and possible treatment after emergency pulp extirpation performed by others. The location of the physiologic terminus and radiographic apex did not appear to coincide even remotely in the PA radiograph **(A)**, so a CBCT-aided assessment was required. A CBCT scan was able to clearly show the exact location of the canal terminus and radiographic apex **(B)**, the presence of a mesioaccessory canal with an isthmus **(C)**, and the oval canal **(D)**, facilitating treatment of this case. (Courtesy Dr. Anastasia Mischenko, Chevy Chase, Md. Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

hypoesthesia, and dysesthesia.[162] The IAN is located in the cribriform bone-lined mandibular canal and courses obliquely through the ramus of the mandible and horizontally through the mandible body to the mental foramen and the incisive foramen.[8] There are many anatomic variations of the IAN, including the anterior loop and bifid mandibular canals.[35] Kovisto et al.[97] used CBCT measurements from 139 patients to show that the apices of the mandibular second molar were closest to the IAN. In females, the mesial root of the second molar was closer than in males, and the distances in all roots measured increased with the age of the patient. There was a high correlation between the measurements from left to right side in the same patient; an average distance of 1.51 to 3.43 mm in adults.[97] Procedures involving the mandibular second molar were most likely to cause nerve damage.[106] Further research is required to clarify the risks and benefits of CBCT when endodontic treatment is contemplated on teeth with a proximal relationship between the IAN and root apices. Porgrel[148] treated 61 patients with involvement of the IAN after root canal therapy during a 7-year period. Eight patients were asymptomatic; 42 patients were seen for mild symptoms or were examined more than 3 months postoperatively, with only 10% experiencing improvement. Five patients underwent surgical treatment before 48 hours elapsed and recovered completely.

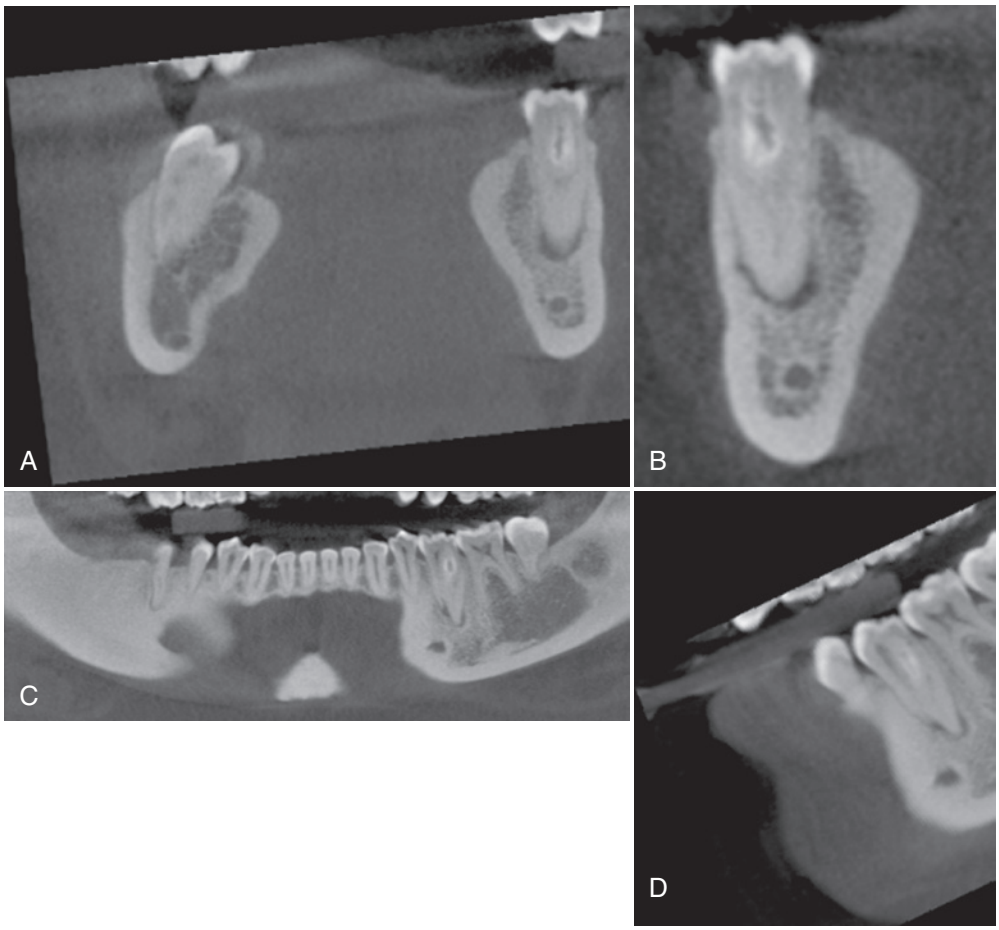

FIG. 2-19 **A,** Dens in dente of the mandibular left second bicuspid; coronal slice. **B,** Coronal view. **C,** Panoramic reconstruction from CBCT. **D,** Sagittal view. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

Six patients underwent surgery between 10 days and 3 months, with four experiencing partial recovery; the remaining two had no improvement.[148]

New imaging technologies, such as high-resolution magnetic resonance imaging (MRI-HR) and magnetic resonance neurography (MRN), promise to improve isolation of the IAN from the neighboring artery and vein contained within the inferior alveolar bony canal. MRN studies have documented the ability to demonstrate nerve continuity and localize extraneural nerve compression before surgical nerve exploration. Postoperative periapical radiographs should be exposed on the day of endodontic treatment completion or a suspected iatrogenic event, and any suspected compromise of the IAN or other vital structures should be evaluated immediately. In all cases in which trauma to the IAN is suspected from periapical or panoramic radiography or by the report of symptoms consistent with nerve injury, exposure of a CBCT image volume should be considered. It is generally accepted that immediate surgical debridement should be attempted to maximize recovery.[51,149] With the introduction of MRI algorithms for dental diagnostic purposes, it is expected that this imaging modality will be increasingly used in diagnostic and treatment planning. MRI has the capability to demonstrate vascularity to the tooth of interest, in addition to the presence of inflammatory exudates in the apical regions, without exposing the patient to ionizing radiation. Receiver coils are being developed to enhance the image quality of maxillofacial and dental magnetic resonance studies.

The accidental introduction of root canal instruments, irrigating solutions, obturation material, and root tips into the maxillary sinus has been reported. Serious consequences associated with the intrusion of foreign bodies into the maxillary sinus include pain, paresthesia, and aspergillosis, a rare but well-documented complication of endodontic treatment.[16a] Guivarc'h et al.[71a] reported that the overextension of heavy metal–containing root canal sealers, such as zinc oxide eugenol cement, may promote fungal infection in immunocompromised patients, leading to bone destruction and damage to adjacent structures. This case report described the use of computed tomography to assess the patient before surgery and at 6 months.[71a] The use of CBCT as an aid in the localization and retrieval of an extreme overextension of thermoplasticized injectable gutta-percha into the sinus and contiguous soft tissues has been described by Brooks and Kleinman.[26]

Fractured Instruments

Instrument fracture can occur at any stage of endodontic treatment, and in any canal location. The incidence of this complication, reported in clinical studies on a per canal or per tooth basis, ranges from 0.39% to 5.0%.[44,135] Molars are

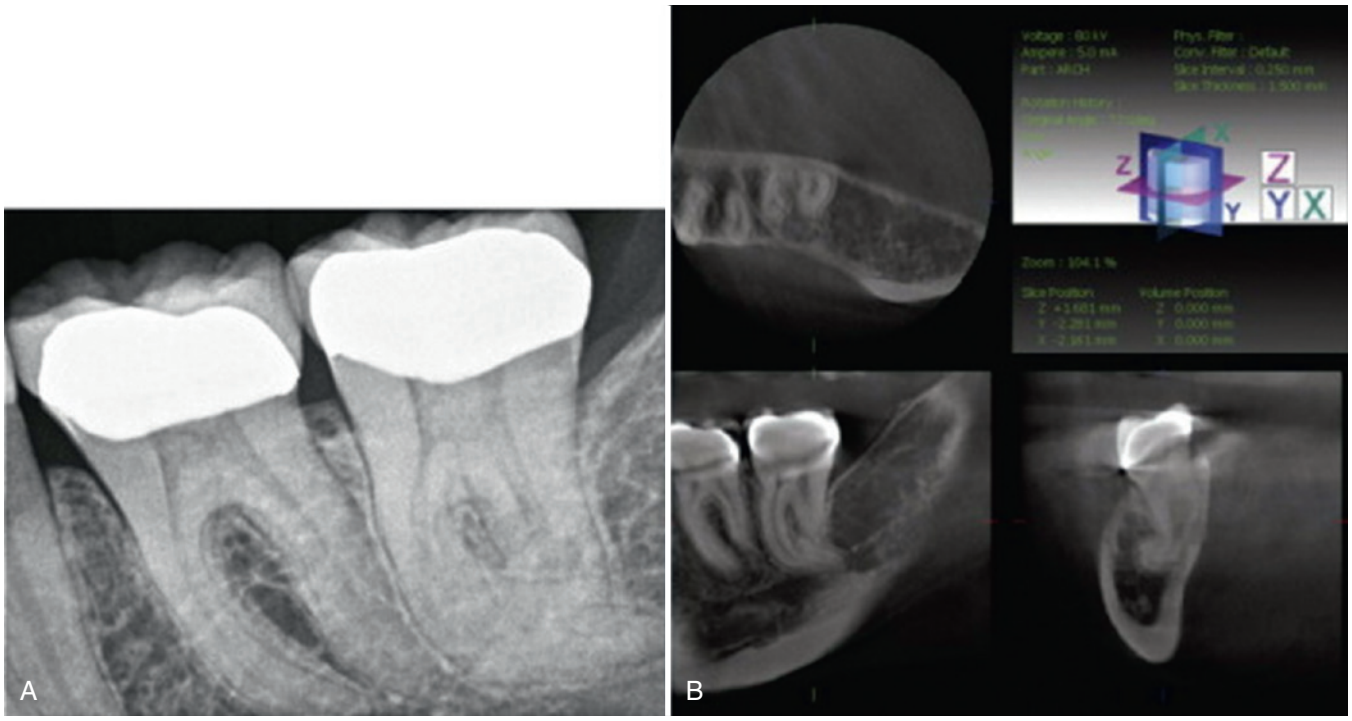

FIG. 2-20 A, Mandibular left second molar referred for endodontic evaluation and possible treatment. This 2D radiograph reveals significant pulp stones and canal calcification developing not only in the coronal aspect of the root canal system, but also extending down the visible distal canal. The apical third of the canal system appears unusual and dilacerated. Cone beam volumetric tomography (CBVT) would be beneficial in visualizing the root canal anatomy, so as to create the ideal endodontic access. B, Single slice of the CBVT image for tooth #18. Information about the direction of the root canal anatomy is provided in all three planes of the section: axial, coronal, and sagittal. Interestingly, the axial slice shows that the mesial lingual root actually traverses buccally as it approaches its terminal extent. This is valuable information for the clinician before the entire root canal system is cleaned and shaped; it also establishes a higher degree of treatment predictability.

predominantly affected by instrument fracture, with the highest incidence found in the apical third of mandibular molars.[4,40,103,127]

A systematic review and meta-analysis showed that when endodontic treatment was performed at a high technical standard, instrument fracture did not significantly reduce the prognosis. More specifically, when no initial radiographic periapical lesion was present initially, 92.4% of cases remained healthy; when a periapical lesion was present initially, 80.7% of periapical lesions showed radiographic healing. However, the presence or absence of periradicular lesions on preoperative and postoperative examinations was based on planar radiographic assessments, which calls these findings into question.[134] Other studies showed that the chances of endodontic failure increased if the canal system could not be thoroughly disinfected, if a periradicular periodontitis was present, or if technical standards were compromised.[39,91,168,170] Use of CBCT to triangulate the retained instrument and assess the canal shape, especially in cases in which the operating microscope does not allow direct visualization, can be helpful in formulating a removal strategy. If the fractured instrument is lodged in the lingual aspect of a ribbon-shaped canal, for example, an instrument may be inserted toward the buccal to bypass and remove the imbedded instrument without forcing the fragment further apically. Without the use of CBCT, intracanal instruments can be reliably removed or bypassed in 85.3% of cases if straight-line access is possible; however, reliable removal or bypass is possible in only 47.7% of cases if the instrument is not visible (Fig. 2-22).[127] When a separated instrument is lodged in the apical third of a root canal, the chances of retrieval are the lowest, but the apical terminus may be adequately sealed by treatment of an anastomosing canal, if present.[63] The possibility of instrument removal based on CBCT triangulation has not been published to date.

Calcified Canals

According to the Pew Research Center, 10,000 U.S. individuals will reach the age of 65 every day until 2030, and the nation's 65-year-old and over cohort will grow to 81 million in 2050, up from 37 million in 2005 (also see Chapter 26).[82] This aging population will present increasing challenges for dental clinicians because calcification of the root canal system increases as part of the natural aging process,[69] possibly leading to more untreated canals that may serve as a niche for microorganisms.[19,88] Pulp chambers in the crown of the tooth decrease in size, forming more rapidly on the roof and floor of posterior teeth.[184] Typically, root canals calcify at the coronal aspect first, with decreasing calcification as the canal travels apically. Magnification and illumination are essential tools for the identification and treatment of calcified canals, but CBCT can assist in the perioperative treatment of such conditions.[16] Preoperative assessment of calcified teeth using CBCT can suggest the best tactic for locating calcified canals in the chamber floor and

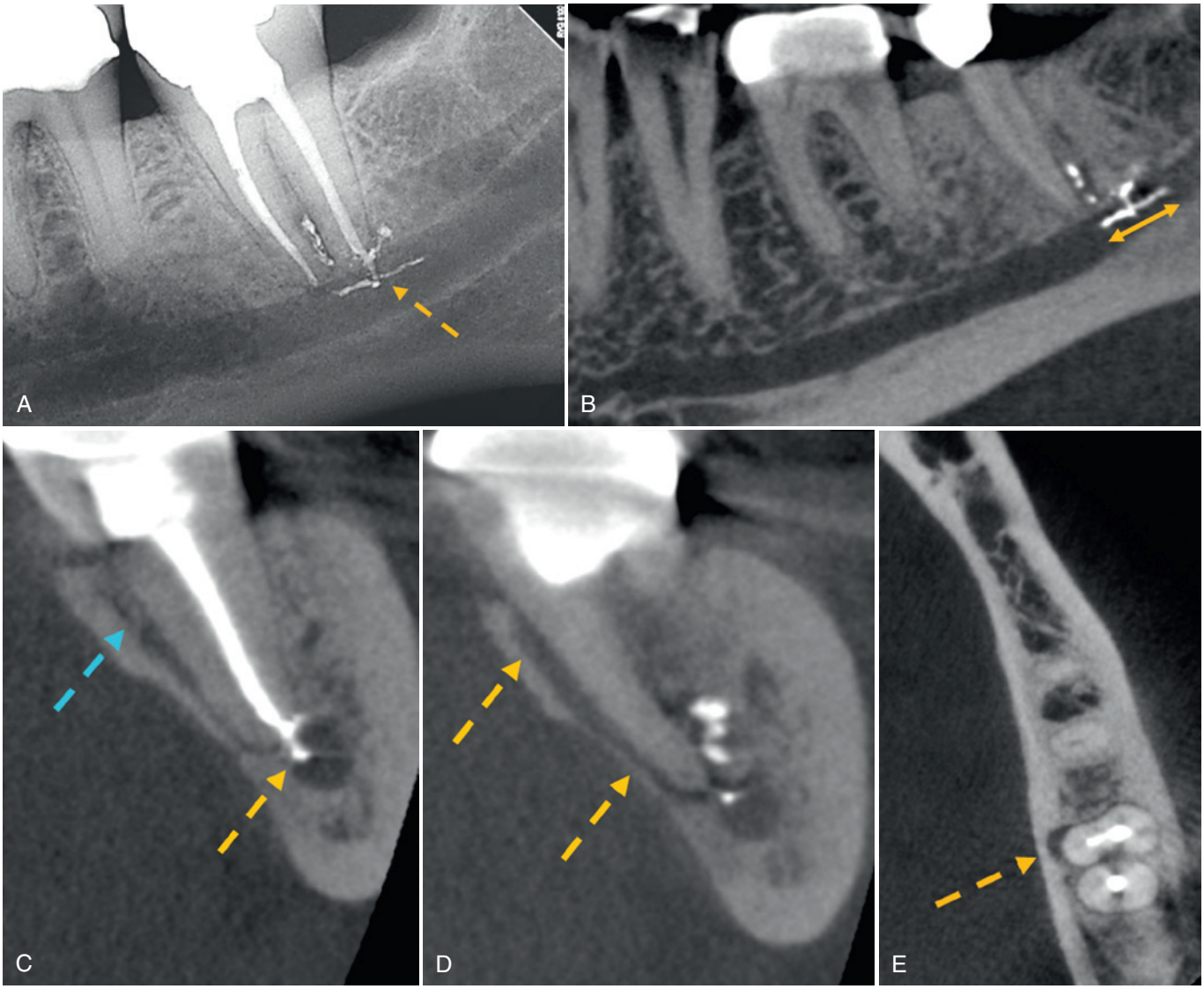

FIG. 2-21 This 64-year-old male patient presented with sensitivity when biting on the mandibular left second molar. The history included endodontic revision more than 6 months earlier and subsequent transient paresthesia and dysesthesia along the distribution of the inferior alveolar nerve, which persisted for 1 week after the retreatment. The results of periodontal probing and microscopic examination of the exposed root surface at the sulcus were normal. The PA radiograph showed the approximate location of the excess radiopaque material, a feature consistent with extruded sealer (**A**, *yellow arrow*). The length of approximately 3.4 mm and the true location of the offending material in the mandibular canal were assessed in the corrected sagittal view (**B**, *yellow arrow*) and corrected cross-sectional view (**C**, *yellow arrow*). The corrected cross-sectional view also showed a lesion of low attenuation extending from the apex to an area near the crest of the alveolus with erosion of the lingual cortical plate (**C**, *blue arrow;* **D**, *yellow arrows*). The same lesion was shown in the corrected axial view (**E**, *yellow arrow*). Examination of the extracted tooth revealed a vertical fracture at the lingual aspect of the distal root. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

roots using software-based measurement tools. The insertion of radiopaque markers, such as instruments or obturation material, can facilitate reliable canal localization using available multiplanar reformations. The increased sensitivity and specificity provided by CBCT can also assist in the determination of the periapical status of calcified root canals that may not require measures that can lead to procedural errors, such as off-course access, instrument fracture, or root perforation.[83] The difficulty in locating calcified canals can be further compounded by morphologic anomalies associated with gender and ethnic origin.[163] CBCT can be an important adjunct to magnification and illumination in these cases.

Perforations

A *perforation* is defined as a "mechanical or pathologic communication between the root canal system and external tooth surface"[6]; it is usually associated with an iatrogenic event,[159] accounting for about 10% of all nonhealed cases.[85] Root perforations can be caused by a post preparation, the search for a calcified canal, a strip perforation, or an attempt to retrieve a

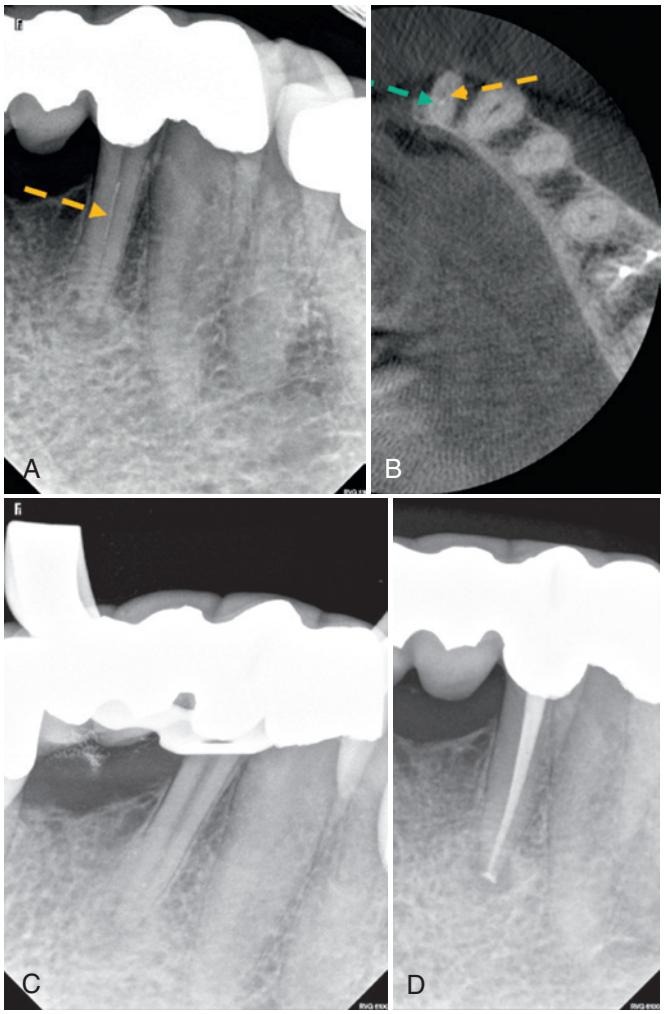

FIG. 2-22 Unexpected torsional and flexural failure of endodontic instruments can occur during instrumentation. This PA radiograph **(A)** shows a separated stainless steel hand file located at the midroot of the mandibular left lateral incisor in a patient referred for revision therapy. To aid development of a retreatment strategy, CBCT was used to localize the instrument (**B**, *yellow arrow*) in the lingual canal. The patient's buccal canal was identified (**B**, *green arrow*), and subsequent instrumentation allowed removal of the offending instrument segment **(C)**, leading to successful completion of treatment (**D**). (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

fractured instrument. They are often difficult to localize with conventional imaging because no information about the buccolingual dimension can be obtained.[195] Shemesh et al.[166] compared the sensitivity and specificity of CBCT scans with two-angulation periapical imaging using phosphor plates to assess the likelihood of detecting root or strip perforations after root canal treatment with laterally compacted gutta-percha and sealer. They found that the two methods showed similar specificity, but the CBCT image volumes showed higher sensitivity. Single-angulated periapical radiographs (PAs) showed 40% of the perforations, and two-angled PAs showed 63%, suggesting that if PAs alone are used, two-angled images were superior. There was no significant difference in the detection of root perforations between PA and CBCT radiography. The researchers noted that the results may have been affected by the small size of the perforations and that the method of obturation did not favor extravasation of obturation material.[166] CBCT images suffer from beam hardening artifact resulting from root canal obturation and restorative materials (e.g., gutta-percha, posts, and perforation repair materials), which creates challenges to the interpretation of root integrity. An approach advocated by Bueno et al.[29] suggested that a map-reading strategy of viewing sequential axial slices reduces the beam hardening effect. Newer root canal obturation materials with lower radiopacity profiles and improved CBCT software algorithms are expected to reduce artifact formation in the future.

Dentoalveolar Trauma

Systematic epidemiologic data suggest that facial trauma is a common occurrence, resulting in injuries to the dentition in 57.8% of household and play accidents, 50.5% of sports accidents, 38.6% of work-related accidents, 35.8% of acts of violence, and 34.2% of traffic accidents, with 31% unspecified.[62] The prevalence of traumatic dental injuries varies according to the population studied, but these injuries occur most commonly in children 7 to 10 years of age (also see Chapter 20).[13] Dental traumatic injuries affect one fourth of all schoolchildren and almost one third of adults, and most injuries occur before the age of 19.[65] Maxillary central incisors sustain approximately 80% of all dental traumatic injuries, followed by maxillary lateral incisors and mandibular incisors.[9] The most common type of traumatic dental injuries in the primary dentition are luxation injuries, whereas crown fractures are the predominant dental injury to the permanent dentition.[98] Determination of the extent of injury to the dentinopulpal complex requires a methodical approach that evaluates the teeth, periodontium, and associated structures (Fig. 2-23) and may result in significant long-term complications.[5,42]

Injuries to the orofacial complex can cause dental trauma that results in the following injuries to the primary and permanent dentition: (1) infraction; (2) crown fracture, uncomplicated and complicated; (3) crown/root fracture; (4) root fracture; (5) concussion; (6) subluxation; (7) lateral luxation; (8) intrusion; (9) extrusion; and (10) avulsion.[12] The International Association of Dental Traumatology's guidelines for the management of traumatic dental injures suggest that CBCT may be beneficial when used to assess and monitor healing in patients after dental traumatic injuries, especially in cases of lateral luxation and root fracture.[45] (These guidelines can be reviewed at *www.dentaltraumaguide.org*.) Intra-alveolar root fractures generally affect the permanent dentition of males and are relatively uncommon, accounting for 0.5% to 7% of dental impact injuries.[12,41,67,132] Root-fractured teeth are a challenging condition to diagnose, and the limitations of planar radiography have been well documented in the dental literature.[38,45,96,136] A systematic retrospective study showed that maxillary central (68%) and lateral (27%) incisors were primarily affected, with only limited occurrence in mandibular incisors (5%). This retrospective study concluded that CBCT allowed improved treatment planning compared with PA imaging alone.[191] At least seven systematic laboratory studies and one systematic in vivo animal study reported significantly improved accuracy for the detection of root fractures when CBCT was compared with periapical radiography.* In a systematic clinical study,

*References 75, 76, 84, 114, 133, and 189.

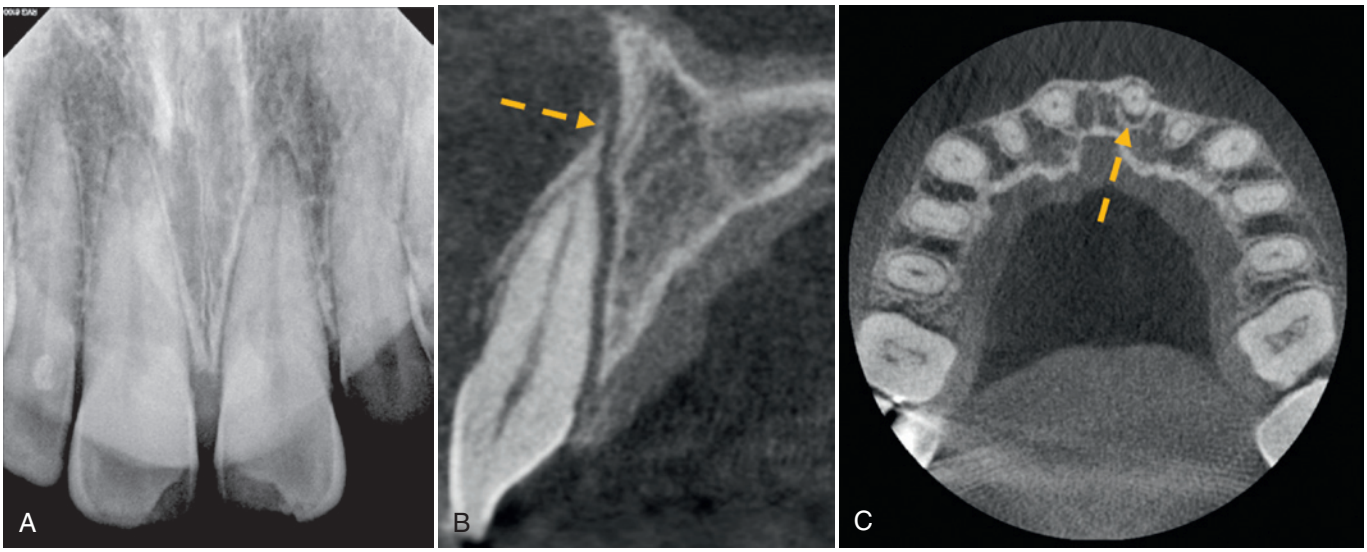

FIG. 2-23 After a traumatic injury to the maxillary right and left central incisors, crown fractures were noted. The PA radiograph showed an extrusive luxation injury in the maxillary left central incisor (**A**). The alveolar fracture (**B**, *yellow arrow*) and true extent of the displacement (**C**, *yellow arrow*) became evident with CBCT. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

Bornstein et al.[23] examined 38 patients with 44 permanent teeth that sustained intra-alveolar fractures. In the study sample reported, 68.2% of teeth had oblique fractures that extended to the cervical third of the root, contradicting the findings of previous studies conducted with periapical imaging alone. CBCT imaging offered improved visualization of the location and angulation of root fractures compared to periapical and occlusal intraoral radiographs.[23]

Several of these studies suggest that lower resolution scans using voxel sizes in excess of 0.3 mm may not improve radiographic assessments.[76,189] A study by Wang et al.[186] showed that the sensitivity and specificity of PA radiography for root fractures were 26.3% and 100%, respectively; the findings for CBCT were 89.5% and 97.5%, respectively. CBCT images of root-filled teeth showed lower sensitivity and unchanged specificity, whereas 2D images showed the same sensitivity and specificity.[186] CBCT allows for the management of traumatic injuries in which a root fracture or alveolar fracture is suspected by providing undistorted multiplanar views of the dentition and supporting bone without the superimposition of anatomic structures.[37,107,157] CBCT image volumes provide superior sensitivity in detecting intra-alveolar root fractures compared with multiple PA radiographs; this allows for the detection of dental and alveolar displacements, including damage to other perioral structures, such as the maxillary sinus and nasal floor.[89] The presence of root canal fillings and posts affected the specificity of the findings as a result of artifact generation.[76,114] Outcome measurements of a region of interest can be compared over time with greater geometric accuracy using CBCT.[74]

The healing of root fractures is influenced by many factors, most prominently the stage of root development, with immature roots showing better healing than mature roots.[59] Other factors that influence healing are the extent of dislocation and repositioning, type of splinting, use of antibiotics, and location of the fracture on the root. The long-term survival of teeth with intra-alveolar root fractures was evaluated in a systematic study by Andreasen et al.[11] This study showed that the type of healing (e.g., hard tissue fusion, PDL interposition with and without bone) and the location of the fracture on the root had the most influence on tooth loss. CBCT should be considered when placement of individual PA radiographs will adversely affect patient management; PA radiographs will produce a higher radiation dose for assessment of the region of interest; or intra-alveolar fracture of the root or supporting structures is suspected and sufficient information cannot be obtained with conventional radiography.[89]

The decision to use CBCT imaging for assessment of traumatic injuries should be based on the diagnostic yield expected and in accordance with the "as low as reasonably achievable" (ALARA) principle. CBCT scan volumes that use the most appropriate detector size and shape, beam projection geometry, and beam collimation should be selected to produce high-resolution images and reduce x-radiation exposure whenever possible.[158] In all cases, it should be recognized that children and young adults are more susceptible to the effects of radiation than adults, and CBCT studies should answer specific clinical questions that cannot be answered by lower dose PA and panoramic imaging technologies.[79] New technologies that allow for comparison of matched CBCT images in a serial fashion at a reduced dose have been tested. This technology promises a dose reduction of 10 to 40 times by using the initial scan as prior knowledge and adaptive prior image constrained compressed sensing (APICCS) algorithms to greatly reduce the number of projections and x-ray tube current levels required (Fig. 2-24).[100]

Internal and External Root Resorption

Most clinicians are aware, and ideally communicate to their patients, that the long-term prognosis for teeth with extensive root resorption may be unpredictable. Endodontic treatment can often resolve these defects, and early diagnosis and treatment typically mean an improved prognosis. CBCT imaging of these resorptive lesions provides the clinician with enhanced

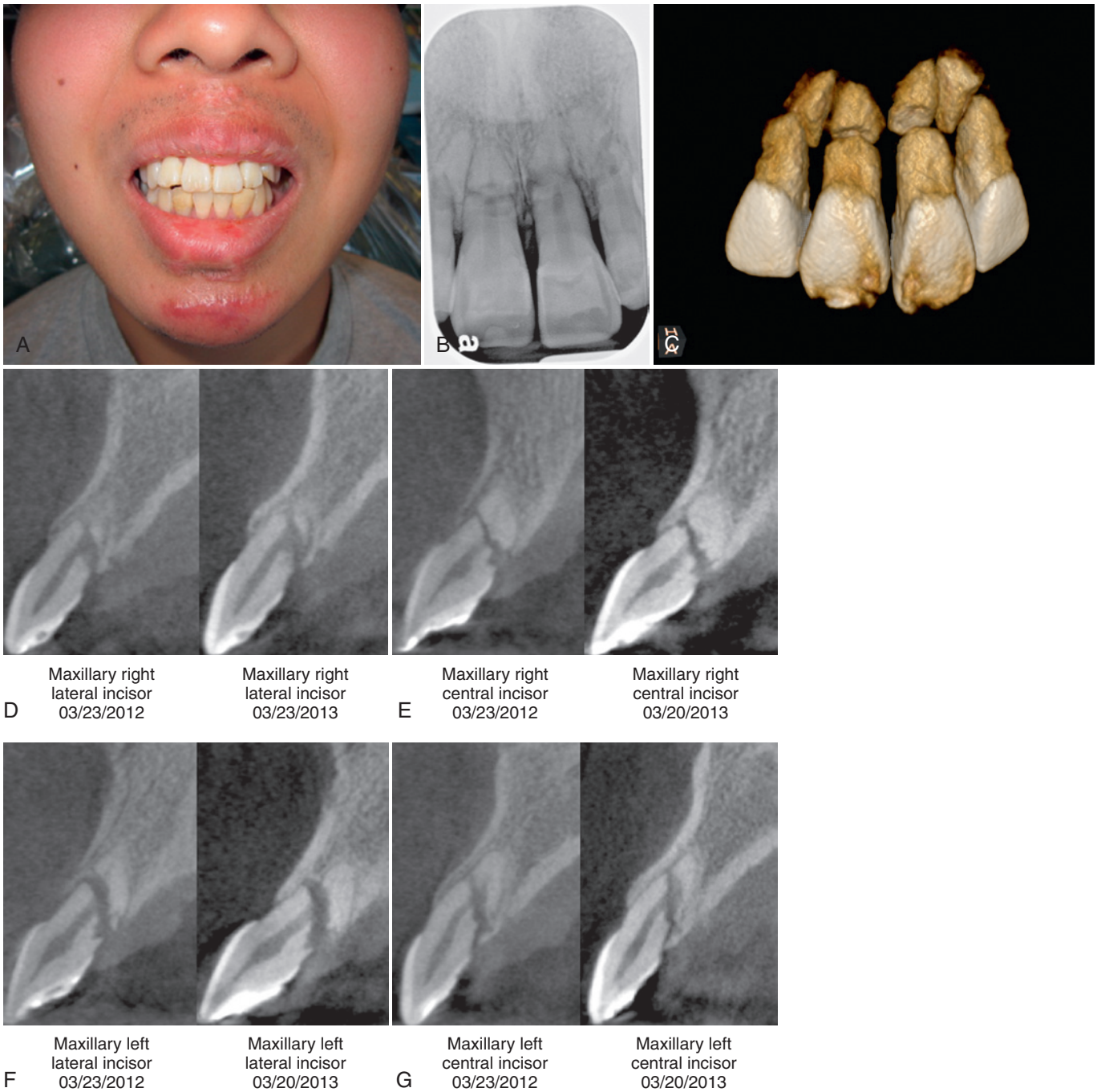

FIG. 2-24 Horizontal root fractures resulting from trauma (A) were evident in this 22-year-old male patient, who was referred with a contemporaneous film–based PA radiograph (B) for evaluation and possible treatment 9 months after trauma to his maxillary lateral and central incisors. Since the trauma, the teeth had been stabilized with a ribbon-type splint on the palatal surface; they were of normal color and responded to all pulp tests within normal limits. There was slight mobility in all of the traumatized teeth. The true nature of the root fractures (C, 3D reconstructed view) was evident in the corrected sagittal views of the maxillary right lateral (D) and central (E) incisors, and in the maxillary left central (F) and lateral (G) incisors. Temporal examination revealed no changes at the initial presentation, 12-month, and 30-month reevaluations (left to right in each group). Task-specific exposure parameters allowed each successive CBCT image volume to be exposed with lower kVp and mA, resulting in a 20% radiation dose reduction. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

information so that more appropriate treatment planning options are available.

As described in Chapter 16, root resorption (RR) results in the loss of dentin, cementum, or bone by the action of clastic cells.[15] In the primary dentition, RR is caused by normal physiologic processes, except when resorption is premature; in the permanent dentition, it is caused by inflammatory processes.[36,52,141] The successful management of RR in the adult dentition depends on clinical and radiographic examination leading to early detection and accurate diagnosis.[142] Unfortunately, teeth affected by RR have a poor prognosis if the causative lesion is not treated.[140] Although parallax intraoral imaging techniques can be helpful in localizing RR,[86] only CBCT assessments can provide the true size and position of all resorptive defects in the region of interest.[36,140] Intraoral imaging produced false-negative results in 51.9% of cases studied and false-positive results in 15.3%.[122] The interpretation of RR should rely on the least invasive test that can reliably detect the occurrence of the condition.[140] The use of CBCT in the evaluation of RR eliminates structure superimposition and compression of 3D features. Patel et al.[140] compared the sensitivity and specificity of PA imaging with CBCT scans using the receiver operating characteristic (ROC) curve, a standard measure of diagnostic performance. PA imaging showed satisfactory (Az 0.78) accuracy, whereas CBCT showed perfect results (Az 1.00).[140]

Although the literature describes many classification systems, this section divides inflammatory root resorption into two groups, according to location: internal root resorption (IRR) and external root resorption (ERR).[177] IRR is a relatively rare occurrence that is usually detected on routine diagnostic PA or panoramic radiographs.[102,139] It is characterized by structural changes in the tooth that appear as oval or round radiolucent enlargements of the pulp canal, usually with smooth, well-defined margins.[32] IRR is usually asymptomatic, associated with pulpal necrosis coronal to the resorptive lesion and vital or partially vital pulps where active.[140] These lesions can easily be confused with extracanal invasive cervical resorption because the radiographic appearance of the two lesions can be identical. CBCT is helpful for diagnosing the location and exact size of IRR. In a study by Estrela et al.,[52] 48 PA radiographs and CBCT scans were exposed on 40 individuals.[52] IRR was detected in 68.8% of PA radiographs, whereas CBCT scans showed 100% of the lesions. Conventional radiographs were able to detect only lesions between 1 and 4 mm in 52.1% of the images, whereas CBCT was able to show 95.8% of the lesions. This finding was in agreement with other studies that demonstrated the value of tomographic analysis.[36,105] In a study by Kim et al.,[92] the extent and location of the IRR was accurately reproduced with the fabrication of a rapid prototyping tooth model. Although relatively few systematic studies on artificially induced IRR have been reported because of the difficulty in creating such defects, Kamburoglu and Kursun[90] concluded that high-resolution CBCT imaging performed better than low-resolution CBCT imaging in detecting simulated small internal resorptive lesions.

ERR is typically idiopathic, but severe luxation and avulsion injuries can result in lesions that may progress rapidly, and early treatment is recommended.[47] The recommended timing of radiographic examinations using CBCT has not been established with a strong evidence grade. Many ERR cases involve young patients, in whom the radiation dose is critical, and multiple scans would be difficult to justify. ERR can be classified as surface resorption, external inflammatory resorption, external replacement resorption, external cervical resorption, and transient apical breakdown (Fig. 2-25).[141] These lesions are always associated with bony resorption, making comparability of laboratory studies problematic because the lesions lack the changes in the periodontal membrane and associated bony changes that would improve visualization. Differentiation between IRR and ERR is challenging, even with multiple changes in x-ray angulation. The early stages of ERR were difficult to view with conventional radiography, and lesions less than 0.6 mm in diameter and 0.3 mm in depth could not be detected. Medium-sized lesions were visible in 6 of 13 cases, with improved visualization for proximal lesions without regard to the root third being examined.[10]

ERR is difficult to detect if the lesion is confined to the buccal, palatal, or lingual surfaces of the root.[20,66] Liedke et al.[105] conducted systematic diagnostic performance tests and showed similar sensitivity and specificity among the different

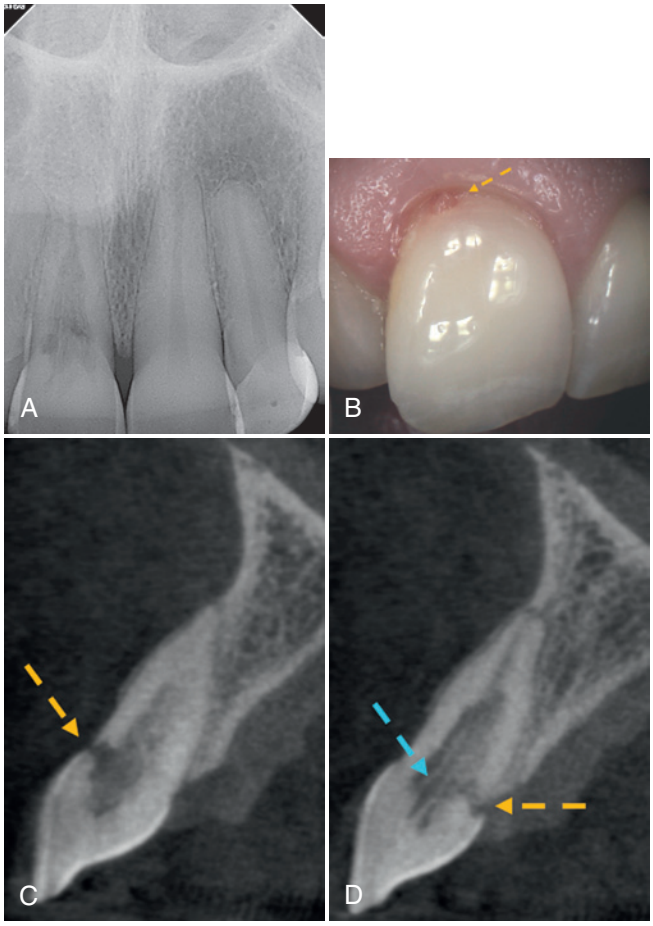

FIG. 2-25 Invasive cervical resorption, a form of external resorption, was evaluated in this patient after a PA radiographic image **(A)** and visual examination **(B)** showed pathognomonic signs of this lesion. A CBCT image was exposed to determine the true extent of resorption and also restorability. The lesion showed a perforative defect at the CEJ on the facial (**C**, *yellow arrow*) and palatal (**D**, *yellow arrow*) aspects. An intact layer of predentin (**D**, *blue arrow*) is a hallmark of this condition. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

voxel sizes studied (0.4, 0.3, and 0.2 mm); however, the likelihood ratio showed better probability of correct identification of ERR with either 0.3 or 0.2 mm scans. These researchers suggested the use of a 0.3 mm voxel size protocol, rather than a 0.2 mm one, to reduce scanning time and the resulting dose.[105] Although voxel size is an important consideration, the SNR of different detectors, the radiation dose, viewing conditions, and the processing algorithms also affect detection probability.

Even though many in vitro studies have been performed on the ability of CBCT to detect RR, additional evaluations that use in vivo methodology will add to the cumulative knowledge.

PRESURGICAL VISUALIZATION

Surgical endodontic treatment is often performed in cases of endodontic nonhealing when nonsurgical retreatment is not possible. In the past, conventional and digital 2D PA radiographs were the only means of assessing the apical region. Unfortunately, the information available from these images may not adequately prepare the clinician to resolve the pathosis surgically. For example, the clinician may be unable to observe whether the lesion has perforated the buccal or palatal cortical plates, as in the example that follows, or even observe which root or roots are involved. Presurgical confusion is resolved with cone beam imaging. Multiplanar views allow the clinician to see the defect and suspected causes from the axial, sagittal, and coronal aspects; 3D grayscale or color imaging helps the clinician visualize the entire defect before the incision is made. This is an immense improvement over conventional imaging (Fig. 2-26).

The relationship of the teeth and the associated pathology to important anatomic landmarks must be taken into consideration in the treatment planning for endodontic surgical procedures. These anatomic landmarks include, but are not limited to, the maxillary sinus, the mandibular canal, the mental foramen, the incisive canal, and the buccal and lingual/palatine cortical plate. The close proximity of the maxillary posterior teeth to the sinus has been linked to maxillary sinusitis of odontogenic origin; changes in the maxillary sinus have ranged from thickening of the schneiderian membrane to actual accumulation of fluid in the sinuses.[110,111,113] The relationship of the roots of the posterior teeth to the sinus during presurgical treatment planning and the changes within the sinus can be best appreciated with the use of CBCT images.[22,111,165]

The relationship of the roots of the mandibular posterior teeth and associated periapical pathology to the mandibular canal, the presence of an anterior loop, and the distance of the mandibular canal from the buccal and lingual cortical plates are pertinent pieces of information when surgical procedures in mandibular posterior teeth are planned.[21] The 3D nature of

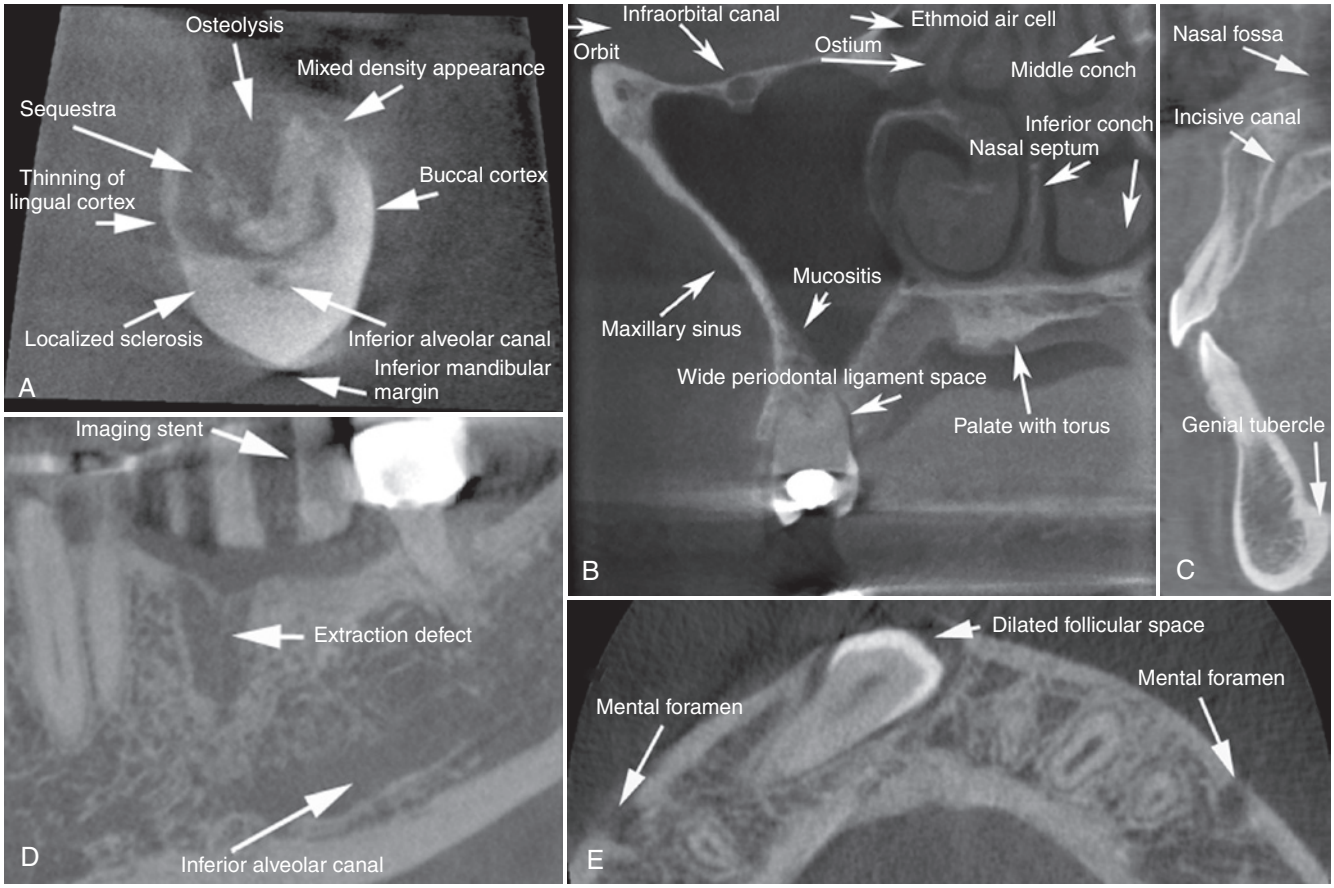

FIG. 2-26 **A** to **W**, Anatomic landmarks on CBCT images acquired using CS 9000, CS 9300, and i-CAT units. (Carestream Dental, Atlanta, Ga [CS 9000 and CS 9300 units]; and Imaging Sciences International, Hatfield, Pa [i-CAT unit].)

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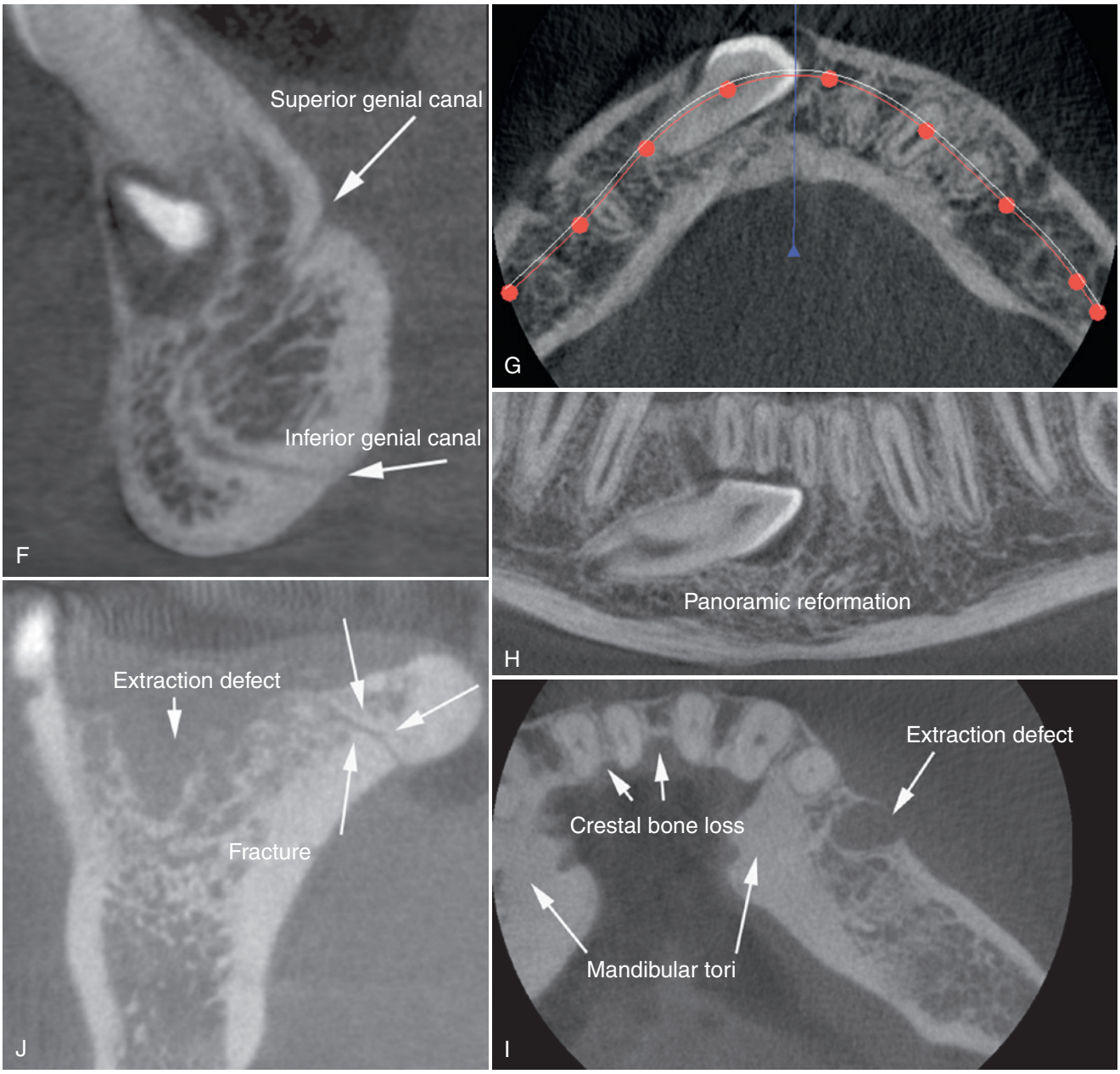

FIG. 2-26, cont'd

this relationship can best be assessed using CBCT.[93] A potential difference in the location of the mandibular canal with respect to age and gender has been reported.[97,167]

Outcomes Assessment

Endodontic treatment outcome predictors using PA radiographs and CBCT imaging have been shown to vary and also are influenced by patient inclusion and exclusion criteria.[104] Historically, PA radiographs and physical examinations were used to determine the success of endodontic treatment, and the absence of posttreatment periradicular radiolucencies and symptoms was considered the criterion for success. However, these planar imaging–based studies have resulted in an overestimation of successful outcomes, compared to CBCT assessments,[192a] because apical periodontitis confined within the cancellous bone or lesions covered by a thick cortex may be undetectable with conventional radiographic assessments.[18] Additional discrepancies between PA radiography and CBCT have resulted from geometric distortion, limiting comparisons of time-based evaluations, even with careful attention to paralleling technique factors.[112] A clinical study comparing the sensitivity, specificity, predictive values, and accuracy of PA and panoramic radiography and CBCT imaging in 888 consecutive patients showed that the prevalence of apical periodontitis in root-treated teeth was 17.6%, 35.3% and 63.3%, respectively. Conventional radiography showed increased accuracy when the evaluation of larger lesions was assessed.[53]

Outcome predictors identified with PA radiographs and CBCT scans may be different depending on the research performed. Liang et al.[104] retrospectively evaluated 115 endodontically treated teeth with vital pulps 2 years after treatment.

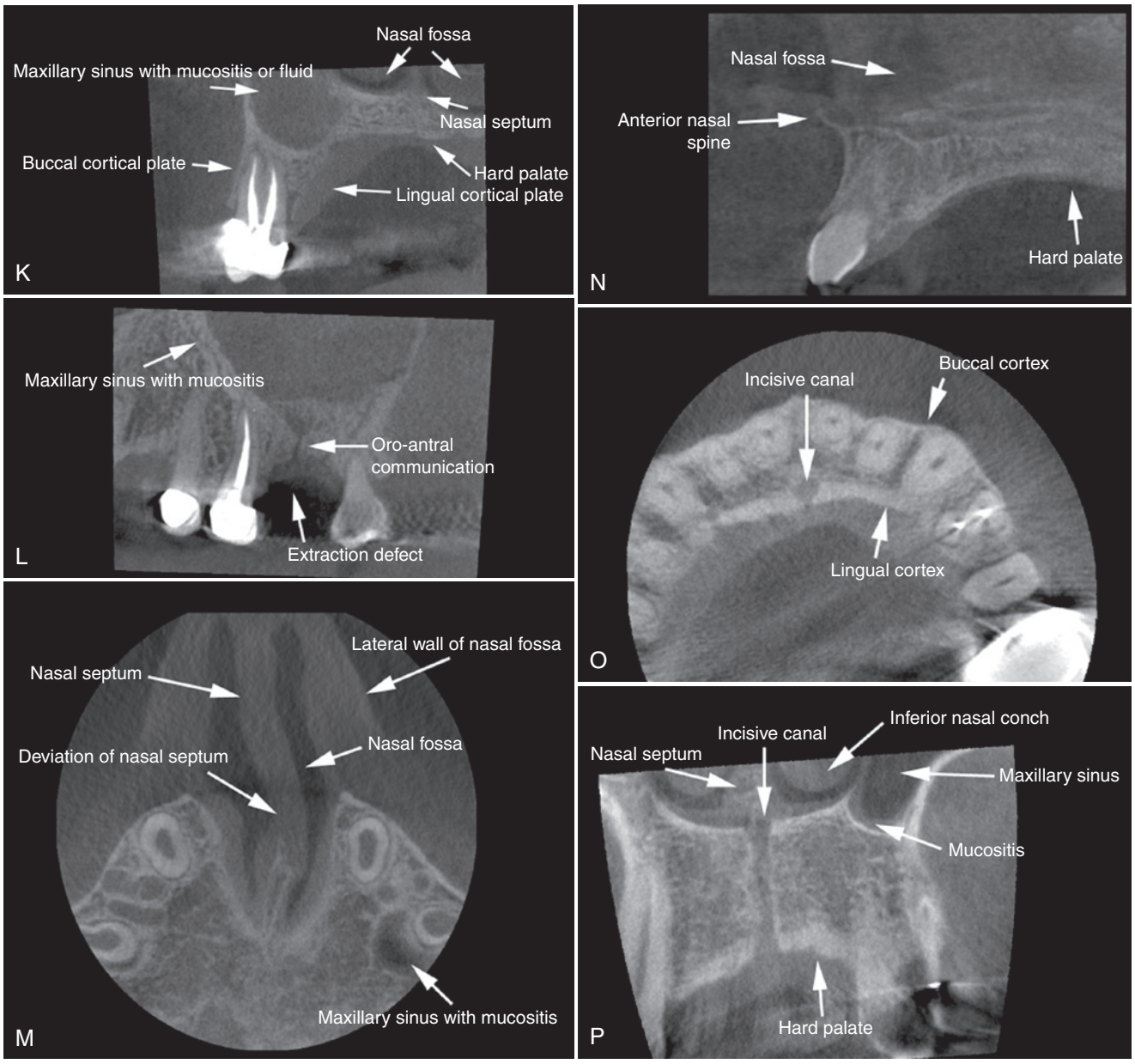

FIG. 2-26, cont'd

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The authors noted that the recall rate of 36% was comparable to that in other studies, but the result may have been affected because patients with symptomatic or already extracted teeth may not have responded. This lost-to-follow-up cohort significantly reduced the level of evidence.[59a] PA radiography identified periapical lesions in only 12.6% of teeth compared to CBCT images, which identified 25.9% of teeth with periapical lesions. In multivariate logistic regression analysis, the extent of root fillings and density were outcome predictors when using PA imaging, whereas density of root fillings and quality of the coronal restoration were outcome predictors when using CBCT scans. The use of cross-sectional tomography in endodontic treatment is not yet supported by high-level studies that show improved outcomes for patients.

The predictive value and diagnostic accuracy of radiologic assessments are critical to the practice of dentistry, and the diagnostic value of radiographs depends on the radiograph's ability to show the histology of apical periodontitis (AP). De Paula-Silva et al.[43] evaluated the periapex of 83 root-treated and untreated dogs' teeth using PA radiography, CBCT scans, and histopathologic analysis. PA radiography detected AP in 71% of roots; CBCT scans detected AP in 84%; and histologic analysis detected AP in 93%. These findings, corroborated by other studies,[27,70,150] emphasized the low negative predictive value (NPV) of PA radiography at 0.25, showing that when the periapical tissues had a normal appearance, 75% actually had AP. CBCT scans resulted in an NPV almost two times higher than that for PA radiography; however, CBCT scans were not able to detect some AP that was confined to the apical foramen or had little volumetric bone loss. The positive predictive value was the same for PA radiography and CBCT scans compared with histologic examination, but true positive and true

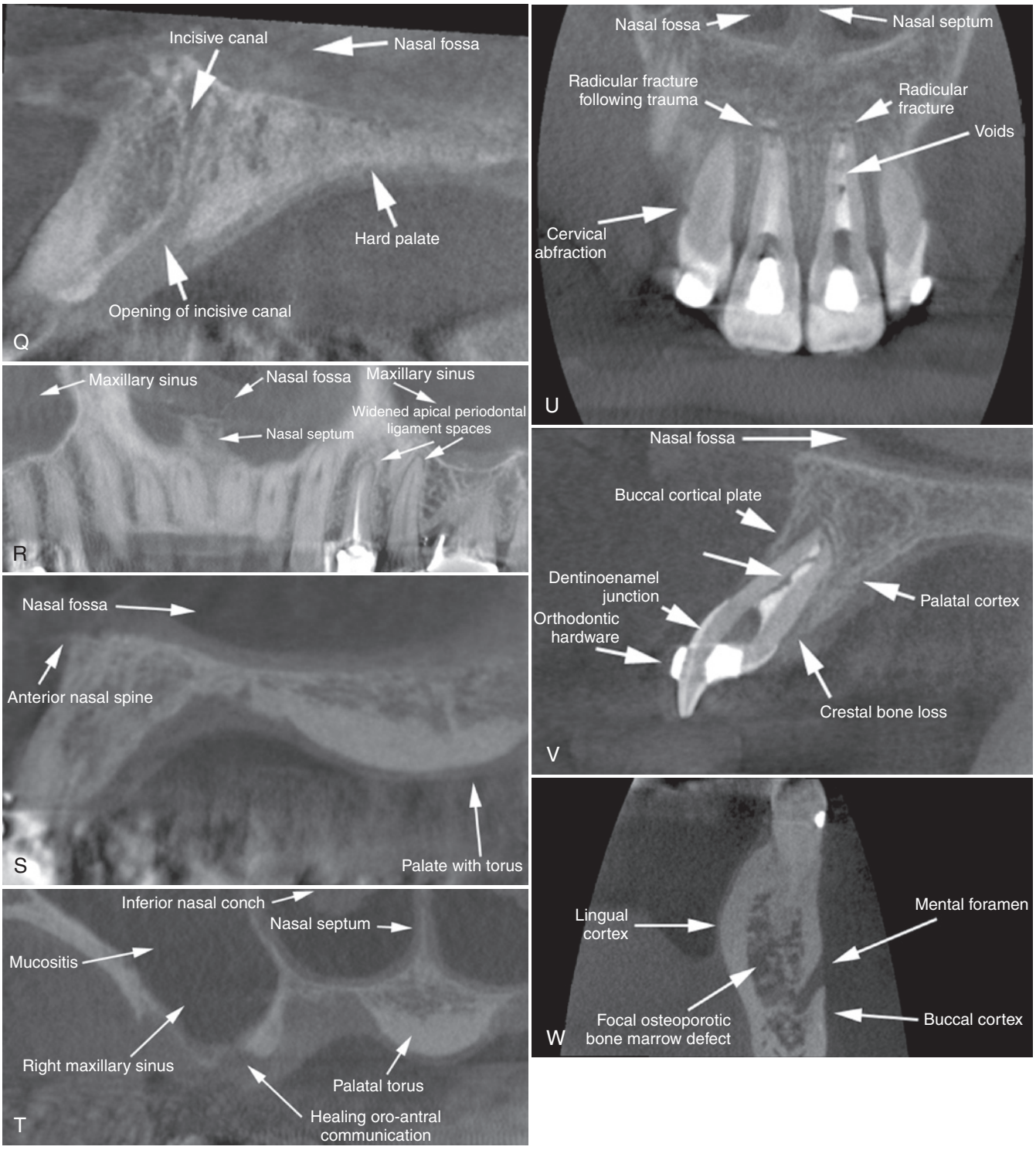
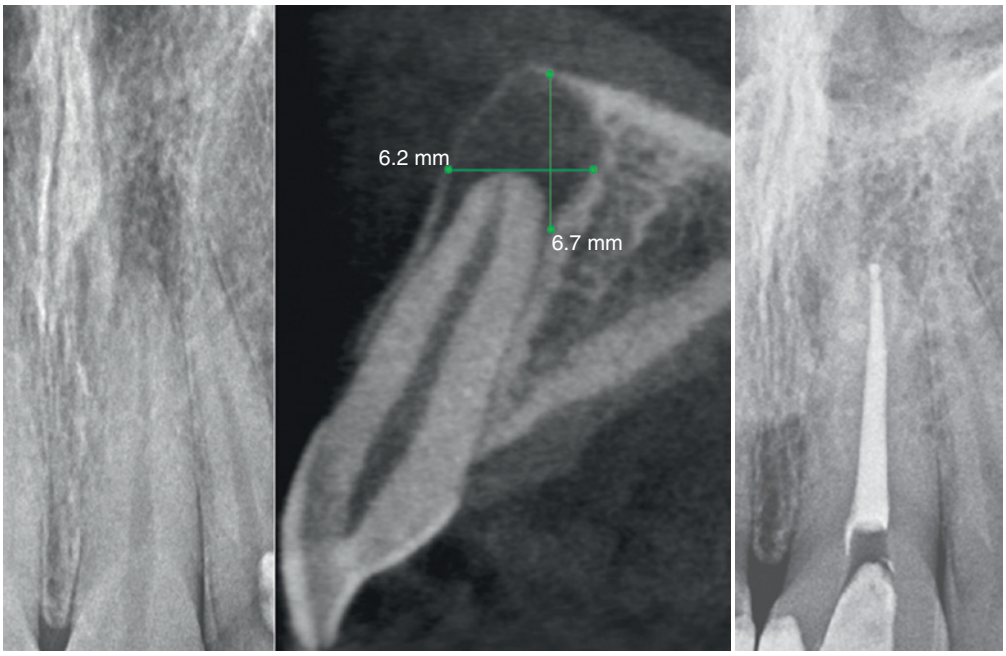


FIG. 2-26, cont'd

negative diagnosis of AP using CBCT scans occurred in 92% of cases.

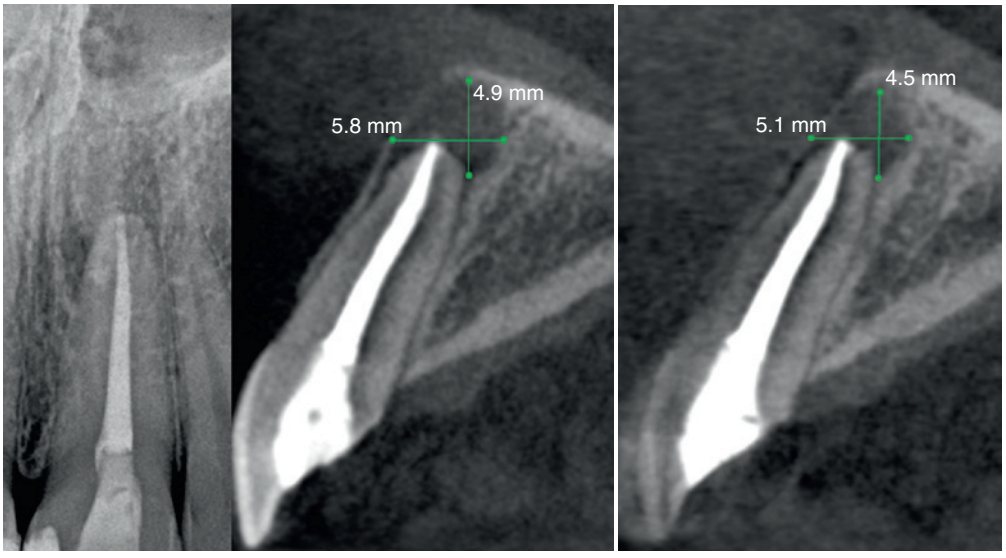
Determination of healing or nonhealing in individual cases should depend on the clinical and radiographic findings, adjusted for risk factors for the patient, radiation dose, and cost (Fig. 2-27). For patients who are medically complex and subject to possible increased detriment from apical periodontitis (i.e., patients with altered immune systems, such as those undergoing chemotherapy or anti-HIV protocols or who have risk factors associated with prosthetic joints and/or infective endocarditis), these factors should be considered in the decision on whether to use CBCT. The American Academy of Periodontology has published a position paper stating that periodontal disease might contribute to adverse systemic health conditions.[156] The scientific basis for the relationship of AP and adverse systemic health conditions has not been



A 02-03-2011 Initial PA

02-03-2011 Initial corrected sagittal view

B 02-14-2011 Obturation PA



05-18-2011 3 month check-up PA

C

05-18-2011 3 month check-up corrected sagittal view

10-10-2011 5 month check-up corrected sagittal view

D

FIG. 2-27 This series of radiographs shows a maxillary left central incisor several months after a bicycle accident. The patient complained of significant discomfort and mobility with slight swelling in the periapical region. The preoperative PA radiograph (**A**, *left*) and contemporaneous CBCT image (**A**, *right,* corrected sagittal view) showed an approximately 6 mm, well-defined periapical radiolucency with a noncorticated diffuse border, centered over the apex, consistent with a periradicular periodontitis. No root or alveolar fractures were observed. A postoperative PA radiograph (**B**) showed satisfactory canal obturation. At a 3-month checkup visit, the patient complained of continued sensitivity to touch and mobility. There was excessive contact with the opposing incisor, which was adjusted. Because the 3-month checkup PA radiograph (**C**, *left*) showed no change in the periradicular radiolucency, a CBCT image (**C**, *right,* corrected sagittal view) was required to assess healing. The periradicular radiolucency was smaller, consistent with progress in healing. A subsequent 5-month CBCT image (**D**, corrected sagittal view) showed continued healing. Consistent with the as low as reasonably achievable (ALARA) principle, no PA radiograph was exposed. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga].)

established[33]; however, new associations between AP and systemic health should be predicated on research that uses CBCT in the detection of endodontic disease.[193]

IMPLANT SITE ASSESSMENTS

Successful endosseous implant site assessment requires the development of a prosthetically driven approach,[130b] with special emphasis on the evaluation of bone volume, the osseous topography, and the location of anatomic structures in relation to positioning of the implant. The AAOMR has published advisory recommendations suggesting all radiographic studies should interface with the dental and medical histories, clinical examinations, and treatment planning. Panoramic radiography, which may be supplemented by PA radiography, should be used for initial imaging assessments. Cross-sectional imaging, including CBCT, should not be used as an initial imaging examination. The AAOMR affirms the need for cross-sectional imaging in the preoperative diagnostic phase, recommending CBCT because it provides the highest diagnostic yield at an acceptable radiation dose risk. CBCT should be used at the smallest FOV necessary, with optimized technique factors, to minimize radiation dose in accordance with the ALARA principle.[177a]

CBCT allows for precise planning and delivery of implants that can reproduce the anatomy with submillimetric accuracy, leading to improved outcomes.[171] The use of CBCT to assess linear measurements, proximity to vital anatomic structures, mapping of the alveolar ridge topography, and fabrication of surgical guides is supported by the dental literature. The use of CBCT to gauge bone density, provide intraoperative surgical navigation, and assess implant integration is generally considered an area that requires further research.[17]

Virtual implant planning using CBCT data allows clinicians to visualize the result before the commencement of treatment, facilitating the virtual investigation of multiple treatment scenarios until the best plan is attained. The evaluation of bone dimensions, bone quality, the long axis of the alveolar bone, internal anatomy, and jaw boundaries; the detection of pathologic features; and the transfer of radiographic information are the main imaging goals. Pathoses of the jaws, such as retained root tips, inflammatory lesions, cysts, and tumors, in addition to extraoral structures, such as the sinuses and temporomandibular joints (TMJs), must also be assessed.[80] CBCT imaging should be considered to evaluate implant sites in the region of teeth with a high likelihood of periradicular pathosis.[200]

IMAGE PERCEPTION AND VIEWING ENVIRONMENT

Medical image perception is an important area of knowledge, and ongoing research relies on an understanding of perceptual issues, such as psychological factors, dwell time, visual search physiology, search tactics, appreciation for the reading environment, and fatigue factors, to increase search satisfaction. Understanding these issues may improve the ability to interpret and report on dental radiographic findings.[155a]

The increasing use of digital radiography in the dental environment has led to a sea change in workflow and the necessity for new ways to view and document radiographic images. Simple to accomplish, but important to improve, are the viewing conditions for softcopy interpretation, including moderately reduced ambient lighting, ranging from 25 to 40 lux.[110a]

FUTURE OF CBCT

The first decade of the twenty-first century saw the development of a wide range of CBCT applications, especially in dentistry. Lower radiation dose, higher spatial resolution, smaller FOV, and relatively lower cost may contribute to CBCT becoming the standard of care in 3D dentomaxillofacial imaging in selected cases. CBCT systems are increasingly being used in medical applications, such as operating rooms, emergency departments, intensive care units, and private otolaryngology offices. Operating room–based C-arm systems have been in use for a number of years, with applications in interventional angiography, cancer surgery, vascular surgery, orthopedic surgery, neurosurgery, and radiotherapy.[55] Applications in otolaryngology and mammography are common, and imaging of extremities in weight-bearing scenarios is under development. Many of these applications rely on systems that use task-specific protocols that benefit from CBCT's 2D flat panel detectors, which allow for a single rotation of the source to generate a study of the region of interest, as opposed to complex MDCTs that use redundant imaging via multiple slice acquisitions to generate a 3D volume.[164]

The introduction of new, high-performance, flat-panel detectors and software algorithms centering on improving the noise-power spectrum and noise-equivalent quanta will continue to increase the utility of CBCT systems in the future. Areas of research include (1) image perception and image quality assessment, to better understand how physicians and dentists analyze radiographic images and thereby improve diagnostic decision making[172]; (2) iterative reconstruction that uses sophisticated algorithms to reduce artifacts; (3) known-component reconstructions that use a model-based 3D image reconstruction and iterative software to reduce image artifacts in the presence of metallic devices such as screws and implants; (4) image registration to align tissues for image-guided surgery and outcomes assessment,[120] (5) image-guided procedures that provide up-to-the-minute surgical navigation; and (6) segmentation to allow discrimination between normal and diseased tissues and permit volumetric measurements (Fig. 2-28).

CONCLUSIONS

Digital radiography has several advantages and has become an indispensable diagnostic tool for many dentists in daily practice. Once the digital image appears on the monitor, the dental x-ray software allows image enhancement, which should be used with caution and based on the diagnostic task. Inappropriate use of enhancement has been shown to adversely affect diagnosis.[121] If digital radiographs are exported using various software packages created for graphic design and image manipulation, digital information can be altered, added, or removed. The DICOM standard has been accepted as the universal standard for image transmission and archiving, so that each image can be transmitted and stored without the use of proprietary software that would seriously limit its distribution. DICOM ensures that all images are readable in any viewing software without loss of fidelity or diagnostic information. Image enhancement features of digital radiography allow

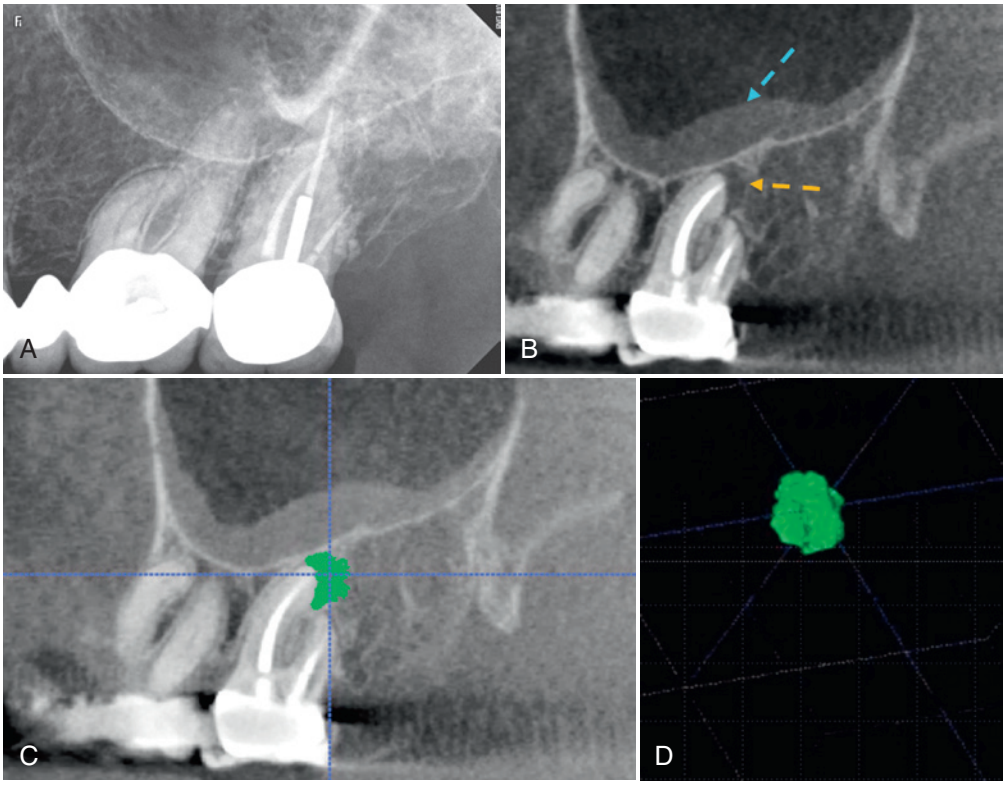


FIG. 2-28 The problem of superimposition of unrelated structures onto the features of interest is reduced when tomographic slices are used instead of images in which an entire volume of data is compressed into a planar image. This PA radiographic image **(A)** of the maxillary left second molar shows no radiographic indicators of pathosis. A contemporaneously exposed CBCT image (**B**, corrected sagittal view) shows a 4.3 × 1.9 mm, well-defined unilocular, noncorticated area of low attenuation centered over the apex of the mesiobuccal root, consistent with a periradicular periodontitis *(yellow arrow).* There is a moderate mucositis in the region of the maxillary sinus adjacent to this tooth *(blue arrow).* Experimental semiautomated segmentation of this image using active contour methods (ITK-SNAP) allowed for measurement of the true volume of the lesion **(C)** and for future temporal comparisons based on volumetric measurements **(D).** This lesion measured 85,112 voxels and 38.1044 cubic mm. (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, Ga]; volumetric segmentation and measurement using ITK-SNAP [Radiology Department, School of Medicine, University of Pennsylvania].)

mishandling of images, leading to potential abuse. Published studies illustrate the potential for fraudulent use of digital radiography.[31]

There is a dearth of studies related to the diagnostic performance of the different sensor types currently available on the market. Slight to moderate differences in spatial resolution capabilities exist. With rapid advancement in sensor technology and frequent software upgrades, selection of one system over another for a specific diagnostic task may appear challenging. A review of the most commonly used solid-state sensors notes that most systems perform comparably with intraoral film and also allow for postprocessing of images, which is not possible with film-based images. Other factors that assume significance in this context are the availability of technical and customer support, frequency of both hardware and software upgrades, dimensions of the sensor and its active area, number of sensors needed in a practice (and thus cost issues), the detector quantum efficiency (DQE), and conformance to the DICOM standard for seamless integration with other systems. CCD/CMOS sensors appear to offer the best contrast and spatial resolution, in addition to facilitating instantaneous image capture, and therefore are recommended for endodontic applications. Careful and appropriate image processing further helps tease out the signal of interest. In an enterprise-wide setting or in larger private practices that have multiple specialty areas, PSP-based sensors may be more cost effective for large volume imaging (e.g., full mouth series of radiographs). However, at least one or two CCD/CMOS-based systems should be available for faster image acquisition, such as for endodontic purposes and intraoperative procedures.

It is recommended that the literature be constantly reviewed for updates on digital radiography and advanced imaging modalities for specific endodontic applications because hardware and software upgrades continue to make rapid progress. Previous studies have shown that most digital images performed comparably to conventional intraoral film for a variety of diagnostic tasks. Most of these studies were done with earlier generation sensors. The advances in sensor technology have resulted in greatly enhanced image quality, and this trend is expected to continue. Also of interest in the future will be the use of task-based, appropriate image processing parameters

that result in a reduced radiation dose and significant enhancement of the diagnostic information. Automation of this process will result in faster and more consistent image processing based on the diagnostic task. Such procedures are routinely carried out in medical radiology.

Three-dimensional imaging will continue to be used extensively as sensor characteristics improve and more user-friendly software is introduced. As bit-depth and spatial resolution of images increase, CBCT will continue to be explored for more applications in endodontics. Image interpretation also is important. Occult pathology and incidental findings in adjacent regions can be easily missed or go unrecognized by those who have not received specific training in the interpretation of regional anatomy. Image processing can greatly alter signal characteristics, thus rendering the task fairly challenging. Besides, if other pathoses are discovered, additional imaging may be necessary, including MRI, nuclear medicine studies, or even MDCT for evaluation of soft tissues, with and without the use of contrast.

The advent of 3D imaging has provided the endodontist with tools that were not available until now, facilitating interactive image manipulation and enhancement and thus significantly increasing the amount of information that can be gleaned from a volume. Lack of distortion, magnification, artifacts associated with conventional radiography, and the relative low radiation dose compared to medical-grade CT will result in more clinicians adopting such technology to enable accurate diagnoses and treatment planning, in addition to long-term follow-up and the evaluation of healing. Judicious use of CBCT and all other imaging modalities using ionizing radiation is advocated. The AAE/AAOMR position paper provides recommendations for the use of CBCT in endodontics.[34] That position paper is presented at the end of this chapter.

When the clinician works with different vendor products, it is important to have a quality assurance program in place. This is not being done currently. Additionally, accreditation of stand-alone imaging laboratories is now a requirement for reimbursement of medical and dental diagnostic procedures from government agencies and some major third-party insurance providers. Several states are considering enforcing this requirement to prevent abuse of CBCT.

Likewise, definitive referral criteria are lacking. Indications, contraindications, and choice of alternate imaging modalities need to be considered before CBCT is used. There is a learning curve to this technology, and appropriate positioning, choice of exposure parameters (and thus the effective dose), reconstruction schemes, choice of postprocessing algorithms based on diagnostic task, voxel sizes, and cost must be considered. The literature contains few studies to help us formulate definitive guidelines for the use of CBCT in dentistry.

It is equally important to record the doses associated with each study. Accreditation criteria have been developed by the Intersocietal Accreditation Commission for cone beam CT in dentistry that are useful for ensuring the safe use of these units. The lowest possible dose must be imparted to the patient as part of a radiologic examination to minimize stochastic effects that have no known threshold for expression. No dose can be considered a "safe dose." The benefits of any radiographic study must outweigh the risks. All studies must be interpreted fully because signals from adjacent areas may appear in the volume of interest, including small FOV studies. Retakes can be avoided by adhering to protocol selection based on the task at hand. The ALARA principle must be followed, regardless of the dose values reported by the vendor, to optimize the dose for the specific examination. Use of thyroid collars and lead aprons is recommended in the NCRP guidelines, as long as they do not interfere with image acquisition.

Joint Position Statement of the American Association of Endodontists and the American Academy of Oral and Maxillofacial Radiology on the Use of Cone Beam Computed Tomography in Endodontics: 2015 Update

This statement was prepared by the Special Committee to Revise the Joint AAE/AAOMR Position Statement on Use of CBCT in Endodontics, and approved by the AAE Board of Directors and AAOMR Executive Council in May 2015.

INTRODUCTION

This updated joint position statement of the American Association of Endodontists (AAE) and the American Academy of Oral and Maxillofacial Radiology (AAOMR) is intended to provide scientifically-based guidance to clinicians regarding the use of cone beam computed tomography (CBCT) in endodontic treatment and reflects new developments since the 2010 statement.[1] The guidance in this statement is not intended to substitute for a clinician's independent judgment in light of the conditions and needs of a specific patient.

Endodontic disease adversely affects quality of life and can produce significant morbidity in afflicted patients. Radiography is essential for the successful diagnosis of odontogenic and non-odontogenic pathoses, treatment of the root canal systems of a compromised tooth, biomechanical instrumentation, evaluation of final canal obturation, and assessment of healing.

Until recently, radiographic assessments in endodontic treatment were limited to intraoral and panoramic radiography. These radiographic technologies provide two-dimensional representations of three-dimensional anatomic structures. If any element of the geometric configuration is compromised, the image may demonstrate errors.[2] In more complex cases, radiographic projections with different beam angulations can allow parallax localization. However, complex anatomy and surrounding structures can render interpretation of planar images difficult.

The advent of CBCT has made it possible to visualize the dentition, the maxillofacial skeleton, and the relationship of anatomic structures in three dimensions.[3] CBCT, as with any technology, has known limitations, including a possible higher radiation dose to the patient. Other limitations include potential for artifact generation, high levels of scatter and noise, and variations in dose distribution within a volume of interest.[4]

CBCT should be used only when the patient's history and a clinical examination demonstrate that the benefits to the patient outweigh the potential risks. CBCT should not be used routinely for endodontic diagnosis or for screening purposes in the absence of clinical signs and symptoms. Clinicians should use CBCT only when the need for imaging cannot be met by lower dose two-dimensional (2D) radiography.

VOLUME SIZE(S)/FIELD OF VIEW

There are numerous CBCT equipment manufacturers, and several models are available. In general, CBCT is categorized into large, medium and limited-volume units based on the size of their "field of view" (FOV). The size of the FOV describes the scan volume of CBCT machines. That volume determines the extent of anatomy included. It is dependent on the detector size and shape, beam projection geometry, and the ability to collimate the beam. To the extent practical, FOV should only slightly exceed the dimensions of the anatomy of interest.

Generally, the smaller the FOV, the lower the dose associated with the study. Beam collimation limits the radiation exposure to the region of interest and helps ensure that an optimal FOV can be selected based on disease presentation. Smaller scan volumes generally produce higher resolution images. Because endodontics relies on detecting small alterations such as disruptions in the periodontal ligament space, optimal resolution should be sought.[5]

The principal limitations of large FOV CBCT imaging are the size of the field irradiated and the reduced resolution compared to intraoral radiographs and limited volume CBCT units with inherent small voxel sizes.[5] The smaller the voxel size, the higher is the spatial resolution. Moreover, the overall scatter generated is reduced due to the limited size of the FOV. Optimization of the exposure protocols keeps doses to a

minimum without compromising image quality. If a low-dose protocol can be used for a diagnostic task that requires lower resolution, it should be employed, absent strong indications to the contrary.

In endodontics, the area of interest is limited and determined prior to imaging. For most endodontic applications, limited FOV CBCT is preferred to medium or large FOV CBCT because there is less radiation dose to the patient, higher spatial resolution, and shorter volumes to be interpreted.

DOSE CONSIDERATIONS

Selection of the most appropriate imaging protocol for the diagnostic task must be consistent with the ALARA principles that every effort should be made to reduce the effective radiation dose to the patient "as low as reasonably achievable." Because radiation dose for a CBCT study is higher than that for an intraoral radiograph, clinicians must consider overall radiation dose over time. For example, will acquiring a CBCT study now eliminate the need for additional imaging procedures in the future? It is recommended to use the smallest possible FOV, the smallest voxel size, the lowest mA setting (depending on the patient's size), and the shortest exposure time in conjunction with a pulsed exposure-mode of acquisition.

If extension of pathoses beyond the area surrounding the tooth apices or a multifocal lesion with possible systemic etiology is suspected, and/or a non-endodontic cause for devitalization of the tooth is established clinically, appropriate larger field of view protocols may be employed on a case-by-case basis.

There is a special concern with overexposure of children (up to and including 18 year-olds) to radiation, especially with the increased use of CT scans in medicine. The AAE and the AAOMR support the Image Gently Campaign led by the Alliance for Radiation Safety in Pediatric Imaging. The goal of the campaign is, "to change practice; to raise awareness of the opportunities to lower radiation dose in the imaging of children." Information on use of CT is available at http://www.imagegently.org/Procedures/ComputedTomography.aspx

INTERPRETATION

If a clinician has a question regarding image interpretation, it should be referred to an oral and maxillofacial radiologist.[6]

RECOMMENDATIONS

The following recommendations are for limited FOV CBCT scans.

Diagnosis

Endodontic diagnosis is dependent upon thorough evaluation of the patient's chief complaint, history, and clinical and radiographic examination. Preoperative radiographs are an essential part of the diagnostic phase of endodontic therapy. Accurate diagnostic imaging supports the clinical diagnosis.

Recommendation 1: Intraoral radiographs should be considered the imaging modality of choice in the evaluation of the endodontic patient.

Recommendation 2: Limited FOV CBCT should be considered the imaging modality of choice for diagnosis in patients who present with contradictory or non-specific clinical signs and symptoms associated with untreated or previously endodontically-treated teeth.

Rationale:

- In some cases, the clinical and planar radiographic examinations are inconclusive. Inability to confidently determine the etiology of endodontic pathosis may be attributed to limitations in both clinical vitality testing and intraoral radiographs to detect odontogenic pathoses. CBCT imaging has the ability to detect periapical pathosis before it is apparent on 2D radiographs.[7]
- Preoperative factors such as the presence and true size of a periapical lesion play an important role in endodontic treatment outcome. Success, when measured by radiographic criteria, is higher when teeth are endodontically treated before radiographic signs of periapical disease are detected.[8]
- Previous findings have been validated in clinical studies in which primary endodontic disease detected with intraoral radiographs and CBCT was 20% and 48%, respectively. Several clinical studies had similar findings, although with slightly different percentages.[9,10] Ex vivo experiments in which simulated periapical lesions were created yielded similar results.[11,12] Results of in vivo animal studies, using histologic assessments as the gold standard also showed similar results observed in human clinical and ex-vivo studies.[13]
- Persistent intraoral pain following root canal therapy often presents a diagnostic challenge. An example is persistent dentoalveolar pain also known as atypical odontalgia.[14] The diagnostic yield of conventional intraoral radiographs and CBCT scans was evaluated in the differentiation between patients presenting with suspected atypical odontalgia vs. symptomatic apical periodontitis, without radiographic evidence of periapical bone destruction.[15] CBCT imaging detected 17% more teeth with periapical bone loss than conventional radiography.

Initial Treatment

Preoperative:

Recommendation 3: Limited FOV CBCT should be considered the imaging modality of choice for initial treatment of teeth with the potential for extra canals and suspected complex morphology, such as mandibular anterior teeth, and maxillary and mandibular premolars and molars, and dental anomalies.

Intraoperative:

Recommendation 4: If a preoperative CBCT has not been taken, limited FOV CBCT should be considered as the imaging modality of choice for intra-appointment identification and localization of calcified canals.

Postoperative:

Recommendation 5: Intraoral radiographs should be considered the imaging modality of choice for immediate postoperative imaging.

Rationale:

- Anatomical variations exist among different types of teeth. The success of non-surgical root canal therapy depends on

identification of canals, cleaning, shaping and obturation of root canal systems as well as quality of the final restoration.

- 2D imaging does not consistently reveal the actual number of roots and canals. In studies, data acquired by CBCT showed a very strong correlation between sectioning and histologic examination.[16,17]
- In a 2013 study, CBCT showed higher mean values of specificity and sensitivity when compared to intraoral radiographic assessments in the detection of the MB2 canal.[18]

Non-Surgical Retreatment

Recommendation 6: Limited FOV CBCT should be considered the imaging modality of choice if clinical examination and 2D intraoral radiography are inconclusive in the detection of vertical root fracture (VRF).

Rationale:

- In non-surgical retreatment, the presence of a vertical root fracture significantly decreases prognosis. In the majority of cases, the indication of a vertical root fracture is more often due to the specific pattern of bone loss and periodontal ligament space enlargement than direct visualization of the fracture. CBCT may be recommended for the diagnosis of vertical root fracture in unrestored teeth when clinical signs and symptoms exist.
- Higher sensitivity and specificity were observed in a clinical study where the definitive diagnosis of vertical root fracture was confirmed at the time of surgery to validate CBCT findings, with sensitivity being 88% and specificity 75%.[19] Several case series studies have concluded that CBCT is a useful tool for the diagnosis of vertical root fractures. In vivo and laboratory studies[20,21] evaluating CBCT in the detection of vertical root fractures agreed that sensitivity, specificity, and accuracy of CBCT were generally higher and reproducible. The detection of fractures was significantly higher for all CBCT systems when compared to intraoral radiographs. However, these results should be interpreted with caution because detection of vertical root fracture is dependent on the size of the fracture, presence of artifacts caused by obturation materials and posts, and the spatial resolution of the CBCT.

Recommendation 7: Limited FOV CBCT should be the imaging modality of choice when evaluating the non-healing of previous endodontic treatment to help determine the need for further treatment, such as non-surgical, surgical or extraction.

Recommendation 8: Limited FOV CBCT should be the imaging modality of choice for non-surgical re-treatment to assess endodontic treatment complications, such as overextended root canal obturation material, separated endodontic instruments, and localization of perforations.

Rationale:

- It is important to evaluate the factors that impact the outcome of root canal treatment. The outcome predictors identified with periapical radiographs and CBCT were evaluated by Liang et al.[22] The results showed that periapical radiographs detected periapical lesions in 18 roots (12%) as compared to 37 on CBCT scans (25%). 80% of apparently short root fillings based on intraoral radiographs images appeared flush on CBCT. Treatment outcome, length and density of root fillings and outcome predictors determined by CBCT showed different values when compared with intraoral radiographs.
- Accurate treatment planning is an essential part of endodontic retreatment. Incorrect, delayed or inadequate endodontic diagnosis and treatment planning places the patient at risk and may result in unnecessary treatment. Treatment planning decisions using CBCT versus intraoral radiographs were compared to the gold standard diagnosis.[23] An accurate diagnosis was reached in 36%-40% of the cases with intraoral radiographs compared to 76%-83% with CBCT. A high level of misdiagnosis was noted in invasive cervical resorption and vertical root fracture. In this study, the examiners altered their treatment plan after reviewing the CBCT in 56%-62.2% of the cases, thus indicating the significant influence of CBCT.

Surgical Retreatment

Recommendation 9: Limited FOV CBCT should be considered as the imaging modality of choice for pre-surgical treatment planning to localize root apex/apices and to evaluate the proximity to adjacent anatomical structures.

Rationale:

The use of CBCT has been recommended for treatment planning of endodontic surgery.[24,25] CBCT visualization of the true extent of periapical lesions and their proximity to important vital structures and anatomical landmarks is superior to that of periapical radiographs.

Special Conditions

a. Implant Placement:

Recommendation 10: Limited FOV CBCT should be considered as the imaging modality of choice for surgical placement of implants.[26]

b. Traumatic Injuries:

Recommendation 11: Limited FOV CBCT should be considered the imaging modality of choice for diagnosis and management of limited dento-alveolar trauma, root fractures, luxation, and /or displacement of teeth and localized alveolar fractures, in the absence of other maxillofacial or soft tissue injury that may require other advanced imaging modalities.[27]

c. Resorptive Defects:

Recommendation 12: Limited FOV CBCT is the imaging modality of choice in the localization and differentiation of external and internal resorptive defects and the determination of appropriate treatment and prognosis.[28,29]

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Special Committee to Revise the Joint AAE/AAOMR Position Statement on Use of Limited FOV CBCT in Endodontics

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CHAPTER 3

Case Selection and Treatment Planning

PAUL A. ROSENBERG | MATTHEW MALEK

CHAPTER OUTLINE

Common Medical Findings That May Influence Endodontic Treatment Planning
- *Cardiovascular Disease*
- *Diabetes*
- *Pregnancy*
- *Malignancy*
- *Medication-Related Osteonecrosis of the Jaws (MRONJ)*
- *Human Immunodeficiency Virus (HIV) and Acquired Immunodeficiency Syndrome (AIDS)*
- *End-Stage Renal Disease and Dialysis*
- *Prosthetic Implants*
- *Behavioral and Psychiatric Disorders*
- *Psychosocial Evaluation*

Development of the Endodontic Treatment Plan

Endodontic Prognosis
- *Single-Visit versus Multiple-Visit Treatment*

Interdisciplinary Treatment Planning
- *Periodontal Considerations*
- *Surgical Considerations*
- *Restorative and Prosthodontic Considerations*
- *Endodontic Therapy or Dental Implant*

Other Factors That May Influence Endodontic Case Selection
- *Anxiety*
- *Scheduling Considerations*

The process of case selection and treatment planning begins after a clinician has diagnosed an endodontic problem. The clinician must determine whether the patient's oral health needs are best met by providing endodontic treatment and maintaining the tooth or by advising extraction. The use of rotary instruments, ultrasonics, and microscopy as well as new materials has made it possible to predictably retain teeth that previously would have been extracted. In addition, even teeth that have failed initial endodontic treatment can often be successfully retreated using nonsurgical or surgical procedures.

Increased knowledge concerning the importance of anxiety control, premedication with a nonsteroidal anti-inflammatory drug (NSAID) or acetaminophen, profound local anesthesia, appropriate occlusal adjustment, and biology-based clinical procedures enables clinicians to complete endodontic procedures without intraoperative or posttreatment pain.

Questions concerning tooth retention and possible referral can be answered only after a complete patient evaluation. The evaluation must include assessment of medical, psychosocial, and dental factors as well as consideration of the relative complexity of the endodontic procedure. Although most medical conditions do not contraindicate endodontic treatment, some can influence the course of treatment and require specific modifications. A number of valuable texts are available that review the subject of dental care for the medically compromised patient.[14,48,110] The American Academy of Oral Medicine (Edmonds, WA) has an excellent website (www.aaom.com) that can be used to elicit information about medically compromised patients.

Perhaps the most important advice for a clinician who plans to treat a medically compromised patient is to be prepared to communicate with the patient's physician. The proposed treatment can be reviewed, and medical recommendations should be documented. Fig. 3-1 depicts a sample medical consultation letter that can be modified as necessary.

The American Society of Anesthesiologists (ASA; Park Ridge, IL) Physical Status Classification system is commonly used to express medical risk (Box 3-1). The ASA classification system remains the most widely used assessment method for preanesthetic patients despite some inherent limitations to its use as a peritreatment risk predictor. This classification system is a generally accepted and useful guide for pretreatment assessment of relative risk but does not advise appropriate treatment modifications. The clinician should go beyond the classification system and gather more information from the patient and physician, including the patient's compliance with suggested medication, frequency of physician visits, and most recent visit.

Typical questions include the following: *Do you take medication as prescribed by your physician?* Or, *when was the last time you were examined by your physician?* Other systems have been proposed that would better reflect the increasing number of

Michael White, MD
1 Walker Street
Brown City, OK

Dear Dr. White,

Your patient, Ms. Mary Smith, presented for consultation on August 10, 2009, concerning tooth #19. The tooth is asymptomatic at this time, but a small (4 mm x 3 mm), well-circumscribed periradicular radiolucency associated with the palatal root was noted on radiographic examination. The tooth was tested for vitality using thermal and electrical modalities and found to be nonvital, indicating an odontogenic cause for the lesion. The tooth will require endodontic treatment in order to be maintained. The prognosis for nonsurgical endodontic treatment in this case is good. I anticipate that her dental medication plan would include lidocaine with epinephrine for anesthesia and ibuprofen for postoperative pain control.

In a review of the patient's medical history she noted that she is being treated for a malignancy of the thyroid gland and is undergoing radiation therapy. She was unable to provide more specific information about her treatment.

I would appreciate information regarding her ability to undergo endodontic treatment at this time. Please call me if there is further information that you would require concerning the dental treatment to be provided. Thank you.

Yours truly,

Peter Jones, DDS

FIG. 3-1 Sample medical consultation letter.

BOX 3-1

American Society of Anesthesiologists Physical Status Classification System

P1: Normal, healthy patient; no dental management alterations required
P2: Patient with mild systemic disease that does not interfere with daily activity or who has a significant health risk factor (e.g., smoking, alcohol abuse, gross obesity)
P3: Patient with moderate to severe systemic disease that is not incapacitating but may alter daily activity
P4: Patient with severe systemic disease that is incapacitating and a constant threat to life

From: www.asahq.org/clinical/physicalstatus.htm.

medically complex patients treated by clinicians as Americans live longer.[35] Regardless of the classification system used, these generalized guidelines need to be individualized for the patient under care.

An alternative means of considering risk assessment is to review the following issues:

- History of allergies
- History of drug interactions, adverse effects
- Presence of prosthetic valves, joints, stents, pacemakers, and so on
- Antibiotics required (prophylactic or therapeutic)
- Hemostasis (normal expected, modification to treatment)
- Patient position in chair
- Infiltration or block anesthesia with or without vasoconstrictor
- Significant equipment concerns (radiographs, ultrasonics, electrosurgery)
- Emergencies (potential for occurrence, preparedness)
- Anxiety (past experiences and management strategy)

A review of these areas provides the clinician with essential background data before initiating treatment.

COMMON MEDICAL FINDINGS THAT MAY INFLUENCE ENDODONTIC TREATMENT PLANNING

Cardiovascular Disease

Patients with some forms of cardiovascular disease are vulnerable to physical or emotional stress that may be encountered during dental treatment, including endodontics. Patients may be confused or ill informed concerning the specifics of their particular cardiovascular problem. In these situations, consultation with the patient's physician is mandatory before the initiation of endodontic treatment. "For patients with symptoms of unstable angina or those who have had an MI [myocardial infarction] within the past 30 days (major risk category), elective care should be postponed."[48] One study found "no significant increase in the risk of experiencing a second vascular event after dental visits, including those that involved invasive procedures, in periods up to 180 days after a first recorded ischemic stroke, transient ischemic attack (TIA) or acute MI."[94]

The use of vasoconstrictors in local anesthetics poses potential problems for patients with ischemic heart disease. In these patients, local anesthetics without vasoconstrictors may be used as needed. If a vasoconstrictor is necessary, patients with intermediate clinical risk factors (i.e., a past history of MI without ischemic symptoms) and those taking nonselective beta-blockers can safely be given up to 0.036 mg epinephrine (two cartridges containing 1:100,000 epinephrine) at one appointment. For patients at higher risk (i.e., those who have

had an MI within the past 7 to 30 days and unstable angina), the use of vasoconstrictors should be discussed with the physician.[48]

Vasoconstrictors may interact with some antihypertensive medications and should be used only after consultation with the at-risk patient's physician. Local anesthetic agents with minimal or no vasoconstrictors are usually adequate for nonsurgical endodontic procedures[48] (see also Chapter 4). A systematic review of the cardiovascular effects of epinephrine concluded that the increased risk for adverse events among uncontrolled hypertensive patients was low, and the reported adverse events associated with epinephrine use in local anesthetics was minimal.[4] Another review highlighted the advantages of including a vasoconstrictor in the local anesthesia and stated that "pain control was significantly impaired in those patients receiving the local anesthetic without the vasoconstrictor as compared to those patients receiving the local anesthetic with vasoconstrictor."[13]

A patient who has specific heart conditions may be susceptible to an infection of the heart valves, induced by a bacteremia. This infection is called *infective* or *bacterial endocarditis* and is potentially fatal. In 2008, the American College of Cardiology and American Heart Association (AHA) Task Force on Practice Guidelines published an update on their previous guidelines, which focused on infectious endocarditis. This guideline stated that "prophylaxis against infective endocarditis is reasonable for the following patients at highest risk for adverse outcomes from infective endocarditis who undergo dental procedures that involve manipulation of either gingival tissue or the periapical region of teeth or perforation of the oral mucosa: patients with prosthetic cardiac valves or prosthetic material used for cardiac valve repair . . . , patients with previous infective endocarditis . . . [and] patients with congenital heart disease."[71]

The specific recommendations are summarized in a reference guide by the American Association of Endodontists (AAE; Chicago, IL), found online at www.aae.org/uploadedfiles/publications_and_research/guidelines_and_position_statements/antibioticprophylaxsisquickrefguide.pdf). Because the AHA periodically revises its recommended antibiotic prophylactic regimen for dental procedures, it is essential that the clinician stay current concerning this important issue. There is a low compliance rate among at-risk patients regarding their use of the suggested antibiotic coverage before dental procedures. Therefore, the clinician must question patients concerning their compliance with the prescribed prophylactic antibiotic coverage before endodontic therapy. If a patient has not taken the antibiotic as recommended, it may be administered up to 2 hours after the procedure.[71]

Patients with artificial heart valves are considered susceptible to bacterial endocarditis. Consulting the patient's physician in such cases regarding antibiotic premedication is essential. Some physicians elect to administer parenteral antibiotics in addition to or in place of the oral regimen.

A dentist may be the first to detect elevated blood pressure if he or she routinely evaluates blood pressure before treatment. Furthermore, patients receiving treatment for hypertension may not be controlled adequately because of poor compliance or inappropriate drug therapy. Abnormal blood pressure readings may be the basis for physician referral.

Some patients may be disposed to serious life-threatening complications due to stress. Acute heart failure during a stressful dental procedure in a patient with significant valvular disease and heart failure or the development of infectious endocarditis represent two such life-threatening disorders.[91] Careful evaluation of patients' medical histories including the cardiac status of patients, the use of appropriate prophylactic antibiotics, and stress reduction strategies will minimize the risk of serious cardiac sequelae.

There is a widespread belief among dentists and physicians that oral anticoagulation therapy in which patients receive drugs such as warfarin (Coumadin) must be discontinued before dental treatment to prevent serious hemorrhagic complications, especially during and after surgical procedures. Aspirin is a drug commonly used as an anticoagulant on a daily basis without the supervision of a physician. Clinical studies do not support the routine withdrawal of anticoagulant therapy before dental treatment for patients who are taking such medications.[10,39,48]

When patients report they are receiving an anticoagulant medication, they can benefit from the clinician using the following guidelines:

- Identify the reason why the patient is receiving anticoagulant therapy.
- Assess the potential risk versus benefit of altering the drug regimen.
- Know the laboratory tests used to assess anticoagulation levels (i.e., the international normalized ratio [INR] value should be 3.5 or less for patients who are taking warfarin to safely undergo dental or surgical endodontic procedures).[48] Be familiar with methods used to obtain hemostasis both intraoperatively and postoperatively.
- Be familiar with the potential complications associated with prolonged or uncontrolled bleeding.
- Consult the patient's physician to discuss the proposed dental treatment and to determine the need to alter the anticoagulant regimen.

Another cardiac complication may occur in patients with Hodgkin disease or breast cancer, who often receive irradiation to the chest as an element of treatment. Although the therapy often cures the malignancy, it has been implicated in causing late-onset heart disease that may influence the development of a treatment plan and subsequent treatment. Dentists must identify patients who have received irradiation to the chest and consult with patients' physicians to determine whether that therapy has damaged the heart valves or coronary arteries. Patients with radiation-induced valvular disease may require prophylactic antibiotics when undergoing specific dental procedures that are known to cause a bacteremia and a heightened risk of developing endocarditis. Patients with radiation-induced coronary artery disease should be administered only limited amounts of local anesthetic agents containing a vasoconstrictor. They may require the administration of sedative agents and cardiac medications to preclude ischemic episodes. Consultation with the patient's physician is an appropriate response when a patient presents with a history that includes prior radiation to the chest.[29]

Diabetes

The Centers for Disease Control and Prevention (CDC, Atlanta, GA) in 2011 reported that 25.8 million people, or 8.3% of the U.S. population, have diabetes. There were also about 1.9 million people aged 20 years or older newly diagnosed with diabetes in 2010. (See Centers for Disease Control and

Prevention *Diabetes Fact Sheet*, available at http://cdc.gov/diabetes/pubs/estimates11.htm#2, 2011.) Diabetes is the seventh leading cause of death in the United States,[61] and, according to the *Diabetes Fact Sheet*, the risk for death among people with diabetes is about twice that of people of similar age but without diabetes. It is likely that patients with diabetes who require endodontic treatment will be increasingly common.

Diabetes mellitus appears to have multiple causes and several mechanisms of pathophysiology.[48] It can be thought of as a combination of diseases that share the key clinical feature of glucose intolerance. Patients with diabetes, even those who are well controlled, require special consideration during endodontic treatment. The patient with well-controlled diabetes, who is free of serious complications such as renal disease, hypertension, or coronary atherosclerotic disease, is a candidate for endodontic treatment. However, special considerations exist in the presence of acute infections. The non–insulin-controlled patient may require insulin, or the insulin dose of some insulin-dependent patients may have to be increased.[79] When surgery is required, consultation with the patient's physician is advisable in order to consider adjustment of the patient's insulin dosage, antibiotic prophylaxis, and dietary needs during the posttreatment period.

The clinician should ask patients with diabetes who self-monitor their glucose levels to bring a glucometer to each visit. If pretreatment glucose levels are below normal fasting range (80 to 120 mg/dl), it may be appropriate to take in a carbohydrate source.[100] A source of glucose (e.g., glucose tablets, orange juice, or soda) should be available if signs of insulin shock (hypoglycemic reaction caused by overcontrol of glucose levels) occur.[48] Signs and symptoms of hypoglycemia include confusion, tremors, agitation, diaphoresis, and tachycardia.[100] The clinician can avoid a hypoglycemic emergency by taking a complete, accurate history of the time and amount of the patient's insulin and meals. When questions arise concerning the appropriate course to follow, the patient's physician should be contacted or treatment deferred.

Appointments should be scheduled with consideration given to the patient's normal meal and insulin schedule.[79] Usually, a patient with diabetes who is well managed medically and is under good glycemic control without serious complications such as renal disease, hypertension, or coronary atherosclerotic heart disease can receive any indicated dental treatment.[57] However, patients with diabetes who have serious medical complications may need a modified dental treatment plan. For instance, although prophylactic antibiotics generally are not required, it "may be prescribed a patient with brittle (very difficult to control) diabetes for whom an invasive procedure is planned but whose oral health is poor and the fasting plasma glucose exceeds 200 mg/dL."[48] Local anesthesia would not be an issue in the presence of well-controlled diabetes, "but for patients with concurrent hypertension or history of recent myocardial infarction, or with a cardiac arrhythmia, the dose of epinephrine should be limited to no more than two cartridges containing 1 : 100,000 epinephrine."[48]

Inadequate diabetic control may predispose such patients to several oral infections, including dental pulp infection.[45] One study determined that although apical periodontitis may be significantly more prevalent in untreated teeth in patients with type 2 diabetes, the disease does not seem to influence the response to root canal treatment.[52] However, other studies suggest that diabetes is associated with a decrease in the success of endodontic treatment in cases with pretreatment periradicular lesions.[12,28] In a prospective study on the impact of systemic diseases on the risk of tooth extraction, it has been also shown that an increased risk of tooth extraction after nonsurgical root canal treatment was significantly associated with diabetes mellitus, hypertension, and coronary heart disease.[103] Patients with diabetes and other systemic diseases may be best served by referral to an endodontist for treatment planning.

Pregnancy

Although pregnancy is not a contraindication to endodontics, it does modify treatment planning. Protection of the fetus is a primary concern when administration of ionizing radiation or drugs is considered. Of all the safety aids associated with dental radiography, such as high-speed film, digital imaging, filtration, and collimation, the most important is the protective lead apron with thyroid collar.[5,107] Although drug administration during pregnancy is a controversial subject, Box 3-2 presents commonly used dental drugs usually compatible with both pregnancy and breast-feeding. Based on U.S. Food and Drug Administration pregnancy risk factor definitions,[35] local anesthetics administered with epinephrine generally are considered safe for use during pregnancy and are assigned to the pregnancy risk classification categories B and C. (See www.fda.gov/Drugs/DevelopmentApprovalProcess/DevelopmentResources/Labeling/ucm093310.htm.) Few anxiolytics are considered safe to use during pregnancy. However, a single, short-term exposure to nitrous oxide–oxygen (N_2O-O_2) for less than 35 minutes is not thought to be associated with any human fetal anomalies, including low birth weight.[48] If a need exists for antibiotic therapy, penicillins, cephalosporins, and macrolides are considered first-line agents.

The analgesic of choice during pregnancy had been acetaminophen (category B).[78] However, a link between acetaminophen and childhood asthma has been suggested. Research has found that "the use of acetaminophen in middle to late but not early pregnancy may be related to respiratory symptoms in the first year of life."[74] This finding, although not completely validated, should be discussed with pregnant patients when an analgesic is being considered. Aspirin and nonsteroidal anti-inflammatory drugs also convey risks for constriction of the ductus arteriosus, as well as for postpartum hemorrhage and delayed labor.[78]

A major concern is that a drug may cross the placenta and be toxic or teratogenic to the fetus. In addition, any drug that is a respiratory depressant can cause maternal hypoxia, resulting in fetal hypoxia, injury, or death. Ideally, no drug should be administered during pregnancy, especially during the first trimester. If a specific situation makes adherence to this rule

BOX 3-2

Partial List of Drugs Usually Compatible with Both Pregnancy and Breast-Feeding

- Local anesthetics including lidocaine, etidocaine, and prilocaine
- Many antibiotics including penicillins, clindamycin, and azithromycin
- Acetaminophen
- Acyclovir
- Prednisone
- Antifungals including fluconazole and nystatin

difficult, then the clinician should review the appropriate current literature and discuss the case with the physician and patient.[11,55,60]

Further considerations exist during the postpartum period if the mother breast-feeds her infant. A clinician should consult the responsible physician before using any medications for the nursing mother. Alternative considerations include using minimal dosages of drugs, having the mother bank her milk before treatment, having her feed the child before treatment, or suggesting the use of a formula for the infant until the drug regimen is completed. Limited data are available on drug dosages and the effects on breast milk.[48]

In terms of treatment planning, elective dental care is best avoided during the first trimester because of the potential vulnerability of the fetus. The second trimester is the safest period in which to provide routine dental care. Complex surgical procedures are best postponed until after delivery.

Malignancy

Some malignancies may metastasize to the jaws and mimic endodontic pathosis, whereas others can be primary lesions (Fig. 3-2). The most common malignancies metastasize to the jaws are breast, lung, thyroid, and prostate.[53] A panoramic radiograph and a cone-beam computer tomography image are useful in providing an overall view of all dental structures. When a clinician begins an endodontic procedure on a tooth with a well-defined apical radiolucency, it might be assumed to result from a nonvital pulp. Pulp testing is essential to confirm a lack of pulp vitality in such cases. A vital response in such cases indicates a nonodontogenic lesion.

Careful examination of pretreatment radiographs from different angulations is important because lesions of endodontic origin would not be expected to be shifted away from the radiographic apex in the various images. Alternative methods, such a cone-beam computed tomography (CBC), may provide important diagnostic information (see Chapter 2).

A useful website for the differential diagnosis of radiographic lesions (Oral Radiographic Differential Diagnosis [ORAD] II) is available online at www.orad.org/index.html. A definitive diagnosis of periradicular lesions can be made only after biopsy. When a discrepancy exists between the initial diagnosis and clinical findings, consultation with an endodontist is advisable.

Patients undergoing chemotherapy or radiation to the head and neck may have impaired healing responses.[48] Treatment should be initiated only after the patient's physician has been

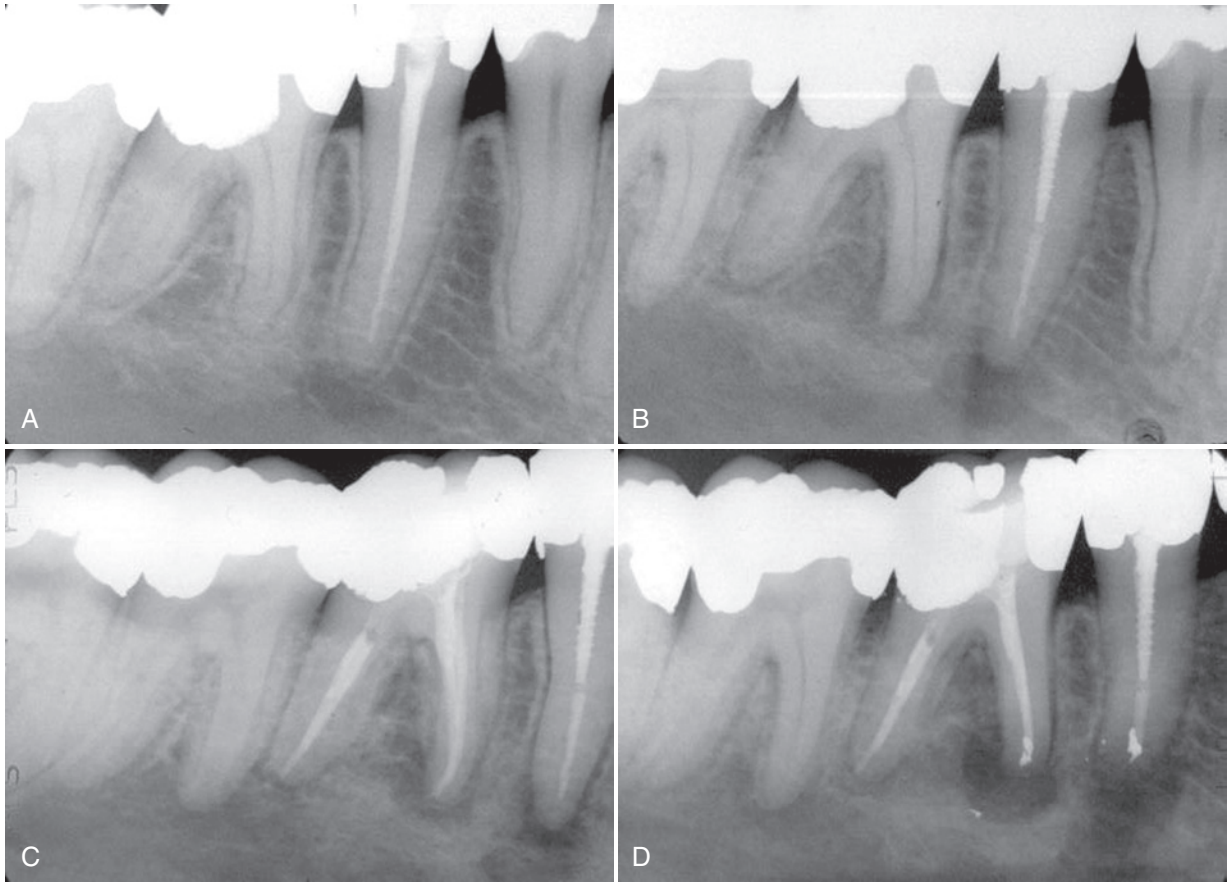

FIG. 3-2 **A**, Periapical view of tooth #29 after endodontic treatment by a general dental clinician. The diagnosis was irreversible pulpitis. **B**, Patient was referred to an endodontist 4 months later to evaluate radiolucencies of teeth #29 and #30. Symptoms indicated irreversible pulpitis of tooth #30, with concurrent lower right lip and chin paresthesia. Past medical history revealed breast cancer in remission. **C**, Nonsurgical endodontics was performed on tooth #30. Immediate referral was made to an oncologist/oral surgeon for biopsy to rule out nonodontogenic origin of symptoms. **D**, Surgical posttreatment radiograph of teeth #29 and #30. The biopsy report confirmed metastatic breast cancer. (Courtesy Dr. R. Sadowsky, Dr. L. Adamo, and Dr. J. Burkes.)

consulted. A dialogue among the dentist, physician, and patient is required prior to determining whether a tooth or teeth should be extracted or endodontically treated prior to radiation.

The effect of the external beam of radiation therapy on normal bone is to decrease the number of osteocytes, osteoblasts, and endothelial cells, thus decreasing blood flow. Pulps may become necrotic from this impaired condition.[48] Toxic reactions during and after radiation and chemotherapy are directly proportional to the amount of radiation or dosage of cytotoxic drug to which the tissues are exposed. Delayed toxicities can occur several months to years after radiation therapy.

Oral infections and any potential problems should be addressed before initiating radiation. It is advised that symptomatic nonvital teeth be endodontically treated at least 1 week before initiating radiation or chemotherapy, whereas treatment of asymptomatic nonvital teeth may be delayed.[48] The outcome of endodontic treatment should be evaluated within the framework of the toxic results of radiation and drug therapy. The white blood cell (WBC) count and platelet status of a patient undergoing chemotherapy should also be reviewed before endodontic treatment. In general, routine dental procedures can be performed if the granulocyte count is greater than 2000/mm^3 and the platelet count is greater than 50,000/mm^3. If urgent care is needed and the platelet count is below 50,000/mm^3, consultation with the patient's physician is required.[48]

Medication-Related Osteonecrosis of the Jaws (MRONJ)

Bisphosphonates offer great benefits to patients at risk of bone metastases and in the prevention and treatment of osteoporosis, although this and other drugs (e.g., denosumab) are associated with a rare occurrence of osteonecrosis.

To distinguish medication-related osteonecrosis of the jaws (MRONJ) from other delayed healing conditions, the following working definition of MRONJ has been adopted by the American Association of Oral and Maxillofacial Surgeons (AAOMS): Patients may be considered to have MRONJ if all of the following three characteristics are present (see American Association of Oral and Maxillofacial Surgeons: Position paper on Bisphosphonate-Related Osteonecrosis of the Jaw—2014 update, available at www.aaoms.org/docs/position_papers/mronj_position_paper.pdf?pdf=MRONJ-Position-Paper):

1. Current or previous treatment with an antiresorptive drug such as a bisphosphonate or an antiangiogenic drug (e.g., sunitinib [Sutent], sorafenib [Nexavar], bevacizumab (Avastin), or sirolimus (Rapamune)
2. Exposed, necrotic bone in the maxillofacial region that has persisted for more than 8 weeks
3. No history of radiation therapy to the jaws

A patient's risk of developing osteonecrosis of the jaw while receiving oral bisphosphonates appears to be low, but there are factors known to increase the risk for MRONJ (Box 3-3). According to the American Association of Endodontists (available at www.aae.org/uploadedFiles/Publications_and_Research/Guidelines_and_Position_Statements/bisphosonatesstatement.pdf, 2012), such risks include a history of taking bisphosphonates, especially intravenous (IV) formulations, previous history of cancer, and a history of a traumatic dental procedure. In addition to the usual risk factors, patients receiving high-dose IV bisphosphonates for more than 2 years are at most risk for developing osteonecrosis of the jaw.

BOX 3-3

Risk Factors for Development of Bisphosphonate-Associated Osteonecrosis

- History of taking bisphosphonates for more than 2 to 3 years, especially with intravenous therapy
- History of cancer, osteoporosis, or Paget disease
- History of traumatic dental procedure
- Patient more than 65 years of age
- History of periodontitis
- History of chronic corticosteroid use
- History of smoking
- History of diabetes

It has been reported that patients with multiple myeloma and metastatic carcinoma to the skeleton who are receiving intravenous, nitrogen-containing bisphosphonates are at greatest risk for osteonecrosis of the jaws. These patients represent 94% of published cases. The mandible is more commonly affected than the maxilla (2:1 ratio), and 60% of cases are preceded by a dental surgical procedure.[108] Treatment outcomes of MRONJ are unpredictable, and prevention strategies are extremely important.

Management of high-risk patients might include nonsurgical endodontic treatment of teeth that otherwise would be extracted. The combination of orthodontic extrusion and bloodless extraction—exfoliation of the extruded roots after their movement—has also been suggested with the aim of minimizing trauma and enhancing the health of the surrounding tissues in patients at risk of developing MRONJ or when a patient refuses to undergo conventional tooth extraction.[95]

For patients at higher risk of MRONJ, surgical procedures such as extractions, endodontic surgery, or placement of dental implants should be avoided. (See www.aae.org/uploadedFiles/Publications_and_Research/Guidelines_and_Position_Statements/bisphosonatesstatement.pdf, 2012.) Sound oral hygiene and regular dental care may be the best approach to lowering the risk of MRONJ. Patients taking bisphosphonates and undergoing endodontic therapy should sign an informed consent form, inclusive of the risks, benefits, and alternative treatment plans. The following recommendations have been suggested to reduce the risk of MRONJ associated with endodontic treatment[58]:

- Apply a 1-minute mouth rinse with chlorhexidine prior to the start of the treatment with the aim of lowering the bacterial load of the oral cavity.
- Avoid the use of anesthetic agents with vasoconstrictors in order to prevent impairment of tissue vascularization.
- Work under aseptic conditions, including removing of all caries and placement of rubber dam prior to intracanal procedures.
- Avoid damage to the gingival tissues during the placement of rubber dam.
- Avoid maintaining patency of the apical foramen to prevent bacteremia.
- Use techniques that reduce the risk of overfilling and overextension.

Aggressive use of systemic antibiotics is indicated in the presence of an infection in a patient taking bisphosphonates.[48] Discontinuing bisphosphonate therapy may not eliminate any

risk of developing MRONJ.[51,54,56] Some clinicians have proposed use of the CTX (C-terminal telopeptide of type I collagen α_1 chain) test (Quest Diagnostics, Madison, NJ) for assessing the risk of developing bone osteonecrosis (BON). For patients who have developed MRONJ, close coordination with an oral maxillofacial surgeon or oncologist is highly recommended.

An astute awareness of the potential risk of MRONJ in patients receiving bisphosphonate therapy is critical. Increased attentiveness to the prevention, recognition, and management of MRONJ will allow the clinician to make the best treatment decisions. Our knowledge of MRONJ is developing rapidly and it is essential that the clinician monitor the literature for changes in treatment protocols.[51,54,56]

Human Immunodeficiency Virus (HIV) and Acquired Immunodeficiency Syndrome (AIDS)

From 1987 through 1994, HIV disease mortality increased and reached a plateau in 1995. Subsequently, the mortality rate for this disease decreased an average of 33% per year from 1995 through 1998, and 5.5% per year from 1999 through 2009.[61] This dramatic improvement seems to be due to the use of a combination of highly active antiretroviral therapy (HAART) and improved preventive strategies.[48]

It is important, when treating patients with AIDS, that the clinician understand the patient's level of immunosuppression, drug therapies, and the potential for opportunistic infections. Although the effect of HIV infection on long-term prognosis of endodontic therapy is unknown, it has been demonstrated that clinicians may not have to alter their short-term expectations for periapical healing in patients infected with HIV.[77] The clinical team must also minimize the possibility of transmission of HIV from an infected patient, and this is accomplished by adherence to universal precautions. (See Universal Precautions for Prevention of Transmission of HIV and Other Bloodborne Infections, available at www.cdc.gov/niosh/topics/bbp/universal.html.)

Although saliva is not the main route for transmission of HIV, the virus has been found in saliva and its transmission through saliva has been reported.[33] Infected blood can transmit HIV, and during some procedures it may become mixed with saliva. Latex gloves and eye protection are essential for the clinician and staff. HIV can be transmitted by needlestick or via an instrument wound, but the frequency of such transmission is low, especially with small-gauge needles. Nevertheless, "Patients at high risk for AIDS and those in whom AIDS or HIV has been diagnosed should be treated in a manner identical to that for any other patient—that is, with standard precautions."[48]

A vital aspect of treatment planning for the patient with HIV/AIDS is to determine the current $CD4^+$ lymphocyte count and level of immunosuppression. In general, patients having a $CD4^+$ cell count exceeding 350 cells/mm^3 may receive all indicated dental treatments. Patients with a $CD4^+$ cell count of less than 200 cells/mm^3 or severe neutropenia (neutrophil count lower than 500/μL) will have increased susceptibility to opportunistic infections and may be effectively medicated with prophylactic drugs. White blood cell and differential counts, as well as a platelet count, should be ordered before any surgical procedure is undertaken. Patients with severe thrombocytopenia may require special measures (platelet replacement) before surgical procedures. Care in prescribing medications must also be exercised with any medications after which the patient may experience adverse drug effects, including allergic reactions, toxic drug reactions, hepatotoxicity, immunosuppression, anemia, serious drug interactions, and other potential problems. The practitioner should also be aware of oral manifestations of the disease as far as it concerns diagnosis and treatment planning. For instance, candidiasis of the oral mucosa, Kaposi sarcoma, hairy leukoplakia of the lateral borders of the tongue, herpes simplex virus (HSV), herpes zoster, recurrent aphthous ulcerations, linear gingival erythema, necrotizing ulcerative periodontitis, necrotizing stomatitis, oral warts, facial palsy, trigeminal neuropathy, salivary gland enlargement, xerostomia, and melanotic pigmentation are all reported to be associated with HIV infection. It is essential that consultation with the patient's physician occurs before performing surgical procedures or initiating complex treatment plans.[48,86]

End-Stage Renal Disease and Dialysis

Consultation with the patient's physician is important before dental care is initiated for patients being treated for end-stage renal disease. Depending on the patient's status and the presence of other diseases common to renal failure (e.g., diabetes mellitus, hypertension, and systemic lupus erythematosus), dental treatment may be best provided in a hospital setting. The goal of dental care for patients being treated for end-stage renal disease is to slow the progression of dental disease and preserve the patient's quality of life.[48,76]

The most recent American Heart Association guidelines do not include a recommendation for prophylactic antibiotics before invasive dental procedures for patients receiving dialysis with intravascular access devices, unless an abscess is being incised and drained.[3,48] Because controversy exists about the need for prophylactic antibiotics, consultation with the physician is important for patients receiving hemodialysis and those who have known cardiac risk factors. When prophylaxis is used, the standard regimen of the American Heart Association is recommended.[3]

Some drugs frequently used during endodontic treatment are affected by dialysis. Drugs metabolized by the kidneys and nephrotoxic drugs should be avoided. Both aspirin and acetaminophen are removed by dialysis and require a dosage adjustment in patients with renal failure. Amoxicillin and penicillin also require dosage adjustment as well as a supplemental dosage subsequent to hemodialysis.[76] It is advisable to consult the patient's physician concerning specific drug requirements during endodontic treatment. Endodontic treatment is best scheduled on the day after dialysis. On the day of dialysis, patients are generally fatigued and could have a bleeding tendency.[48]

Chronic renal failure is a disorder that may stimulate secondary hyperparathyroidism that can cause a variety of bone lesions. In some instances, these lesions appear in the periapical region of teeth and can lead to a misdiagnosis of a lesion of endodontic origin.[50]

Prosthetic Implants

Patients with prosthetic implants are frequently treated in dental practices. The question concerning the need for antibiotic prophylaxis to prevent infection of the prosthesis has been debated for many years. A statement was issued jointly in 2003 by the American Dental Association (ADA; Chicago, IL) and

the American Academy of Orthopaedic Surgeons (AAOS; Rosemont, IL) in an attempt to clarify the issue.[1] The statement concluded that scientific evidence does not support the need for antibiotic prophylaxis for dental procedures to prevent prosthetic joint infections. It went on to state that antibiotic prophylaxis is not indicated for dental patients with pins, plates, and screws, nor is it routinely indicated for most patients with total joint replacements. However, the statement indicated that some "high-risk patients" who are at increased risk for infection and undergoing dental procedures likely to cause significant bleeding should receive antibiotic prophylactic treatment. Such patients would include those who are immunocompromised or immunosuppressed, who have insulin-dependent (type 1) diabetes, who are in the first 2 years following joint replacement, or who have previous joint infections, malnourishment, or hemophilia.[1] The advisory statement concludes that the final decision on whether to provide antibiotic prophylaxis is the responsibility of the clinician, who must consider potential benefits and risks.[1] It should be noted that although endodontics has been shown to be a possible cause of bacteremia,[19,90] the risk is minimal in comparison with extractions, periodontal surgery, scaling, and prophylaxis.[72] In February 2009, the AAOS published a statement entitled "Antibiotic Prophylaxis for Bacteremia in Patients with Joint Replacements." In this updated publication it was stated: "Given the potential adverse outcomes and cost of treating an infected joint replacement, the AAOS recommends that clinicians consider antibiotic prophylaxis for all total joint replacement patients prior to any invasive procedure that may cause bacteremia." (See American Academy of Orthopaedic Surgeons: AAOS releases new statement on antibiotics after arthroplasty, www.aaos.org/news/aaosnow/may09/cover2.asp, 2012.)

However, the American Academy of Oral Medicine's (AAOM) position on this statement is that the "2009 information statement is more an opinion than an official guideline, AAOM believes that it should not replace the 2003 joint consensus statement prepared by the relevant organizations: the ADA, the AAOS and the Infectious Disease Society of America (IDSA)."[49] In 2012, an evidenced-based guideline was published that included recommendations of the AAOS-ADA clinical practice guideline for Prevention of Orthopaedic Implant Infection in Patients Undergoing Dental Procedures. This guideline stated that there is limited evidence for discontinuing the practice of routinely prescribing prophylactic antibiotics for patients with hip and knee prosthetic joint implants undergoing dental procedures. (See American Academy of Orthopaedic Surgeons: Prevention of orthopaedic implant infection in patients undergoing dental procedures, available at www.aaos.org/research/guidelines/PUDP/PUDP_guideline.pdf, 2012.)

Consultation with the patient's physician on a case-by-case basis is advisable to assess the need for prophylaxis.

Behavioral and Psychiatric Disorders

Stress reduction is an important factor in the treatment of patients with behavioral and psychiatric disorders. Sensitivity to the patient's needs must be part of the dental team's approach. Significant drug interactions and side effects are associated with tricyclic antidepressants, monoamine oxidase inhibitors, and antianxiety medications.[48] Consultation with physicians in such cases is essential before using sedatives, hypnotics, antihistamines, or opioids.

Psychosocial Evaluation

The initial visit, during which medical and dental histories are gathered, provides an opportunity to consider the patient's psychosocial status. Although some patients may want to maintain a tooth with a questionable prognosis, others may lack the ability to comprehend the potential risks and benefits. It would be a mistake to lead patients beyond what they can appreciate, and patients should not be allowed to dictate treatment that has a poor prognosis.

The clinician should also assess the patient's level of anxiety as an important part of preparation for the procedure to follow. It is reasonable to assume that most patients are anxious to some degree, especially when they are about to undergo endodontic treatment. A conversation describing the procedure and what the patient can expect is an important part of an anxiety-reduction protocol. It is well documented that a high level of anxiety is a predictor of poor anesthesia and posttreatment pain.[15,62] More than 200 studies indicate that behavioral intervention for the highly anxious patient before treatment decreases anxiety before and after surgery, reduces posttreatment pain, and accelerates recovery.[15]

DEVELOPMENT OF THE ENDODONTIC TREATMENT PLAN

The strategic value of a tooth in question should be considered before presenting alternative treatment plans to the patient. Although some decisions may be straightforward, considering alternative treatment options can be challenging as the clinician weighs multiple factors that will play a role in determining the ultimate success or failure of the case. Referral of the patient to a specialist should be considered when the complexity of a procedure is beyond the ability of a clinician. Factors that affect endodontic prognosis, including periodontal and restorative considerations, must be considered. The alternative of a dental implant is another choice when the endodontic prognosis is poor.

ENDODONTIC PROGNOSIS

Prognostic studies have identified a number of preoperative factors affecting the outcome of primary endodontic treatment. In a systematic review, it was determined that the absence of periapical radiolucency improves the outcome of root canal treatment significantly. The same study also showed that tooth vitality does not have an impact on the endodontic prognosis, as long as the periapex is healthy.[70] Studies have shown that the size of the radiolucency may also affect the outcome of endodontic treatment.[31,70] Another study found that the existence of sinus tract, narrow but deep periodontal probing depth, pain, and discharging sinus have a significant effect on the outcome of nonsurgical root canal treatment.[67] Preoperative pain is not only one of the most important predictors for postoperative pain,[64] it also has an impact on tooth survival after endodontic treatment.[68]

These findings suggest that all preexisting signs and symptoms that could affect the prognosis of treatment, along with the prognostic factors associated with other disciplines—which will be discussed in the next segment—should be taken into consideration when developing a treatment plan. It is of utmost importance that the prognosis and risks and benefits of the

treatment be relayed to the patient before the initiation of the treatment as well.

There is a general belief that the prognoses for retreatment cases are less than those for primary treatment, but this is not universally supported. In a systematic review it was suggested that the outcome of retreatment cases should be similar to treatment cases as long as access to the apical infection can be reestablished.[65] However, there is some evidence indicating that the incidence of postoperative pain and flare-up is higher in retreatment cases in comparison to treatments.[36] The presence of preoperative periapical lesion, apical extent of root filling, and quality of coronal restoration has been proved to significantly affect the outcome of retreatment cases.[65]

Retreatment cases offer a particular set of challenges to the clinician (Figs. 3-3 and 3-4), and this topic is covered extensively in Chapter 8. Important questions to be considered before retreatment include the following:

- Why did the treatment fail?
- Can the point of bacterial entry to the canal space be identified?
- Are prior radiographs available for review?
- Is there an obvious procedural problem that can be corrected?
- Is the canal system readily accessible for reentry?
- Are there additional factors (other than endodontic) that may have contributed to the failure?
- Is the tooth critical to the treatment plan?
- Does the patient understand the prognosis for the tooth and want to attempt retreatment?

A retreatment plan should be developed after the clinician has determined the cause of failure and weighed other factors that may affect the prognosis (e.g., root fracture, defective restoration) (Figs. 3-5 to 3-8). Retreatment cases may require surgical endodontics in combination with nonsurgical retreatment. Referral to a specialist is often helpful when planning treatment for complex cases. If retreatment (with or without surgery) on a tooth with a new restoration is being considered, it must be weighed against the possibility of an implant. Many variables must be considered before a reasonable conclusion can be reached.

Single-Visit versus Multiple-Visit Treatment

Some vital cases are suitable for single-visit treatment. The number of roots, time available, and the clinician's skills are factors to be considered. Severity of the patient's symptoms is

FIG. 3-3 Incision and drainage should be performed on this fluctuant swelling *(arrow)* in conjunction with canal instrumentation.

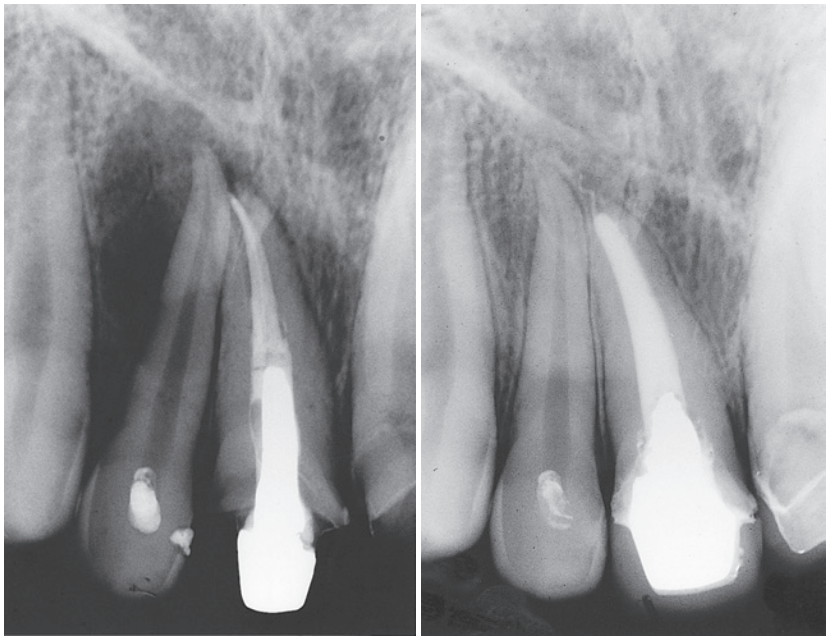

FIG. 3-4 Two years after endodontic therapy of tooth #8, the patient returned with pain and swelling. A clinician mistakenly began endodontic access on tooth #7, without confirming the apparent radiographic diagnosis by sensibility testing. Tooth #7 was vital, and tooth #8 was successfully retreated after removal of the post. (Courtesy Dr. Leon Schertzer.)

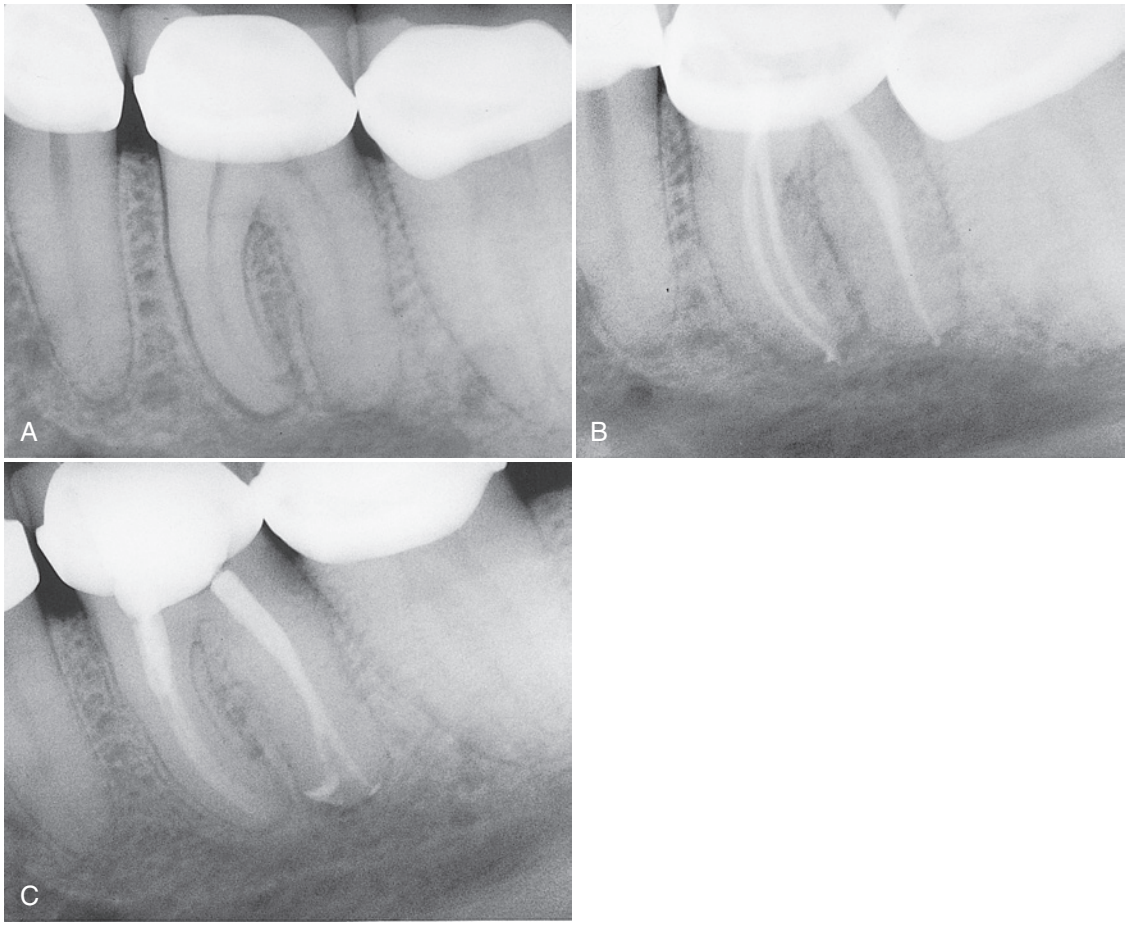

FIG. 3-5 Many years after endodontic treatment of tooth #19, the patient returned with a chief complaint of pain and an inability to chew with the tooth. Despite the radiographic appearance of excellent endodontic treatment, the tooth was retreated and the patient's pain disappeared. Note the unusual distal root anatomy, which was not apparent during the initial procedure. **A,** Initial radiograph. **B,** Completion of initial endodontic therapy. **C,** Retreatment.

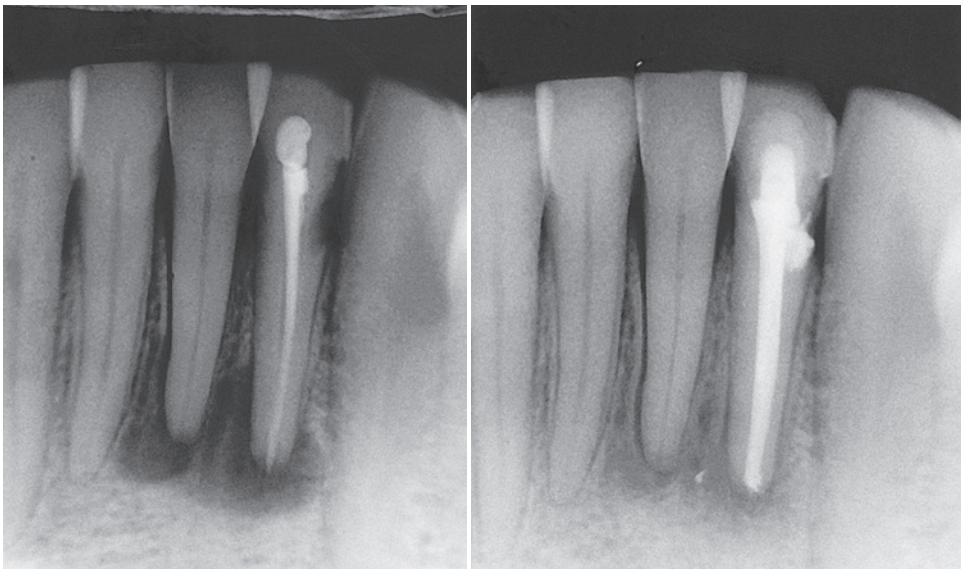

FIG. 3-6 Initial radiograph was misleading and implicated tooth #23 and tooth #24. Pulp testing indicated a vital pulp in tooth #24, and it was not treated. Retreatment of #23 resulted in healing of the periradicular lesion. (Courtesy Dr. Leon Schertzer.)

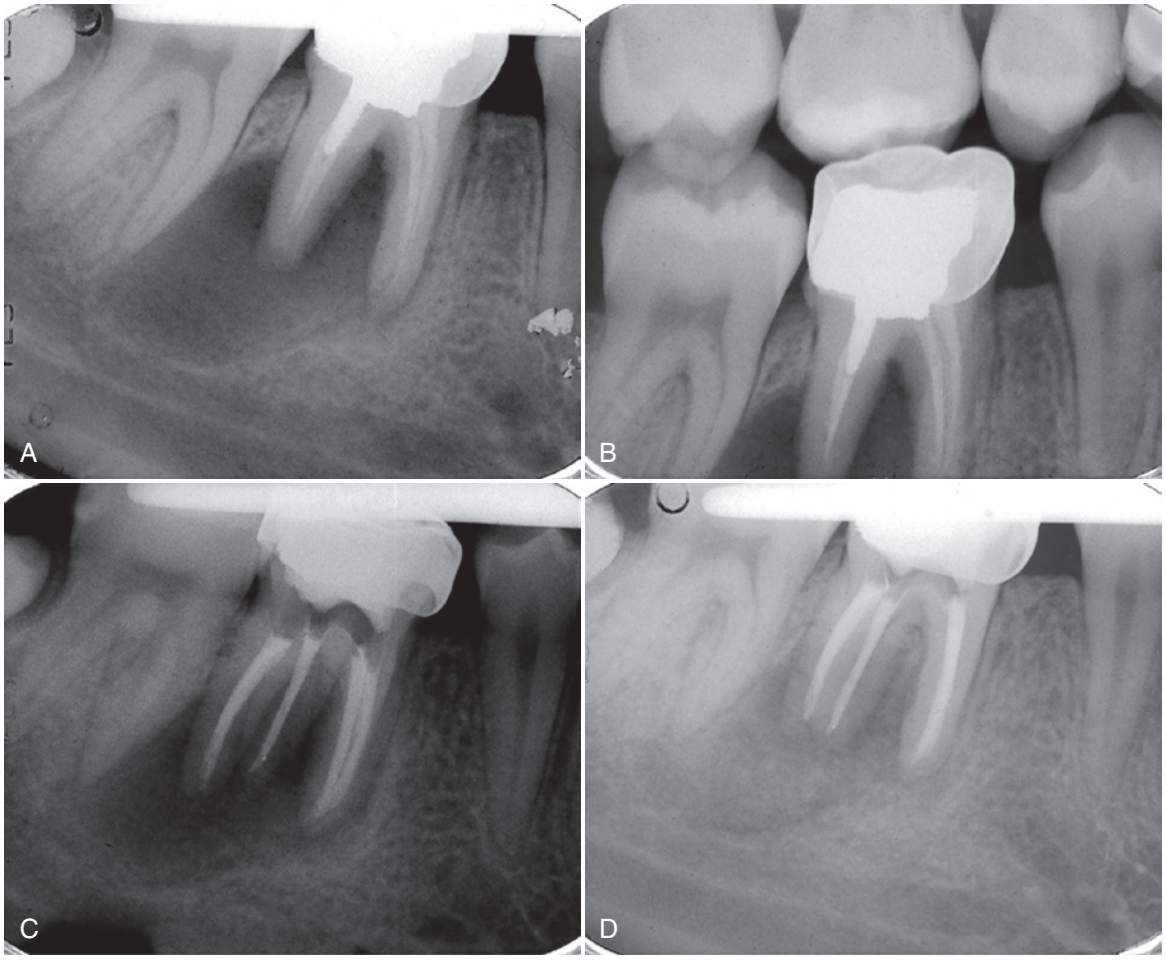

FIG. 3-7 Nonsurgical retreatment of tooth #30. An additional root was located and treated. **A**, Note inadequate endodontic treatment and large periapical lesion. **B**, Bitewing radiograph. **C**, Retreatment after post removal. **D**, Eighteen-month recall radiograph indicates periapical healing.

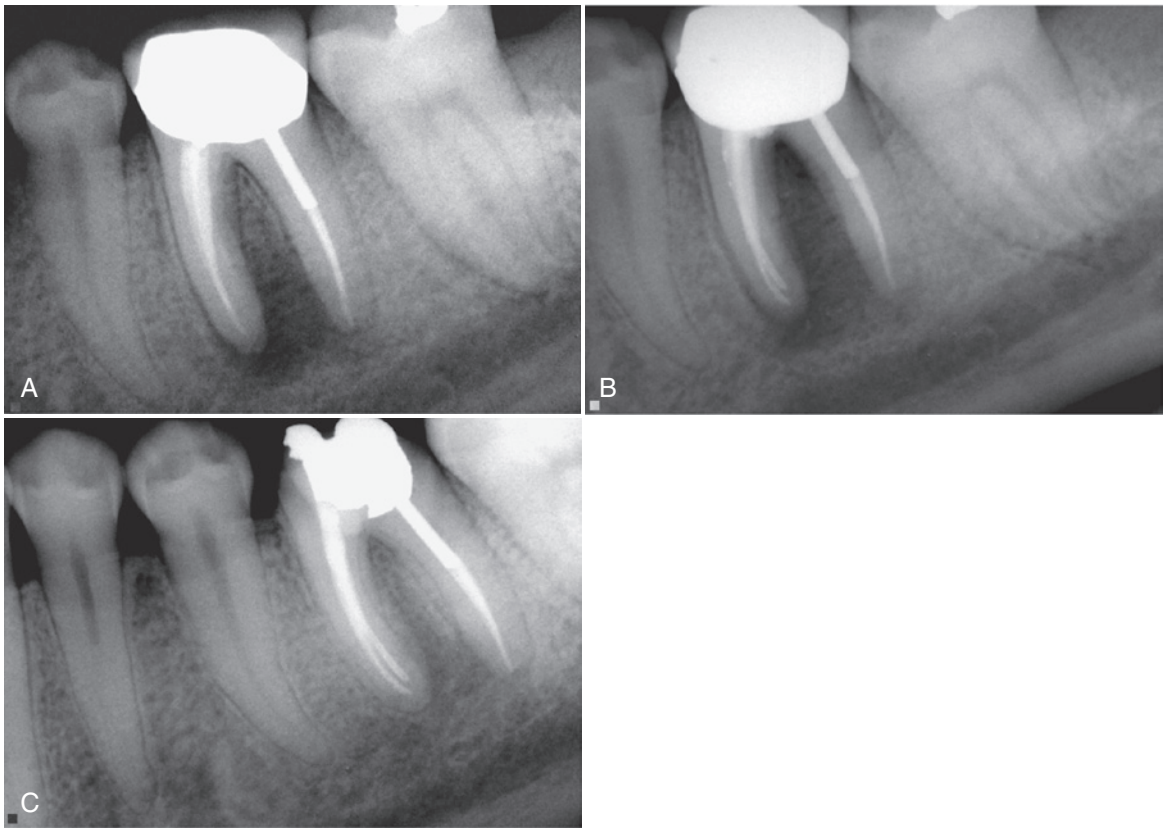

FIG. 3-8 Despite several exacerbations during endodontic retreatment, this case responded rapidly after the completion of therapy. Radiographic evaluation indicates a good periapical response after only 4 months.

another important consideration. For example, a patient in severe pain, with or without swelling, should not experience a long visit including access, instrumentation, and obturation. Treatment in such cases should be directed at alleviating pain, with filling of the canal postponed until a later visit. The clinician's judgment of what the patient can comfortably tolerate (regarding duration of the visit) is made on a case-by-case basis.

There are advantages in having a patient return for a second visit, after initially presenting with an endodontic emergency due to pain or swelling with or without a stoma. The second visit allows the clinician to determine the effect of the treatment on inflamed and infected tissues. Deferring the filling of the canal(s) leads to a shorter initial visit for the emergency patient.

Although some studies have reported less posttreatment pain in single-visit cases,[26,80,96] a systematic review found that the incidence of postobturation discomfort was similar in the single- and multiple-visit approaches.[27] In another systematic review it was concluded that there was a lack of compelling evidence indicating a significantly different prevalence of posttreatment pain/flare-up with either single- or multiple-visit root canal treatment.[85] Differences in research methodology explain the conflict in these findings.

Regarding the healing rate of single- and multiple-visit cases, a systematic review found that there was no detectable difference in the effectiveness of root canal treatment in terms of radiologic success between single and multiple visits.[27] A more recent systematic review also found that the healing rate of single- and multiple-visit root canal treatment is similar for infected teeth.[96]

Teeth with nonvital pulps and apical periodontitis pose a microbiologic problem. It is important to avoid pushing bacterial debris into the periapical tissues. Agreement is lacking concerning the appropriateness of single-visit endodontics for treating these patients. Some have postulated that the intervisit use of an antimicrobial dressing is essential to thoroughly disinfect the root canal system.[92,93,99] In contrast, other researchers have found no statistically significant difference in success when using the single-visit or multiple-visit approach to the nonvital tooth with apical periodontitis.[27,31,59,73,75,106] A systematic review found that, single-visit root canal treatment appeared to be slightly more effective than multiple-visit treatment (6.3% higher healing rate). However, the difference between these two treatment regimens was not statistically significant.[84] This is a complicated issue because the inability to detect differences between groups might also be due to variations in research methodology, including sample size, duration of follow-up, and treatment methods.

It is possible that total elimination of bacteria may not be absolutely necessary for healing. Perhaps maximal reduction of bacteria, effective root canal filling, and a timely satisfactory coronal restoration can result in a high level of clinical success. However, regardless of the number of appointments, effective bacteriologic disinfection of the root canal system is critical.[46]

Treatment planning for an endodontic case should be based on biologic considerations. Patients who present with acute symptoms present a different set of biologic issues than those with an asymptomatic tooth. Swelling associated with an abscess, cellulitis, or presence of a stoma represent signs of pathologic processes. The biologic significance of these conditions should be considered before determining specific goals for each visit.

Developing specific goals at each visit helps to organize the treatment. For example, for an uncomplicated molar or premolar, some clinicians will set a specific goal for the first visit that includes access and thorough instrumentation while deferring the obturation to a second visit. Uncomplicated single-rooted, vital teeth may be planned for a single-visit approach. It is important that ample time be allowed so that the procedure can be adequately completed without undue stress.

These recommendations have a biologic basis. Biologically, it is not reasonable to partially instrument root canal systems, thereby leaving residual inflamed pulpal remnants or necrotic debris in the canal, because such remnants may cause pain and be susceptible to infection. The clinician would be well advised to begin canal instrumentation only if time permits the extirpation of all pulp tissue and debridement of the root canal system.

Although in most cases the clinical procedures required to complete endodontic treatment can be accomplished in a single visit, that does not mean it is the better course of treatment. What can be done and what should be done represent two very different approaches to endodontic treatment planning. The patient's systemic health, level of anxiety, and symptoms, as well as the complexity of the root canal system, are factors that must be considered.

A study concerning the outcome of initial treatment noted the complexity of treating apical periodontitis.[31] The author commented:

Treatment of this disease cannot be improved merely by changing treatment techniques. Because apical periodontitis results from interactions between microorganisms, their environment and the host immune system, only use of effective modifiers of any of these three factors might significantly improve the outcome of treatment.

INTERDISCIPLINARY TREATMENT PLANNING

Periodontal Considerations

Extensive periodontal lesions may complicate endodontic prognosis. Lesions with endodontic and periodontal components may necessitate consultation with an endodontist or periodontist in order to gather more information about the tooth's prognosis.

A 4-year retrospective study found that attachment loss and periodontal status affected endodontic prognosis of endodontically treated molars.[87] It is crucial that the dental practitioner be aware of the periodontal factors that may influence the prognosis of endodontic treatment, such as root perforations, bone loss, and clinical attachment loss.

When establishing the prognosis of a tooth with an endodontic/periodontal lesion, there are essential factors to be considered. Determination of pulp vitality and the extent of the periodontal defect are central to establishing the prognosis and developing a treatment plan for a tooth with an endodontic/periodontal lesion (see also Chapter 25).

In primary endodontic disease, the pulp is nonvital (Figs. 3-9 and 3-10), whereas in primary periodontal disease, the pulp retains vitality. True combined endodontic-periodontal disease occurs less frequently. The combined lesion is found

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FIG. 3-9 A, Inflamed, edematous interproximal tissue *(arrow)* caused by acute endodontic pathosis. B, Soft tissue healing *(arrow)* 3 days after initiation of endodontic treatment. C, Periradicular pathosis. D, Completed endodontic therapy. E, Periradicular healing at 1-year recall.

when the endodontic disease process advances coronally and joins with a periodontal pocket progressing apically. There is significant attachment loss with this type of lesion, and the prognosis is guarded.[81] The radiographic appearance of combined endodontic-periodontal lesions may be similar to that of a vertically fractured tooth. Therapy for true combined lesions requires both endodontic and periodontal therapy. Sequencing of treatment is based on addressing the initial chief complaint.

The prognosis and treatment of each type of endodontic-periodontal disease vary. Primary endodontic disease should be treated solely by endodontic therapy, and the prognosis is usually good. Primary periodontal disease should be treated only by periodontal therapy, and the prognosis varies depending on the seventy of the disease and patient's response to treatment.[81]

Pathogenesis of the lesion can be better understood after sensibility testing, periodontal probing, radiographic assessment, and evaluation of dental history. When extensive prostheses are planned, the potential risk of including a tooth with a questionable prognosis must be considered. It is not prudent to incorporate a chronic problem into a new complex prosthesis (Fig. 3-11).

Surgical Considerations

Surgical evaluation is particularly valuable in the diagnosis of lesions that may be nonodontogenic. Biopsy is the definitive means of diagnosing osseous pathosis, which may mimic a lesion of endodontic origin. When retreatment is being considered, the clinician must determine whether nonsurgical, surgical, or combined treatment is appropriate. This decision is influenced by the presence of complex restorations, posts, and the radiographic assessment of prior endodontic therapy.

Endodontic surgery is most often performed in an attempt to improve the apical seal and correct failure of nonsurgical therapy. Bacteria are the essential cause of failure. It is important that the clinician determine the bacterial path of ingress. For example, a deficient restoration or recurrent decay will result in microleakage into the root canal space. Unless that issue is addressed, apical surgery may not be predictable (Fig. 3-12).

When a deficient restoration is identified, it must be replaced to prevent continued bacterial penetration. Endodontic surgery (see Chapter 9) may also be performed as a primary procedure when there are complications such as calcific metamorphosis. In those cases, by using surgery as primary therapy, an apical seal can be established while preserving the crown of the tooth.

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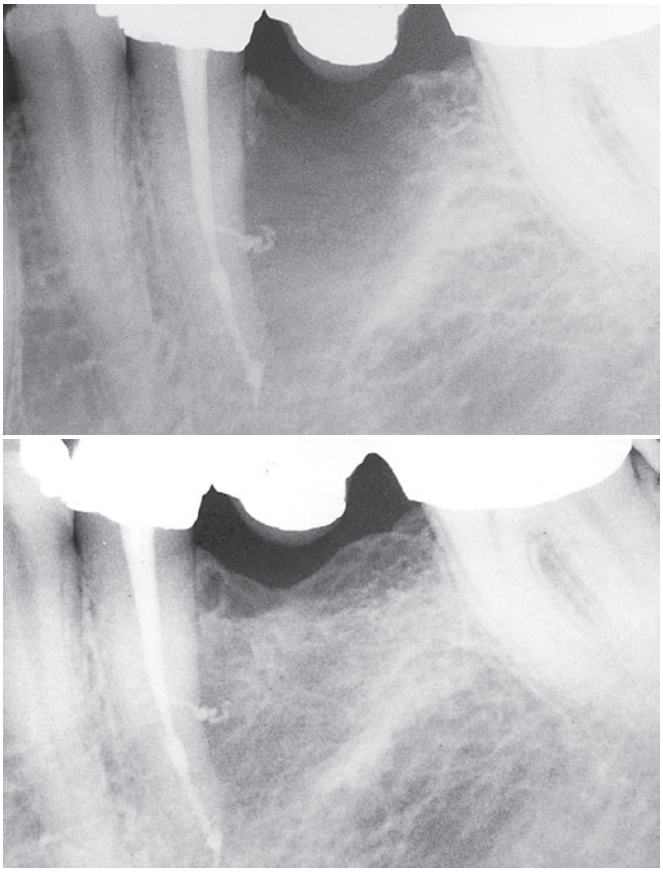

FIG. 3-10 A large, bony defect associated with tooth #20 healed after endodontic therapy. The tooth was nonvital, and no significant periodontal probing depth indicated pulpal disease.

The treatment plan for these cases is determined after reviewing multiple radiographs and considering the possibility of completing nonsurgical therapy without destroying an otherwise functional crown or natural tooth. Endodontic surgery without prior nonsurgical therapy should be a treatment of last resort and only when nonsurgical treatment is not possible.

Reviewing the best available evidence for alternative treatments is an important aspect of treatment planning for a tooth with failed endodontics. Evidence concerning healing potential after endodontic surgery is an important consideration in the management of posttreatment disease.[30] Numerous studies

FIG. 3-11 Tooth #30 has a poor prognosis. Periodontal probing reached the apex of the distal root. Extraction is indicated and should be done as soon as possible to prevent further damage to the mesial bone associated with tooth #31. Implant site preservation is another consideration in treatment planning for this case.

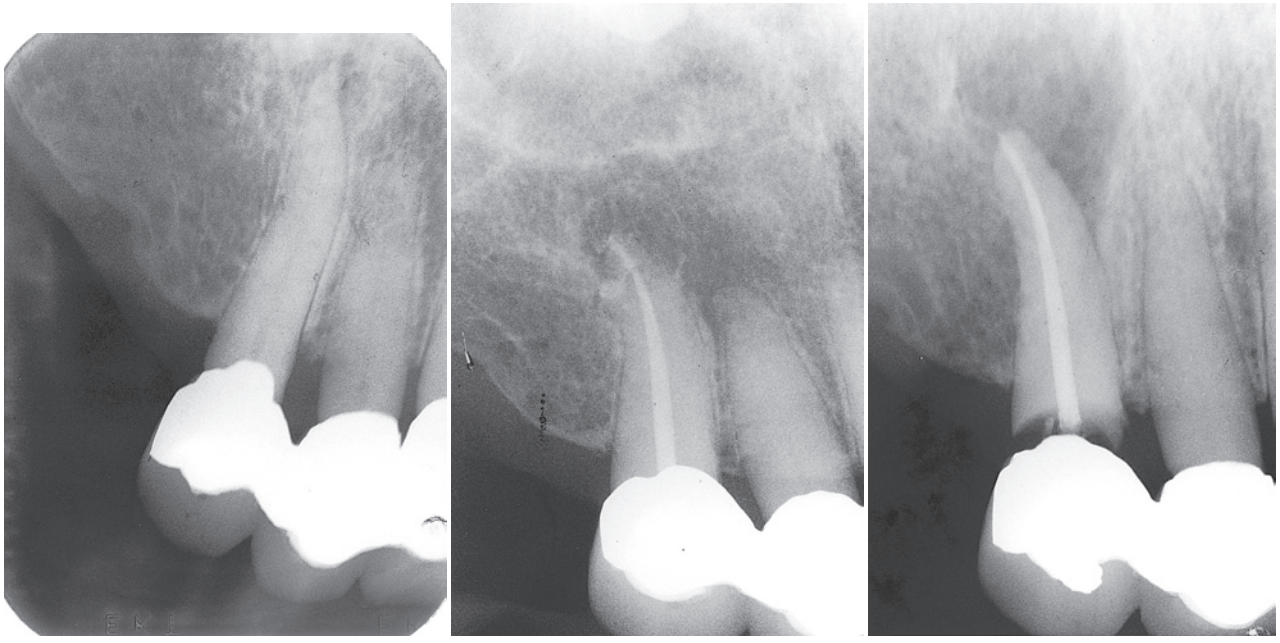

FIG. 3-12 Four years after endodontic therapy, the patient complained of pain and swelling associated with tooth #6. The initial impression was that apical surgery was indicated. However, further radiographs revealed the true cause of the endodontic failure. The initial endodontic access through the crown or caries damaged the coronal seal and recurrent decay followed.

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have examined the outcome of apical surgery and the results vary considerably.[101,104,112] This variability may reflect actual outcome differences or reflect variations in case selection techniques, recall periods, and methodology.

One prospective study indicated that there is an increased odds ratio for disease persistence for teeth with larger pretreatment lesions and pretreatment root canal filling of adequate length.[104] Another study found that patients presenting with pain at the initial examination before surgery had a significantly lower rate of healing at the 1-year follow-up compared with patients who did not have pain at the initial examination.[101]

It should also be noted that the periodontal condition of the tooth, including the interproximal bone levels and the amount of marginal bone loss, has been shown to significantly affect the long-term prognosis of periapical surgery.[102,105] Moreover, it has been shown that isolated endodontic lesions have much higher success rates at the 1- to 5-year follow-up (95.2%) in comparison with endodontic-periodontal combined lesions (77.5%).[40]

Dramatic changes in surgical technique and materials have occurred: The advent of microscopy, endoscopy, and ultrasonics, as well as improved retrograde filling materials, represents important modifications of surgical technique. An outcome study comparing traditional root-end surgery (TRS) and endodontic microsurgery (EMS) found that the probability of success for EMS was 1.58 times greater than the probability of success for TRS.[89] Another study comparing the EMS techniques with and without the use of higher magnification found that the difference of probability of success between the groups was statistically significant for molars, but no significant difference was found for the premolar or anterior teeth.[88] Cone-beam computed tomography has proved to be of value in some surgical cases. It produces three-dimensional images of a tooth, pathosis, and adjacent anatomic structures. It is beneficial for localizing the mandibular canal,[42] mental foramen, maxillary sinus, and nasal cavity.[16]

Restorative and Prosthodontic Considerations

A satisfactory restoration may be jeopardized by a number of factors. Subosseous root caries (perhaps requiring crown lengthening), poor crown-to-root ratio, and extensive periodontal defects or misalignment of teeth may have serious effects on the final restoration. These problems must be recognized before endodontic treatment is initiated. For complex cases, a restorative treatment plan should be in place before initiating endodontic treatment (see Chapter 22). Some teeth may be endodontically treatable but nonrestorable, or they may represent a potential restorative complication because of a large prosthesis. Reduced coronal tooth structure under a full-coverage restoration makes endodontic access more difficult because of reduced visibility and lack of radiographic information about the anatomy of the chamber. It is not unusual for restorations to be compromised during endodontic access (see Fig. 3-12). Whenever possible, restorations should be removed before endodontic treatment.

Full coverage restorations are usually suggested after endodontic treatment. In a systematic review on tooth survival following nonsurgical root canal treatment, four factors were found to be of significance in tooth survival[66]:

- A crown restoration after root canal treatment
- Tooth having both mesial and distal proximal contacts
- Tooth not functioning as an abutment for removable or fixed prosthesis
- Tooth type or specifically nonmolar teeth

Another systematic review found that the odds for healing of apical periodontitis increase with both adequate root canal treatment and adequate restorative treatment. However, poorer clinical outcomes may be expected with adequate root filling/inadequate coronal restoration and inadequate root filling/adequate coronal restoration, with no significant difference in the odds of healing between these two combinations.[34] These findings suggest that the quality of the coronal restoration is as important as the quality of the root canal treatment. Therefore, to increase the success of the treatment, it is strongly suggested that the clinician discuss the restorative plan of the tooth with both the patient and—if it is a referred patient—with the referring dentists before initiation of treatment.

Endodontic Therapy or Dental Implant

The successful evolution of dental implants as a predictable replacement for missing teeth has had a positive impact on patient care. A clinician now has an additional possibility to consider when developing a treatment plan for a patient with a missing tooth or teeth. More challenging is the decision concerning whether or not to provide endodontic therapy for a tooth with a questionable prognosis or extract and use a single-tooth implant as a replacement. Numerous studies have evaluated both nonsurgical endodontic therapy[18,69,70,82,97,98] and endosseous dental implants.[2,17,37,47]

It is not possible to compare outcome studies because of variations in research methodologies, follow-up periods, and criteria associated with determining success or failure. A review of outcome studies points to the need for randomized controlled trials with standardized or similar methodologies that could provide a higher level of evidence to use in answering important clinical prognostic questions.

A synthesis of available evidence indicates that both primary root canal treatment and single-tooth implants are highly predictable procedures when treatment is appropriately planned and implemented. A study assessed clinical and radiographic success of initial endodontic therapy of 510 teeth over a 4- to 6-year period. It was found that 86% of teeth healed and 95% remained asymptomatic and functional.[18] Another study considered the outcomes of endodontic treatment on 1,462,936 teeth. More than 97% of teeth were retained after 8 years.[82] In a similar study it was found that overall, 89% of the 4744 teeth were retained in the oral cavity 5 years after the endodontic retreatment.[83] A study concerning the outcome of endodontic therapy on 1312 patients in general practice with a mean follow-up time of 3.9 years found a combined failure rate of 19.1%, concluding that "failure rates for endodontic therapy are higher than previously reported in general practices, according to results of studies based on dental insurance claims data."[7]

The American Dental Association's Council on Scientific Affairs has reported high survival rates for endosseous implants. An evaluation of 10 studies with more than 1400 implants demonstrated survival rates ranging from 94.4% to 99% with a mean survival rate of 96.7%.[2] With such high survival rates reported for endodontics and single-tooth implants, a clinician must consider a multiplicity of factors within the context of the

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best available evidence. Most current studies indicate no significant difference in the long-term prognosis between restored endodontically treated teeth and single-tooth implants.[37]

In a retrospective cross-sectional comparison of initial nonsurgical endodontic treatment and single-tooth implants, it was suggested that restored endodontically treated teeth and single-tooth implant restorations have similar survival rates, although the implant group showed a longer average and median time to obtain function and a higher incidence of posttreatment complications requiring subsequent treatment intervention.[20] A review summarized the best available evidence concerning factors influencing treatment planning involving preservation of a tooth with endodontic therapy or replacement by a single-tooth implant. Factors considered included prosthetic restorability of the natural tooth, quality of bone, aesthetic concerns, cost-to-benefit ratio, systemic factors, potential for adverse effects, and patient preferences.[37] The authors concluded that "endodontic treatment of teeth represents a feasible, practical and economical way to preserve function in a vast array of cases and that dental implants serve as a good alternative in selected indications in which prognosis is poor."[37]

Aside from treatment outcome, there are other factors involved in any treatment that the practitioner has to consider when making a treatment planning decision. In a study evaluating the quality of life of endodontically treated versus implant treated patients, the results showed a high rate of satisfaction with both treatment modalities.[32] One study found that comparing to endodontic molar retreatment and fixed partial dentures, implant-supported restoration, despite its high survival rate, has been shown to be the least cost-effective treatment option.[41] Another study found that "endodontically treated natural teeth may provide more effective occlusal contact during masticatory function compared with implant-supported restorations, leading to more efficient mastication."[109]

Another important factor is the patient's health status. Implants require a surgical procedure that may not be possible due to the patient's medical status. One area of concern is diabetes mellitus. However, it has been shown that dental implant osseointegration can be accomplished in these subjects as long as they have good glycemic control.[38]

It seems clear that patients are best served by retaining their natural dentition as long as the prognosis for long-term retention is positive. It is not reasonable to extract a tooth if endodontics with a good prognosis can be completed. It is also not reasonable for a patient to invest in root canal therapy, a post, and a crown if the prognosis is highly questionable and an implant with a good prognosis can be placed. An important advantage of providing endodontic therapy is to allow rapid return of the patient's compromised dentition to full function and aesthetics. This rapid return is in marked contrast to the use of provisional restorations associated with dental implants while waiting for osseous integration. The challenge is to weigh all pretreatment variables and reach a reasonable conclusion concerning the prognosis for tooth retention or implant placement.

Interestingly, some endodontic advanced education programs are now including implant training in their curricula. This training will enable the endodontist to provide more value to the patient and referring the dental clinician as treatment plans are determined. Such dually trained endodontists will be well positioned to provide endodontic therapy or place an implant as best serves the patient.

OTHER FACTORS THAT MAY INFLUENCE ENDODONTIC CASE SELECTION

A variety of factors may complicate proposed endodontic therapy. Calcifications, dilacerations, or resorptive defects may compromise endodontic treatment of a tooth with potentially strategic value (Fig. 3-13). The inability to isolate a tooth is also a problem and may result in bacterial contamination of the root canal system. Extra roots and canals pose a particular

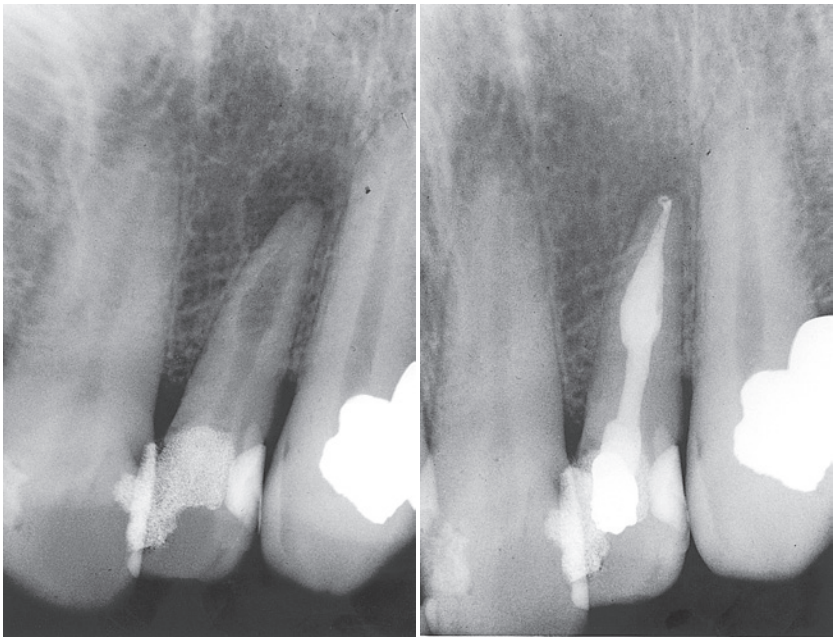

FIG. 3-13 Resorptive defects can be successfully treated. Early intervention, before there is perforation of the root, increases the chance of success. (Courtesy Dr. Leon Schertzer.)

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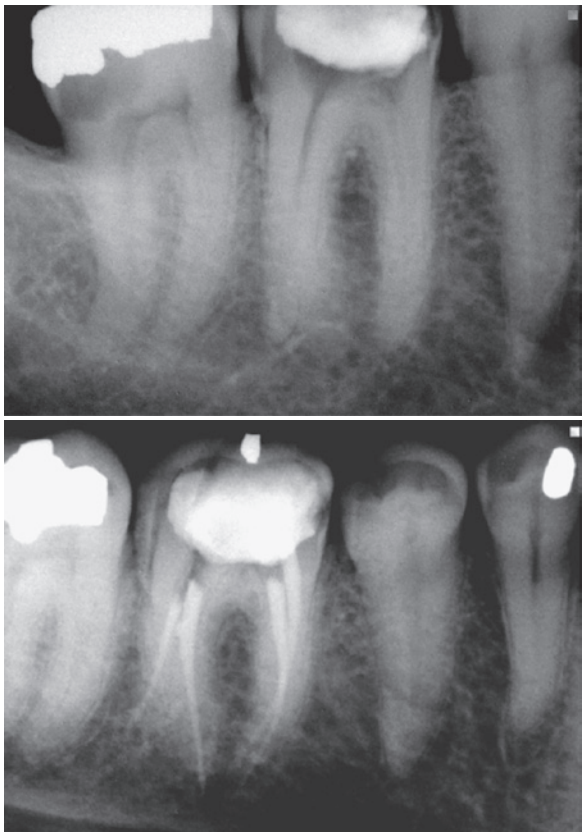

FIG. 3-14 The presence of curved roots and multiple canals is a complicating factor.

anatomic challenge that radiographs do not always reveal (Fig. 3-14). A bitewing radiograph is useful in providing an accurate image of the pulp chambers of posterior teeth. The clinician should recognize these potential problems and be able to manage and factor them into the decision concerning the tooth's prognosis, including the possibility that the patient should be referred to a specialist.

Another consideration is the stage of maturity of the tooth. Primary and immature permanent teeth may have a pulpal pathosis caused by caries or trauma; preserving these young teeth is essential. Premature loss of an anterior tooth can lead to malocclusion, predispose the patient to tongue habits, impair aesthetics, and damage the patient's self-esteem. (See Chapters 20 and 24 for further information.)

Some clinicians use a simple formula for determining which endodontic cases they treat and which they refer to a specialist. The number of roots may be the determining factor in a decision concerning referral, or the key factor may be the chronic or acute status of the case. Others consider the complexity of the ultimate prosthesis as a factor in considering an endodontic referral. The most important variables in determining whether to refer a patient to a specialist are the skills of the clinician and the complexity of the case.

The American Association of Endodontists (AAE) developed guidelines for assessing endodontic case difficulty (available at www.aae.org/uploadedFiles/Publications_and_Research/Guidelines_and_Position_Statements/2006CaseDifficultyAssessmentFormB_Edited2010.pdf) (see Fig. 2-1). The AAE Endodontic Case Difficulty Assessment Form enables a clinician to assign a level of difficulty to a particular case. The form describes cases with minimal, moderate, and high degrees of difficulty. This form lists criteria that can be used to identify cases that should be referred to a specialist. The use of surgical operating microscopes, endoscopes, and ultrasonics enables the specialist to predictably treat teeth that would not previously have been treatable.

Anxiety

Anxiety presents a problem at many levels of dental care (see also Chapter 28). Avoidance of dental treatment due to anxiety appears to be associated with significant deterioration of oral and dental health.[111] Even at the diagnostic stage, severe anxiety may confuse the process.[23] Several studies support the hypothesis that pain or fear of pain is a primary source of anxiety as well as an obstacle to seeking dental care.[44,111] Also, highly anxious patients appear to be more sensitive to pain.[24,43] High levels of anxiety have been found to negatively affect clinical procedures including local anesthesia.[62] In 2009, Binkley tested the hypothesis that having natural red hair color, which is caused by variants of the melanocortin-1 receptor (*MC1R*) gene, could predict a patient's experiencing dental care–related anxiety and dental care avoidance.[8] She found that participants with *MC1R* gene variants reported significantly more dental care–related anxiety and fear of dental pain than did participants with no *MC1R* gene variants. A more recent study confirmed the relationship between the red hair phenotype and anxiety. However, this study found no association between the red hair phenotype and the success of local anesthesia.[21]

It has been demonstrated that dental anxiety and expectation of pain had a profound effect on a patient's ability to understand information provided.[25] A person's cognitive ability to process information is significantly affected by stress.[25] A study found that 40% of patients who had minor oral surgery did not remember receiving both written and verbal instructions, contributing to 67% noncompliance with antibiotic prescriptions.[9] Patients' anxiety can compromise their understanding of complex treatment plans. Decisions made by a patient concerning options involving tooth retention or loss may be markedly affected by anxiety.

Unfortunately, the impact that a high level of anxiety can have on patient's cognition, local anesthesia, and intraoperative and postoperative experiences is not always recognized. A landmark medical study found that pretreatment discussion of surgical treatments and associated discomfort reduced by 50% the need for posttreatment morphine and reduced the time to discharge.[22] Existing research has focused primarily on the effect of pretreatment information on reducing anxiety and stress during surgery.[25]

More than 200 studies indicate that preemptive behavioral intervention decreases anxiety before and after surgery, reduces posttreatment pain intensity and intake of analgesics, and accelerates recovery.[15] A calm setting, reassurance by the clinician and explanation of the treatment plan, as well as a discussion about pain prevention strategies are all important steps even before treatment starts.[63] A written description as well as a verbal description of the proposed treatment are helpful. It may also be of value to have a family member or friend accompany the patient for a discussion of the treatment plan.

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Scheduling Considerations

If a vital case is to be treated by a multivisit approach, it is suggested that the clinician allow 5 to 7 days between canal instrumentation and obturation to allow periradicular tissues to recover. When a vital case is to be treated in a single visit, adequate time must be scheduled so that the clinician can comfortably complete the procedure. Because profound inferior alveolar nerve block anesthesia can require approximately 15 to 20 minutes, it is wise to include that time when scheduling a patient's appointment (see also Chapter 4).

Appointments to fill nonvital cases should be scheduled approximately 1 week after instrumentation to maximize the antimicrobial effect of the intracanal dressing when calcium hydroxide is used.[6,92,93] Acute (pain or swelling) nonvital cases should be seen every 24 to 48 hours to monitor the patient's progress and bring the acute symptoms under control. Further cleaning and shaping are important components of the treatment as the clinician seeks to eliminate persistent microbes in the canal system. Long delays between visits contribute to the development of resistant microbial strains and should be avoided.

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CHAPTER 4

Pain Control

AL READER | JOHN NUSSTEIN | ASMA KHAN

CHAPTER OUTLINE

I. LOCAL ANESTHESIA FOR RESTORATIVE DENTISTRY AND ENDODONTICS

Mechanisms of Action for Anesthetics

Clinically Available Local Anesthetics

Selection of a Local Anesthetic: Possible Adverse Effects, Medical History, and Preoperative Anxiety

Possible Adverse Effects

Effects of Systemic Diseases or Conditions on Local Anesthetics

Clinical Anesthesia and Routes of Anesthetic Administration

Important Clinical Factors in Local Anesthesia

Traditional Methods of Confirming Anesthesia

Determining Pulpal Anesthesia in Asymptomatic Vital Teeth

Determining Pulpal Anesthesia in Symptomatic Vital Teeth

Patient Who Has Had Previous Difficulty with Anesthesia

Failure to Achieve Anesthesia in Patients with Pain

Use of Topical Anesthetics

Reversing the Action of Local Anesthetics

Inferior Alveolar Nerve Block for Restorative Dentistry

2% Lidocaine and 1:100,000 Epinephrine

Anesthetic Success

Anesthetic Failure

Noncontinuous Anesthesia

Slow Onset

Duration

Alternative Anesthetic Solutions for the Inferior Alveolar Nerve Block

Plain Solutions: 3% Mepivacaine (Carbocaine, Polocaine, Scandonest) and 4% Prilocaine (Citanest Plain)

4% Prilocaine with 1:200,000 Epinephrine (Citanest Forte) and 2% Mepivacaine with 1:20,000 Levonordefrin (Carbocaine with Neo-Cobefrin)

Articaine with 1:100,000 Epinephrine (Septocaine, Articadent, Zorcaine)

Long-Acting Anesthetics

Buffered Lidocaine

Use of Mannitol

Alternative Injection Sites

Gow-Gates and Vazirani-Akinosi Techniques

Incisive Nerve Block/Infiltration at the Mental Foramen

Lidocaine Infiltrations

Articaine Infiltrations

Attempts to Increase Success of the Inferior Alveolar Nerve Block

Increasing the Volume of Anesthetic

Increasing the Epinephrine Concentration

Addition of Hyaluronidase

Carbonated Anesthetic Solutions

Diphenhydramine as a Local Anesthetic Agent

Addition of Meperidine to Lidocaine

Factors in Failure of the Inferior Alveolar Nerve Block

Accessory Innervation: Mylohyoid Nerve

Accuracy of Injection

Needle Deflection

Needle Bevel and Success

Speed of Injection and Success

Cross-Innervation

Red Hair

A Theory on Why Failure Occurs with the Inferior Alveolar Nerve Block in Restorative Dentistry

Enhancement of Mandibular Anesthesia for Restorative Dentistry

Supplemental Articaine Infiltrations

Supplemental Intraosseous Anesthesia

Supplemental Intraligamentary Anesthesia

Maxillary Anesthesia for Restorative Dentistry

2% Lidocaine with 1:100,000 Epinephrine

Anesthetic Success

Onset of Pulpal Anesthesia

Duration of Pulpal Anesthesia

Time Course of Pulpal Anesthesia for the Maxillary First Molar

Significance of Lip Numbness

Alternative Anesthetic Solutions for Infiltrations

Plain Solutions; 3% Mepivacaine (Carbocaine, Polocaine, Scandonest) and 4% Prilocaine (Citanest Plain)

4% Prilocaine with 1:200,000 Epinephrine (Citanest Forte), 2% Mepivacaine with 1:20,000 Levonordefrin (Carbocaine with Neo-Cobefrin), and 4% Articaine with 1:100,000 Epinephrine (Septocaine, Articadent, Zorcaine)

0.5% Bupivacaine with Epinephrine (Marcaine)

Extending the Duration of Pulpal Anesthesia for Maxillary Teeth

Increasing the Solution Volume

Increasing the Epinephrine Concentration

Repeating the Infiltration

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Alternative Maxillary Injection Techniques for Restorative Dentistry
Posterior Superior Alveolar (PSA) Nerve Block
Infraorbital Nerve Block
Second Division Nerve Block
Palatal–Anterior Superior Alveolar (P-ASA) Nerve Block
Anterior Middle Superior Alveolar (AMSA) Nerve Block
Supplemental Anesthesia for Restorative Dentistry in the Mandible and Maxilla
Indications
Infiltrations
Intraosseous Anesthesia
Intraligamentary Anesthesia
Local Anesthesia for Endodontics
Success of the Inferior Alveolar Nerve Block in Patients Presenting with Symptomatic Irreversible Pulpitis
Success of Maxillary Molar Infiltration in Patients Presenting with Irreversible Pulpitis
Asymptomatic Irreversible Pulpitis versus Symptomatic Irreversible Pulpitis
Supplemental Techniques
Management of Anesthesia in Endodontic Cases
Symptomatic Irreversible Pulpitis
Anesthetizing Mandibular Posterior Teeth
Anesthetizing Mandibular Anterior Teeth
Anesthetizing Maxillary Posterior Teeth
Anesthetizing Maxillary Anterior Teeth
Anesthetizing Symptomatic Teeth with Total Pulpal Necrosis and Periapical Abscess
Anesthetizing Asymptomatic Teeth with Total Pulp Necrosis and Periapical Radiolucencies
Interim Treatment for Irreversible Pulpitis Using Pulpotomy
Pain Reduction in Irreversible Pulpitis When Endodontic Treatment Is Impossible
Oral Conscious Sedation with Triazolam (Halcion) and Alprazolam (Xanax)
Conscious Sedation with Nitrous Oxide
Preemptive Nonsteroidal Antiinflammatory Drugs (NSAIDs)
Patient Satisfaction with Painful Dental Procedures
Anesthesia for Surgical Procedures
Incision for Drainage
Periapical Surgery
Summary and Future Directions for Effective Anesthesia
II. ANALGESICS AND THERAPEUTIC RECOMMENDATIONS
Non-Narcotic Analgesics
Limitations and Drug Interactions
Acetaminophen
Opioid Analgesics
Corticosteroids
Intracanal Administration
Systemic Administration
Antibiotics
Pain Management Strategies
Pretreatment
Long-Acting Local Anesthetics
Flexible Plan
Future Directions
Summary

I. LOCAL ANESTHESIA FOR RESTORATIVE DENTISTRY AND ENDODONTICS

Effective local anesthesia is the bedrock of pain control in endodontics and restorative dentistry. Regardless of the clinician's skills, both treatment and patient management are difficult or impossible to deliver without effective pain control. This chapter reviews the pharmacology of local anesthetics and the relative advantages and limitations of various anesthetics and routes of administration. Other chapters in this book provide complementary information on the use of local anesthetics in diagnosis (see Chapter 1) and the treatment of emergency patients (see Chapter 18). The authors assume that the reader is familiar with various anesthetic injection techniques; several excellent texts are available for review regarding this point.[195,262,353]

MECHANISMS OF ACTION FOR ANESTHETICS

Most dental pharmacology courses teach that local anesthetics block sodium channels by partitioning into two types, the uncharged basic form of the molecule (RN), which crosses cell membranes, and the charged acid form of the molecule (RNH^+), which binds to the inner pore of the sodium channel. As a first approximation, this model is reasonably accurate. However, molecular research has demonstrated the existence of at least nine subtypes of voltage-gated sodium channels (VGSCs) that differ in their expression pattern, biophysical properties, and roles in mediating peripheral pain (Table 4-1). These channels have a clear clinical relevance.[39,170,241] Indeed, several groups of patients have been described with genetic mutations to a VGSC, with significant reported effects on pain sensitivity.

The broad class of VGSCs can be divided into channels that are blocked by a toxin (tetrodotoxin [TTX]) and those that are resistant to the toxin (TTX-R). Most TTX-R channels are found primarily on nociceptors (e.g., Na_v 1.8 and Na_v 1.9).[433] These channels also are relatively resistant to local anesthetics and are sensitized by prostaglandins.[148] As is explained later in the chapter, the presence of TTX-R sodium channels may explain why local anesthetics are less effective when administered to patients with odontalgia. Many of the adverse effects of local anesthetics are attributed to their ability to block other VGSCs expressed in the central nervous system (CNS) or heart (see Table 4-1).

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VGSCs consist of an alpha and a beta subunit. The alpha subunit serves as a voltage sensor, leading to channel activation and sodium ion passage when the channel detects an electrical field. The biologic basis for an electrical pulp tester, therefore, is the generation of a small electrical field across the dental pulp that can activate VGSCs.[170] Interestingly, sensitization of TTX-R channels by prostaglandins lowers the activation threshold and increases the number of sodium ions that flow through the channel.[148] Put another way, an inflammation-induced elevation in prostaglandin levels sensitizes TTX-R channels, leading to greater activation with weaker stimuli. This may explain the increased responsiveness to electrical pulp testing seen in patients with irreversible pulpitis.

TABLE 4-1
Voltage-Gated Sodium Channels and Pain

Channel Subtype	Tissue Expression	Tetrodotoxin Sensitive	Peripheral Role in Pain
Na_v 1.1	Central nervous system (CNS), sensory neurons	Yes	?
Na_v 1.2	CNS	Yes	No
Na_v 1.3	CNS	Yes	No
Na_v 1.4	Muscle	Yes	No
Na_v 1.5	Heart	Somewhat	No
Na_v 1.6	CNS, sensory neurons	Yes	?
Na_v 1.7	CNS, sensory neurons	Yes	?
Na_v 1.8	Sensory neurons	No	Yes
Na_v 1.9	Sensory neurons	No	Yes

Local anesthetics have other mechanisms that may contribute to their pharmacology for treating odontogenic pain. For example, local anesthetics modulate certain G protein–coupled receptors (GPCRs). The GPCRs are a major class of cell membrane receptors, and many classes of dental drugs (e.g., opioids, catecholamines) and endogenous mediators produce their effects by activating specific GPCRs and their related second messenger pathways. Studies suggest that local anesthetics inhibit the G-alpha-q ($G_{\alpha q}$) class of GPCRs, which includes receptors activated by inflammatory mediators such as bradykinin.[183] Local anesthetics may therefore block the actions of a major hyperalgesic agent.

Other studies have indicated that local anesthetics potentiate the actions of the G-alpha-i ($G_{\alpha i}$) class of GPCRs.[32] This could have a major effect in potentiating the actions of vasoconstrictors, including the newly recognized analgesic role that vasoconstrictors play in inhibiting pulpal nociceptors.[43,169] Prolonged alteration of GPCR function might explain why analgesia obtained with long-acting local anesthetics persists well beyond the period of anesthesia.[78,104,300] More research is needed on this exciting aspect of local anesthetic pharmacology.

CLINICALLY AVAILABLE LOCAL ANESTHETICS

The most common forms of injectable local anesthetics are in the amide class. In 2003, the American Dental Association specified a uniform color code for dental cartridges to prevent confusion among brands (Table 4-2). Local anesthetics can be divided roughly into three types: short duration (30 minutes of pulpal anesthesia), intermediate duration (60 minutes of pulpal anesthesia), and long duration (over 90 minutes of pulpal anesthesia). However, clinical anesthesia does not always follow these guidelines, depending on whether the local anesthetic is used as a block or for infiltration. For example, bupivacaine is classified as a long-acting agent, and when it is used in an inferior alveolar nerve (IAN) block, this is true.[112] However, when it is used for infiltration for anterior teeth, it

TABLE 4-2
Local Anesthetics Available in the United States*

Anesthetic	Vasoconstrictor	Dental Cartridge Color Codes†	Maximum Allowable Dose	Typical Maximum Dose
2% Lidocaine	1:100,000 epinephrine	Red	13	8
2% Lidocaine	1:50,000 epinephrine	Green	13	8
2% Lidocaine	Plain (no vasoconstrictor)	Light blue	8	8
2% Mepivacaine	1:20,000 levonordefrin	Brown	11	8
3% Mepivacaine	Plain (no vasoconstrictor)	Tan	7	5½
4% Prilocaine	1:200,000 epinephrine	Yellow	5½	5½
4% Prilocaine	Plain (no vasoconstrictor)	Black	5½	5½
0.5% Bupivacaine	1:200,00 epinephrine	Blue	10	10
4% Articaine	1:100,000 epinephrine	Gold	7	7
4% Articaine	1:200,000 epinephrine	Silver	7	7

*This table provides the maximum dosage in two formats. The maximum allowable dose generally is approached only with complex oral and maxillofacial surgical procedures. The typical maximum dose is the usual outer envelope of drug dosage for most endodontic, surgical, and restorative dental procedures. Both columns show the number of cartridges that would be required for an adult weighing 67.5 kg (150 pounds).
†Uniform dental cartridge color codes were mandated by the American Dental Association in June, 2003.

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has a shorter duration of anesthetic action than 2% lidocaine with 1:100,000 epinephrine[81,156] (this is discussed in more detail later in the chapter).

SELECTION OF A LOCAL ANESTHETIC: POSSIBLE ADVERSE EFFECTS, MEDICAL HISTORY, AND PREOPERATIVE ANXIETY

Possible Adverse Effects

Possible adverse reactions to local anesthetics can be divided into six major categories: cardiovascular reactions, systemic effects, methemoglobinemia, peripheral nerve paresthesia, allergic reactions to the anesthetic and/or latex, and reactions to anesthetics containing a sulfite antioxidant. These reactions range from fairly common (e.g., tachycardia after intraosseous injection of 2% lidocaine with 1:100,000 epinephrine) to extremely rare (e.g., allergic reactions to lidocaine).

Cardiovascular Reactions

Although classic research studies have reported that large dosages or intravenous (IV) injections of local anesthetics were required to produce cardiovascular effects,[192,419] it now is well recognized that even comparatively small amounts of epinephrine can induce measurable tachycardia after nerve block or intraosseous injection.[113,150,358] Several authors have reported increases in heart rate with infiltration injections and nerve blocks using 2% lidocaine with 1:100,000 epinephrine[2,172,236,375,418]; others have reported that no significant changes in heart rate occurred or that the changes were clinically insignificant.[282,413,422] When specific information was given on dosing and heart rate increases, several studies found mean heart rate increases.[2,172,235,418] Two studies found increases on average of about 4 beats/min with approximately 20 μg of epinephrine[172,235]; three studies recorded increases of 10 to 15 beats/min with 45 to 80 μg of epinephrine[2,235,375]; and one study found increases of approximately 21 beats/min using 144 μg.[418] Increasing the amount of epinephrine in an infiltration or block injection, therefore, increases the likelihood of an elevated heart rate.

Tachycardia after injection is primarily a pharmacologic effect. The cardiovascular effects are the result of alpha-adrenoceptor stimulation by systemic distribution of the vasoconstrictor throughout the vascular compartment. The patient may also report heart palpitations associated with anxiety or fear and may experience transient tachycardia and changes in blood pressure. Large doses or inadvertent IV injection may lead to lidocaine toxicity and CNS depression.[115,308] To reduce this risk, the clinician should always aspirate before making the injection, inject slowly, and use dosages within accepted guidelines. The maximal dosages for local anesthetics are listed in Table 4-2.

Systemic Effects

Acute toxicity from an overdose of a local anesthetic often is the result of inadvertent IV administration or of a cumulative large dose (e.g., repeated injections). As shown in Table 4-1, VGSCs are found in the CNS and the myocardium, the two major sites of anesthetic-induced toxicity. Although systemic effects from a local anesthetic are rare, they can include an initial excitatory phase (e.g., muscle twitching, tremors, grand mal convulsions) and a subsequent depressive phase (e.g., sedation, hypotension, and respiratory arrest).[86,115] It should be noted that symptomatic management (possibly including cardiopulmonary resuscitation [CPR], airway support, and supplemental oxygen) is the primary response to this adverse event.[229,233] An acute hypotensive crisis with respiratory failure also has been interpreted as the result of hypersensitivity to local anesthetics[62]; these patients should be evaluated with allergy testing. To reduce the risk of systemic effects from anesthetics, the clinician must always aspirate before giving the injection and must use dosages within accepted guidelines (see Table 4-2). Finder and Moore[115] proposed a "rule of 25" as a simple means of remembering maximal local anesthetic dosages: with currently formulated local anesthetic cartridges, it generally is safe to use one cartridge of local anesthetic for every 25 pounds of patient weight (e.g., six cartridges for a patient weighing 150 pounds [67.5 kg]).

Methemoglobinemia

Metabolism of certain local anesthetics (e.g., prilocaine, benzocaine, articaine, and to a lesser extent lidocaine) can produce a metabolite that causes methemoglobinemia; this effect often occurs several hours after injection of the local anesthetic.[265,439] Typical signs and symptoms include cyanosis, dyspnea, emesis, and headache. In a study on benzocaine-induced methemoglobinemia, 67% of reported adverse effects of benzocaine were associated with methemoglobinemia; of these events, 93% occurred with spray formulations of benzocaine, and only one case involved the gel formulation.[301] To reduce the risk of methemoglobinemia, clinicians should take care to refrain from giving excessive dosages of local anesthetics.

Peripheral Nerve Paresthesia

Postinjection paresthesia is a rare adverse effect of local anesthetics.[161,265,453] The incidence of paresthesia (which involved the lip and/or tongue) associated with articaine and prilocaine was higher than that found with either lidocaine or mepivacaine.[132,140,161] Another study evaluated patients referred with a diagnosis of damage to the inferior alveolar and/or lingual nerve that could only have resulted from an IAN block.[345] In 35% of these cases, the paresthesia was caused by a lidocaine formulation, and in 30%, it was caused by an articaine formulation. The conclusion was that there was not a disproportionate nerve involvement from articaine, although this interpretation does not account for large differences in clinical usage of the two local anesthetics. However, with any paresthesia, documentation of the patient's reported area of altered sensation, the type of altered sensation (e.g., anesthesia, paresthesia, dysesthesia), and regular follow-up are important.[463]

Allergic Reactions to Local Anesthetics and Latex

The amide local anesthetics appear to have little immunogenicity and therefore have an extremely low rate of allergic reactions.[382] One study included more than 140 patients specifically referred for allergy testing because of adverse effects after injection of a local anesthetic; none of these patients had hypersensitivity reactions to intradermal local anesthetics,[368] but case reports of hypersensitivity reactions after administration of local anesthetics have been published.[41,62,302,382] Some concern has been raised that the rubber latex stopper in dental anesthetic cartridges might be a source of allergen to patients allergic to latex. In a review of this literature (1966 to 2001), Shojaei and Haas[385] concluded that some evidence for exposure

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to the latex allergen exists, although no causal study has been published.

Local anesthetic formulations that contain vasoconstrictors also contain sulfite to prevent oxidation of this agent. Sulfite-induced reactions came to prominence with the report of six deaths after exposure to salad bars or homemade wine.[20] Common reported signs and symptoms include allergic-like reactions, such as urticaria, bronchospasm, and anaphylaxis. Risk factors include an active history of asthma (perhaps 5% of asthmatics are at risk) and atopic allergy. The use of local anesthetics without vasoconstrictors is a possible alternative with these patients. No sulfite reaction in dental practice has ever been documented, possibly because the amount of sulfite in local anesthetic cartridges is relatively small.

EFFECTS OF SYSTEMIC DISEASES OR CONDITIONS ON LOCAL ANESTHETICS

It has been stated that vasoconstrictors should be avoided in patients with high blood pressure (higher than 200 mmHg systolic or 115 mmHg diastolic), cardiac dysrhythmias, unstable angina, less than 6 months since myocardial infarction or cerebrovascular accident, or severe cardiovascular disease.[262] However, these conditions are contraindications to routine dental treatment. Patients taking antidepressants, nonselective beta-blocking agents, medicine for Parkinson disease, and cocaine have a potential for problems.[262,353] In patients taking these medications, plain mepivacaine (3% Carbocaine) can be used for the inferior alveolar nerve block.

Alcoholics have been found to be more sensitive to painful stimulation.[406] Alcoholics with a history of depression/unhappiness may also have reduced pulpal anesthesia.[116] In contrast, alcoholics in recovery may not be at increased risk for inadequate pain control with local anesthesia.[116]

Any of the commonly available local anesthetics are safe for use in pregnant or lactating women.[162] The most important aspect of care with pregnant patients is to eliminate the source of pain by performing the indicated endodontic treatment; this reduces the need for systemic medications.[162]

Local anesthetics may interact with a patient's medications, so a thorough review of the medical history is an absolute requirement. Potential drug-drug interactions occur primarily with the vasoconstrictors in local anesthetic formulations (Table 4-3). Judicious use of local anesthetic solutions without vasoconstrictors (e.g., 3% mepivacaine) is a reasonable alternative for adult patients.

Studies have found that women try to avoid pain more than men, accept it less, and fear it more.[99,114,249,303] One study found that women find postsurgical pain more intense than males, but men are more disturbed than women by low levels of pain that lasts several days.[303] Another study found gender differences in analgesia for postoperative endodontic pain.[374] Anxiety

TABLE 4-3
Possible Drug Interactions with Vasoconstrictors

Drugs	Possible Adverse Effects	Recommendations
Tricyclic Antidepressants		
Amitriptyline, doxepin	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
Nonselective Beta-Blockers		
Nadolol, propranolol	Hypertension, bradycardia	Reduce or eliminate vasoconstrictors
Recreational Drugs		
Cocaine	Hypertension, myocardial infarction, dysrhythmias	Instruct patient to abstain from drug use for 48 hours before procedure; do not use vasoconstrictors
COMT Inhibitors		
Entacapone, tolcapone	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
Antiadrenergic Drugs		
Guanadrel, guanethidine	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
Nonselective Alpha-Adrenergic Blockers		
Chlorpromazine, clozapine, haloperidol	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
Digitalis		
Digoxin	Dysrhythmias (especially with large dosage of vasoconstrictor)	Reduce or eliminate vasoconstrictor
Hormone		
Levothyroxine	Dysrhythmias (especially with large dosage of vasoconstrictor)	*Euthyroid:* No precaution *Hyperthyroid:* Reduce or eliminate vasoconstrictors
Monoamine Oxidase Inhibitors		
Furazolidone, linezolid, selegiline, tranylcypromine	No interaction	None

Modified from Naftalin L, Yagiela JA: Vasoconstrictors: indications and precautions, *Dent Clin North Am* 46:733, 2002.
COMT, Catecholamine *O*-methyl transferase.

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may also modulate differences in pain responses between males and females. In addition, pain threshold response varies greatly at different stages of the menstrual cycle.[42] Other studies have shown that women report greater pain relief from kappa opioid agonists (e.g., pentazocine) after endodontic treatment.[420] We should be aware that women might react differently to pain than men.[114]

CLINICAL ANESTHESIA AND ROUTES OF ANESTHETIC ADMINISTRATION

Recognition is growing that evidence-based therapeutics offers an excellent source of information that should become an aspect of treatment in conjunction with the practitioner's clinical skills and the patient's particular needs. In many areas of dentistry, this is a limited concept because few randomized, placebo-controlled, double-blind clinical trials have been conducted. However, this is not the case with dental pharmacology. The astute clinician can make informed decisions on various local anesthetics and routes of injection based on a large collection of well-designed clinical trials. The following discussion focuses on the clinical aspects of local anesthesia, with special emphasis on restorative dentistry and endodontics.

IMPORTANT CLINICAL FACTORS IN LOCAL ANESTHESIA

Traditional Methods of Confirming Anesthesia

Traditional methods of confirming anesthesia usually involve questioning the patient ("Is your lip numb?"), soft tissue testing (e.g., lack of mucosal responsiveness to a sharp explorer), or simply beginning treatment. However, these approaches may not be effective for determining pulpal anesthesia.[60,179,277,424]

Determining Pulpal Anesthesia in Asymptomatic Vital Teeth

Anesthesia in asymptomatic vital teeth can be measured more objectively by applying a cold refrigerant (Fig. 4-1) or by using an electrical pulp tester (EPT), as described in Chapter 1 (Fig. 4-2). Application of cold or the electrical pulp tester can be used to test the tooth under treatment for pulpal anesthesia before a clinical procedure is started.[56,100,201,257]

Determining Pulpal Anesthesia in Symptomatic Vital Teeth

In symptomatic (painful) vital teeth and after administration of a local anesthetic, testing with a cold refrigerant or an electrical pulp tester can be used to evaluate pulpal anesthesia before an endodontic procedure is started.[68,100,323,355] If the patient responds to the stimulus, pulpal anesthesia has not been obtained, and supplemental anesthetic should be administered. However, in patients presenting for an emergency appointment with a painful vital tooth (e.g., symptomatic irreversible pulpitis), the lack of a response to pulp testing may not guarantee pulpal anesthesia.[100,323,355] Therefore, if a patient experiences pain when the endodontic procedure is started, supplemental anesthetic is indicated, regardless of the responsiveness to pulpal testing. If the chamber is necrotic and the canals are vital, no objective test can predict the level of clinical anesthesia.

FIG. 4-1 A cold refrigerant may be used to test for pulpal anesthesia before the start of a clinical procedure. (Coltène/Whaledent Inc., Cuyahoga Falls, OH.)

FIG. 4-2 An electrical pulp tester also may be used to test for pulpal anesthesia before a clinical procedure is started. (Courtesy SybronEndo, Corporation Orange, CA.)

Patient Who Has Had Previous Difficulty with Anesthesia

Anesthesia is more likely to be unsuccessful in patients who report a history of previous difficulty with anesthesia.[214] These patients generally make comments such as, "Novocaine doesn't work on me" or "It takes a lot of shots to get my teeth numb." A good clinical practice is to ask the patient if dentists previously have had difficulty obtaining anesthesia. If the answer is yes, supplemental injections should be considered.

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Failure to Achieve Anesthesia in Patients with Pain

Obtaining anesthesia is often difficult in patients with endodontic pain of pulpal origin. A number of explanations have been proposed for this.[170] One is that conventional anesthetic techniques do not always provide profound pulpal anesthesia, and patients with preexisting hyperalgesia may be unable to tolerate any noxious input. Another explanation relates to the theory that inflamed tissue has a lower pH, which reduces the amount of the base form of anesthetic that penetrates the nerve membrane. Consequently, less of the ionized form is available in the nerve to achieve anesthesia. However, this explanation does not account for the mandibular molar with pulpitis that is not readily blocked by an inferior alveolar injection administered at some distance from the area of inflammation. Correlating localized inflammatory changes with failure of the IAN block is difficult.

Another explanation for failure is that nerves arising from inflamed tissue have altered resting potentials and decreased excitability thresholds.[51,427] Two studies demonstrated that local anesthetics were unable to prevent impulse transmission because of these lowered excitability thresholds.[292,427] Another factor might be the TTX-R sodium channels, which are resistant to the action of local anesthetics,[373] are increased in inflamed dental pulp,[399,431,433] and are sensitized by prostaglandins.[148] A related factor is the increased expression of sodium channels.[399,431,433]

Finally, patients in pain are often apprehensive, which lowers the pain threshold. Therefore, practitioners should consider supplemental techniques (e.g., intraosseous injections[320,323,332,355] or periodontal ligament injections[68]) if an IAN block fails to provide pulpal anesthesia for patients with irreversible pulpitis.

Use of Topical Anesthetics

Fear of needle insertion is a major cause of apprehension in dental patients.[234,289,290] Although some studies have demonstrated the effectiveness of topical anesthetics,[146,175,186,312,372] others have shown no significant pain reduction.[146,222,270] Interestingly, one study showed that patients who thought they were receiving a topical anesthetic anticipated less pain regardless of whether they actually received the anesthetic.[270] The most important aspect of a topical anesthetic may not be its clinical effectiveness, but rather its psychological effect on the patient, who believes the practitioner is doing everything possible to prevent pain.

Reversing the Action of Local Anesthetics

Phentolamine mesylate (0.4 mg in a 1.7-ml cartridge [OraVerse, Novalar Pharmaceuticals, San Diego, California]) is a recently developed agent that shortens the duration of soft tissue anesthesia. The duration of soft tissue anesthesia is longer than pulpal anesthesia and is often associated with difficulty eating, drinking, and speaking.[176,245] The best use of OraVerse is after dental procedures when postoperative pain is not a concern. Asymptomatic endodontic patients may benefit from the use of a reversal agent when they have speaking engagements or important meetings or must perform in musical or theatrical events.[121] Therefore, OraVerse may be used to shorten the duration of soft tissue anesthesia if the patient presents with an asymptomatic tooth and little postoperative pain is anticipated.[121]

INFERIOR ALVEOLAR NERVE BLOCK FOR RESTORATIVE DENTISTRY

2% Lidocaine and 1:100,000 Epinephrine

Because failure occurs most often with the IAN block,[214] factors that modify mandibular anesthesia must be carefully reviewed. The technique for administering an IAN block can be reviewed in available textbooks.[195,262] The following discussion reviews the expected outcomes after administration of a conventional IAN block to asymptomatic patients using 1.8 ml of 2% lidocaine with 1:100,000 epinephrine (Xylocaine, Lignospan, Octocaine). Although anesthesia requirements vary among dental procedures, the following discussion concentrates on *pulpal* anesthesia in asymptomatic patients and thus is directly relevant to endodontic therapy.

Anesthetic Success

One way to define anesthetic success for nerve blocks is the percentage of subjects who achieve two consecutive nonresponsive readings on electrical pulp testing within 15 minutes and continuously sustain this lack of responsiveness for 60 minutes. In other words, the objective is to achieve anesthesia within 15 minutes and to have it last 1 hour. This endpoint is as important for restorative dentistry as it is for endodontic treatment, so it is used as a benchmark for clinically significant information from research on local anesthetics. Using this criterion, the percentage of cases in which anesthesia was obtained after IAN block injections ranged from 10% (central incisor) to 65% (second molar).* It is important to note that all patients from these studies reported a positive lip sign (e.g., profound lip numbness); therefore, profound lip numbness *does not* predict pulpal anesthesia. However, lack of soft tissue anesthesia is a useful indicator that the block injection was not administered accurately for that patient. Missed blocks occur in about 5% of cases, and the clinician should readminister the nerve block before continuing with treatment.

Anesthetic Failure

Anesthetic failure can be defined as the percentage of subjects who never achieved two consecutive nonresponsive EPT readings at any time during a 60-minute period. Using this criterion, anesthetic failure rates ranged from 17% (second molar) to 58% (central incisor).†

Noncontinuous Anesthesia

Another measure of mandibular anesthesia is noncontinuous anesthesia, which may be related to the action of the anesthetic solution on the nerve membrane (blocking and unblocking the sodium channels). This occurs in about 12% to 20% of patients.‡

Slow Onset

After a conventional IAN block injection, the onset of pulpal anesthesia occurs within 10 to 15 minutes in most cases (Fig. 4-3).§ Slow onset can be defined as the percentage of

*References 60, 165, 179, 277, 322, 353, and 424.
†References 60, 112, 165, 179, 277, 322, 353, 388, 424, and 425.
‡References 60, 158, 165, 179, 277, 322, 353, and 424.
§References 60, 112, 165, 179, 277, 322, 353, 388, 424, and 425.

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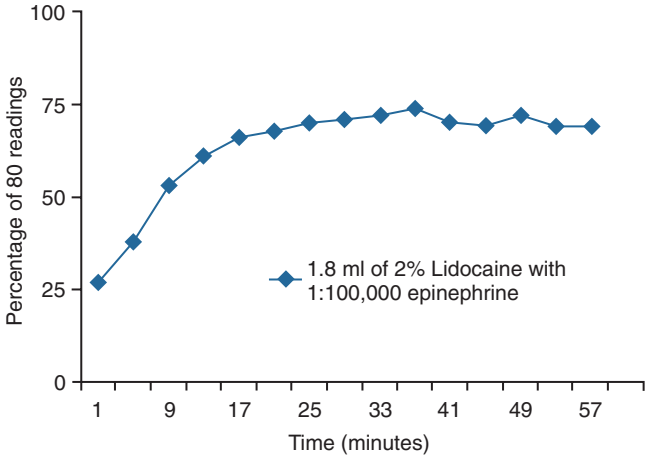

FIG. 4-3 Incidence of first mandibular molar anesthesia as determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes.

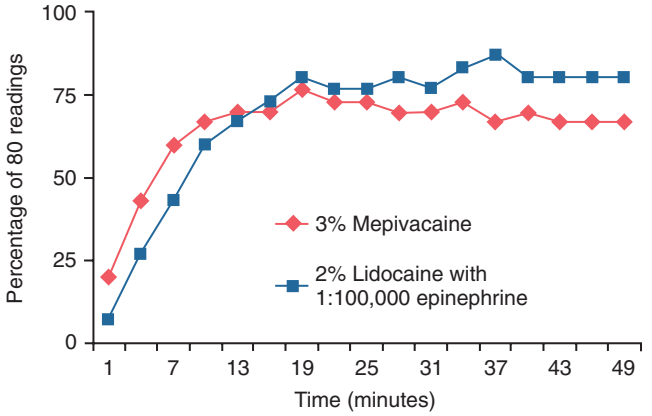

FIG. 4-4 Incidence of first mandibular molar anesthesia: comparison of 3% mepivacaine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 50 minutes. No significant difference between the two solutions was noted.

subjects who achieved a nonresponsive EPT reading after 15 minutes. In mandibular teeth, slow onset occurs in 12% to 20% of patients.

Duration

The duration of action for pulpal anesthesia in the mandible is very good.* If patients are anesthetized initially, anesthesia usually persists for approximately 2½ hours.[112] Figure 4-3 depicts the time course for complete pulpal anesthesia for an asymptomatic first molar, as defined by the percentage of patients who did not respond to a stimulus (EPT) across time for 60 minutes. Most patients achieved pulpal anesthesia within 15 minutes and had a duration of anesthesia of at least 1 hour, but the success rate was not 100% for the population.

ALTERNATIVE ANESTHETIC SOLUTIONS FOR THE INFERIOR ALVEOLAR NERVE BLOCK

Plain Solutions: 3% Mepivacaine (Carbocaine, Polocaine, Scandonest) and 4% Prilocaine (Citanest Plain)

In a study of volunteers without dental pathosis, anesthesia from IAN injection of 3% mepivacaine plain and 4% prilocaine plain was as effective as that from 2% lidocaine with 1:100,000 (Fig. 4-4).[277] A clinical study of patients with irreversible pulpitis also found that 3% mepivacaine and 2% lidocaine with 1:100,000 epinephrine were equivalent for IAN blocks.[68] These findings support the selection of 3% mepivacaine as a local anesthetic when medical conditions or drug therapies suggest caution in the administration of solutions containing epinephrine.

4% Prilocaine with 1:200,000 Epinephrine (Citanest Forte) and 2% Mepivacaine with 1:20,000 Levonordefrin (Carbocaine with Neo-Cobefrin)

In a study of volunteers without dental pathosis, IAN injection of 4% prilocaine with 1:200,000 epinephrine or 2% mepivacaine with 1:20,000 levonordefrin worked as well as 2% lidocaine with 1:100,000 epinephrine in achieving pulpal anesthesia.[179]

Levonordefrin has 75% alpha activity and only 25% beta activity, making it seemingly more attractive than epinephrine (50% alpha activity and 50% beta activity).[262] However, levonordefrin is marketed as a 1:20,000 concentration in dental cartridges.[262] Clinically, the higher concentration of levonordefrin makes it equipotent to epinephrine in clinical and systemic effects,[158,179] so 1:20,000 levonordefrin offers no clinical advantage over 1:100,000 epinephrine.

Articaine with 1:100,000 Epinephrine (Septocaine, Articadent, Zorcaine)

Articaine has been reported to be a safe and effective local anesthetic.[378] It was approved for use in the United States in April, 2000, and is marketed as a 4% solution with either 1:100,000 or 1:200,000 epinephrine.[265,299] Articaine is classified as an amide. It has a thiophene ring (instead of a benzene ring, as do the other amide local anesthetics) and an extra ester linkage, which results in hydrolysis of articaine by plasma esterases.[271] A number of studies have evaluated articaine and concluded that it is safe when used in appropriate doses.* Lidocaine and articaine have the same maximal dose of 500 mg for adult patients (recommended dose, 6.6 to 7 mg/kg), but the maximum number of cartridges is different because of the differences in drug concentration (see Table 4-2).[262]

*References 60, 112, 165, 179, 277, 322, 353, 388, 424, and 425.

*References 82, 178, 193, 263, 265, 293, 328, 389, and 449.

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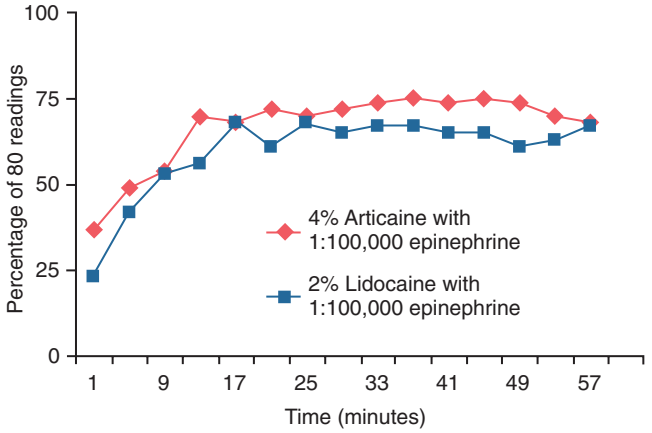

FIG. 4-5 **Incidence of first mandibular molar anesthesia: comparison of 4% articaine with 1:100,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine.** Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two solutions was noted.

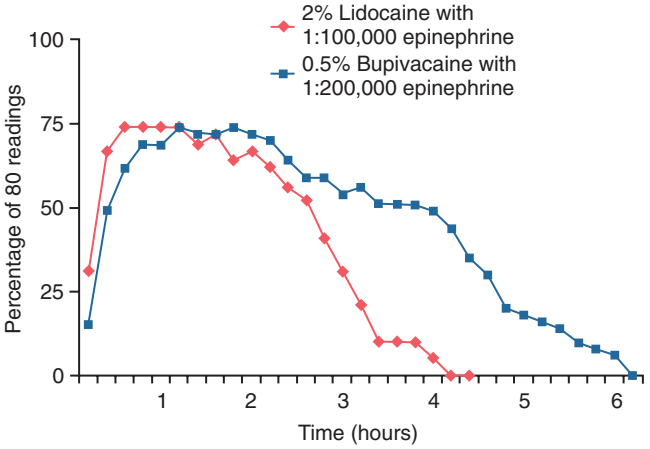

FIG. 4-6 **Incidence of first mandibular molar anesthesia: comparison of 0.5% bupivacaine with 1:200,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine.** Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 6 hours. The bupivacaine solution showed a longer duration of anesthesia than the lidocaine solution.

Clinical Effectiveness of Articaine for Inferior Alveolar Nerve Blocks

The available literature indicates that articaine is equally effective for IAN blocks when statistically compared to other local anesthetics.* In comparing the anesthetic efficacy of 4% articaine with 1:100,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine for IAN blocks, one study found that the two solutions were not significantly different (Fig. 4-5).[288] Two studies found no difference in efficacy between 4% articaine with 1:100,000 and 1:200,000 epinephrine.[299,412] In summary, repeated clinical trials have failed to demonstrate any statistical superiority of articaine over lidocaine for IAN blocks.

Articaine and Uncorroborated Insurance Carrier Warning

A letter was sent to thousands of U.S. dentists in 2006 by insurer Emery and Webb/Ace USA stating, "... we have noticed an increase in reversible and, in some cases, nonreversible paresthesias (with Septocaine) … We are writing you to alert you to these events in hopes that you will not fall victim to one of these incidents."[261] Knowledgeable dentists and educators communicated their concerns, and a Notice of Retraction was issued:

> Unfortunately, we at Emery & Webb discovered upon further review, and subsequent to the mailings, that both documents contained inaccuracies and an alarmist tone, which was not warranted … Emery and Webb has not noted an increase in malpractice claims or lawsuits in connection with articaine … It should be made clear that Emery and Webb has not conducted any scientific investigation, sampling, testing, or other investigation of the articaine anesthetic, and has no independent knowledge or data which would restrict the use of the product.[261]

Astute clinicians should be very careful of Web chat sites and colleagues' clinical endorsements, because they may not accurately reflect the correct information regarding articaine.

Long-Acting Anesthetics

Clinical trials with bupivacaine (Marcaine) and etidocaine (Duranest) have been conducted in patients undergoing oral surgery,[84,371] endodontic treatment,[104,300] and periodontic treatment.[78,254] Etidocaine was withdrawn from the market by Dentsply Pharmaceuticals (York, Pennsylvania). Bupivacaine was found to have a slower onset of pulpal anesthesia than lidocaine for IAN blocks.[112] Generally, bupivacaine provides prolonged analgesia and is indicated when postoperative pain is anticipated, but not all patients want lip numbness for an extended period.[371] Patients should be questioned about their preference. Although bupivacaine has a somewhat slower onset than lidocaine, its duration of pulpal anesthesia in the mandible is almost twice as long (approximately 4 hours; Fig. 4-6).[112]

Ropivacaine (Naropin), a relatively new long-acting local anesthetic, is a structural homolog of bupivacaine.[223] A number of studies have shown that ropivacaine has a lower potential for toxic CNS and cardiovascular effects than bupivacaine but produces equivalent pharmacologic effects.[223] Ropivacaine and levobupivacaine are being developed as potentially new local anesthetics based on their stereochemistry. Both are S-isomers and are thought to cause less toxicity than the racemic mixture of bupivacaine currently marketed.[392] A clinical trial has indicated that levobupivacaine showed significantly better postoperative pain control at 4 and 24 hours after infiltration injection than ropivacaine.[331] Because of their decreased potential for cardiac and CNS toxicity, ropivacaine and levobupivacaine may replace bupivacaine with epinephrine in clinical dental practice.

Buffered Lidocaine

Buffering lidocaine using sodium bicarbonate raises the pH of the anesthetic solution. In medicine there is evidence that buffering lidocaine results in less pain during the injection.[55,164]

*References 64, 95, 159, 160, 263, 264, 288, 421, and 450.

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In dentistry, some studies[15,16,44,210] found that buffered lidocaine produced less pain on injection and a faster onset of anesthesia. However, other dental studies[350,435] did not find less pain on injection or a faster onset with buffered lidocaine for IAN block. Using a commercial buffering system (Onpharma, Los Gatos, California) in asymptomatic subjects, one study[266] found a reduction in onset time and injection pain, whereas another study[180] found no difference in these measurements. In symptomatic patients with a diagnosis of pulpal necrosis and associated acute swelling, no significant decrease in pain of infiltrations or significant decrease in pain of an incision and drainage procedure was found when the buffered anesthetic formulation was used.[29] Most patients who had the incision and drainage procedure experienced moderate to severe pain.

Use of Mannitol

An Ohio State University research group studied the use of mannitol to increase the efficacy of nerve blocks. Mannitol, a hyperosmotic sugar solution, is thought to temporarily disrupt the protective covering (perineurium) of sensory nerves, allowing the local anesthetic to gain entry to the innermost part of the nerve.[22] These researchers found that the use of mannitol in combination with lidocaine increased anesthetic success in IAN blocks about 15% to 20% but did not provide complete pulpal anesthesia for restorative or endodontic treatment.[239,398,443] The drug combination may be introduced sometime in the future.

ALTERNATIVE INJECTION SITES

Gow-Gates and Vazirani-Akinosi Techniques

Some clinicians have reported that the Gow-Gates technique[154] has a higher success rate than the conventional IAN block injection,[259,262] but controlled experimental studies have failed to show superiority of the Gow-Gates technique.[11,149,294,411] Neither has the Vazirani-Akinosi technique[12,149,262] been found superior to the standard inferior alveolar injection.[149,271,394,411,460] In a small study of 21 patients, no difference was found between lidocaine (11 patients) and articaine (10 patients) formulations for the Gow-Gates injection in patients with irreversible pulpitis.[384] Another study found the Gow-Gates technique had a higher success rate (52%) than the Vazirani-Akinosi technique (41%) in patients with irreversible pulpitis.[4] Further research is indicated with both techniques in patients presenting with symptomatic irreversible pulpitis. The Vazirani-Akinosi technique is indicated for cases involving a limited mandibular opening (trismus).

Incisive Nerve Block/Infiltration at the Mental Foramen

The incisive nerve block is successful 80% to 83% of the time in anesthetizing the premolar teeth for about 20-30 minutes.[30,202,313,437] It is not effective for the central and lateral incisors.[313]

Lidocaine Infiltrations

Labial or lingual infiltration injections of a lidocaine solution alone are not effective for pulpal anesthesia in the mandible.[118,281,459]

Articaine Infiltrations

Articaine is significantly better than lidocaine for buccal infiltration of the mandibular first molar.[75,203,205,362] However, articaine alone does not predictably provide pulpal anesthesia of the first molar. There is no difference between 4% articaine with 1:100,000 and 1:200,000 epinephrine for buccal infiltration.[275]

In anterior teeth, buccal and lingual infiltrations of articaine provide initial pulpal anesthesia, but the anesthesia declines over 60 minutes.[190,326]

ATTEMPTS TO INCREASE SUCCESS OF THE INFERIOR ALVEOLAR NERVE BLOCK

Increasing the Volume of Anesthetic

One possible method for increasing anesthetic success could be to double the injection volume of local anesthetic solution. However, increasing the volume of 2% lidocaine with epinephrine to 3.6 ml (two cartridges) does not increase the incidence of pulpal anesthesia with the IAN block (Fig. 4-7).[322,455]

Increasing the Epinephrine Concentration

A second approach for increasing the success of the IAN block could be to increase the concentration of epinephrine. However, when this technique was evaluated in clinically normal teeth, no advantage was seen in using a higher concentration (1:50,000 versus 1:100,000) of epinephrine.[80,425]

Addition of Hyaluronidase

Hyaluronidase reduces the viscosity of the injected tissue, permitting a wider spread of injected fluids.[21] Early studies in dentistry found that an IAN block was more easily attained and was more complete when hyaluronidase was added to an anesthetic solution.[230,258] A recent study found that hyaluronidase may increase the duration of the effects of lidocaine.[377] However, a controlled clinical trial found that adding hyaluronidase to a lidocaine solution with epinephrine did not statistically increase the incidence of pulpal anesthesia in IAN blocks.[360] In addition, hyaluronidase increased the occurrence of adverse effects (i.e., increased pain and trismus).[360]

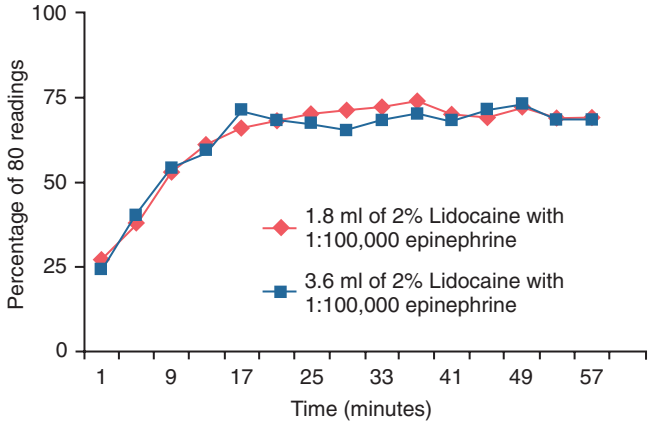

FIG. 4-7 Incidence of first mandibular molar anesthesia: comparison of 1.8 ml and 3.6 ml of 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two volumes was noted.

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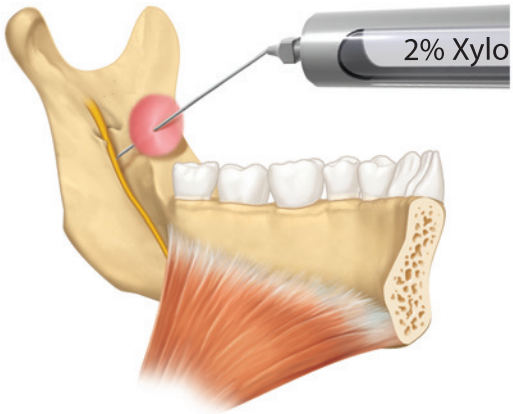

FIG. 4-8 Injection site for the mylohyoid nerve block.

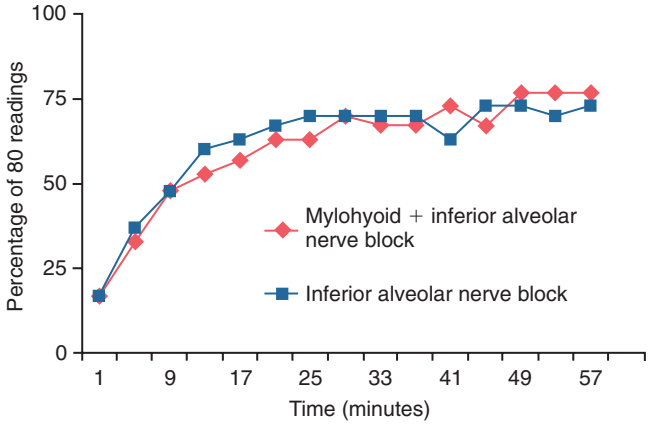

FIG. 4-9 Incidence of first mandibular molar anesthesia: comparison of the combination mylohyoid infiltration plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two techniques was noted.

CARBONATED ANESTHETIC SOLUTIONS

Experimentally, carbonated anesthetic solutions are more effective because the anesthetic is trapped in the nerve.[60] In addition, carbon dioxide (CO_2) has a synergistic relationship with local anesthetics and a direct depressant action on nerves.[60] However, a controlled clinical study was unable to demonstrate a superior effect of lidocaine hydrocarbonate in IAN blocks.[60]

Diphenhydramine as a Local Anesthetic Agent

Diphenhydramine (Benadryl) has been advocated for patients who are allergic to commonly used local anesthetics. Two studies found that diphenhydramine was less effective than lidocaine for extractions.[286,432] Another study found that the combinations of lidocaine/diphenhydramine with epinephrine, and diphenhydramine with epinephrine, were significantly less effective for pulpal anesthesia than lidocaine with epinephrine for IAN blocks.[440] These researchers also found that the diphenhydramine solutions were more painful on injection and had a high incidence of moderate postoperative pain.

Addition of Meperidine to Lidocaine

Two studies found that the addition of meperidine (Demerol) to a lidocaine formulation did not increase the success of the IAN block.[37,151]

FACTORS IN FAILURE OF THE INFERIOR ALVEOLAR NERVE BLOCK

Accessory Innervation: Mylohyoid Nerve

The mylohyoid nerve is the accessory nerve most often cited as a cause of failure of mandibular anesthesia.[127,441] A controlled clinical trial compared the IAN block alone to a combination of the IAN block and a mylohyoid nerve block using 2% lidocaine with 1:100,000 epinephrine (Fig. 4-8), which was aided by the use of a peripheral nerve stimulator.[66] The investigators found that the mylohyoid injection did not significantly enhance pulpal anesthesia of the IAN block (Fig. 4-9), so the study does not support the hypothesis that the mylohyoid nerve is a major factor in failure of the IAN block.

Accuracy of Injection

It has been theorized that an inaccurate injection contributes to inadequate mandibular anesthesia, but a number of studies determined that the use of ultrasound, a peripheral nerve stimulator, or radiographs to guide needle placement for IAN blocks did not result in more successful pulpal anesthesia.[35,134,165,387] The authors of these studies speculated that the anesthetic solution migrated along the path of least resistance, which was determined by fascial planes and structures encountered in the pterygomandibular space. These studies highlight an important clinical point: Lack of pulpal anesthesia is not necessarily the result of an inaccurate injection.

Needle Deflection

Needle deflection has been proposed as a cause of failure with the IAN block.[70,83,182] Several in vitro studies have shown that beveled needles tend to deflect toward the nonbeveled side (i.e., away from the bevel).* To compensate for this, a bidirectional needle rotation technique using the computer-controlled local anesthetic delivery system (CCLAD) (Milestone Scientific, Livingston, New Jersey) has been proposed in which the CCLAD handpiece assembly and needle are rotated in a fashion similar to the rotation of an endodontic hand file.[182] The technique was found to reduce deflection during insertion of the needle. A controlled clinical trial compared the anesthetic success of the conventional IAN block using two needle insertion methods.[224] However, no significant difference in anesthetic success was seen when the needle bevel was oriented away from the mandibular ramus (so that the needle would deflect toward the mandibular foramen [50% success]) compared with the bidirectional CCLAD needle rotation technique (56% success).[224] Neither technique resulted in an acceptable rate of anesthetic success in patients with symptomatic irreversible pulpitis.

Needle Bevel and Success

In asymptomatic subjects, the orientation of the needle bevel away or toward the mandibular ramus for an IAN block did

*References 13, 70, 83, 182, 199, and 363.

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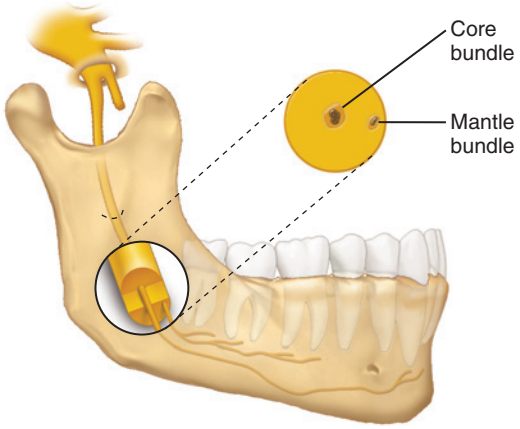

FIG. 4-10 Central core theory. The axons in the mantle bundle supply the molar teeth, and those in the core bundle supply the anterior teeth. The extraneural local anesthetic solution diffuses from the mantle to the core. (Modified and redrawn from De Jong RH: *Local anesthetics,* St Louis, 1994, Mosby.)

not affect anesthetic success or failure.[405] Therefore, the use of commercial needles with markers to indicate the needle bevel is not necessary.

Speed of Injection and Success

A slow inferior alveolar nerve block increases success over a fast injection[204] but not for patients diagnosed with irreversible pulpitis.[9]

Cross-Innervation

Cross-innervation from the contralateral inferior alveolar nerve has been implicated in failure to achieve anesthesia in anterior teeth after an IAN injection. Experimentally, cross-innervation occurs in incisors[367,458] but plays a very small role in failure of an IAN block.

Red Hair

In medicine, red-haired females have shown reduced subcutaneous efficacy of lidocaine and increased requirements for desflurane.[102] However, in dentistry, having red hair was unrelated to success rates of the inferior alveolar nerve block,[102] although it has been shown to be associated with higher levels of dental anxiety.[102]

A Theory on Why Failure Occurs with the Inferior Alveolar Nerve Block in Restorative Dentistry

The central core theory may be the best explanation of why failure occurs with the IAN block.[85,407] According to this theory, nerves on the outside of the nerve bundle supply molar teeth, and nerves on the inside of the nerve bundle supply anterior teeth (Fig. 4-10). Even if deposited at the correct site, the anesthetic solution may not diffuse into the nerve trunk and reach all nerves to produce an adequate block. Although this theory may explain the higher experimental failure rates with the IAN block in anterior teeth compared with posterior teeth,* it does not explain the increased failure rate observed in painful teeth.

*References 60, 112, 165, 179, 277, 322, 353, 388, 424, and 425.

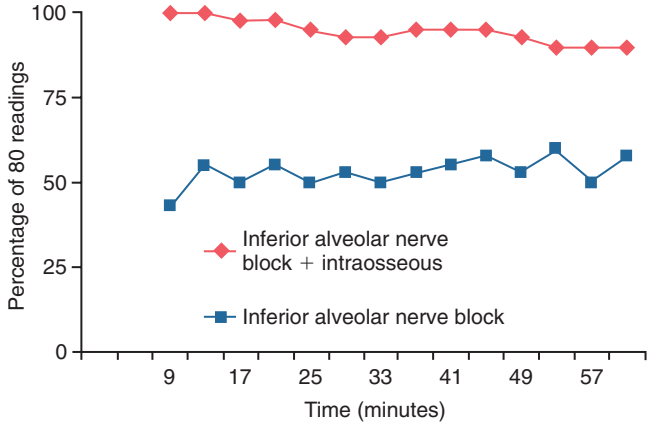

FIG. 4-11 Incidence of first mandibular molar anesthesia: comparison of the combination intraosseous injection of 2% lidocaine with 1:100,000 epinephrine plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The combination technique was significantly better at all postinjection times.

ENHANCEMENT OF MANDIBULAR ANESTHESIA FOR RESTORATIVE DENTISTRY

Supplemental Articaine Infiltrations

An important clinical finding is that an articaine infiltration of the first molar, premolars, and anterior teeth after an IAN block should provide pulpal anesthesia for approximately 1 hour.[163,206,326] The second molar may require a supplemental intraosseous (IO) or intraligamentary (IL) injection to achieve success.

Supplemental Intraosseous Anesthesia

Supplemental IO injections of lidocaine and mepivacaine with vasoconstrictors allow quick onset and increase the success of the inferior alveolar nerve block for approximately 60 minutes (Fig. 4-11).[103,158] The addition of a supplemental IO injection reduced the incidence of slow onset of pulpal anesthesia to zero compared with the IAN block alone (18% incidence).[103] Using 3% mepivacaine plain for IO injection results in pulpal anesthesia for approximately 30 minutes (Fig. 4-12).[137]

Supplemental Intraligamentary Anesthesia

Supplemental IL injections of 2% lidocaine with 1:100,000 epinephrine increase the success of the inferior alveolar nerve block, but the duration is approximately 23 minutes.[61]

MAXILLARY ANESTHESIA FOR RESTORATIVE DENTISTRY

Descriptions of conventional techniques for maxillary anesthesia are available for review in numerous articles and textbooks.[195,262]

2% Lidocaine with 1:100,000 Epinephrine

As a frame of reference, the most commonly used injection for anesthetization of maxillary teeth is infiltration with a cartridge of 2% lidocaine with 1:100,000 epinephrine.

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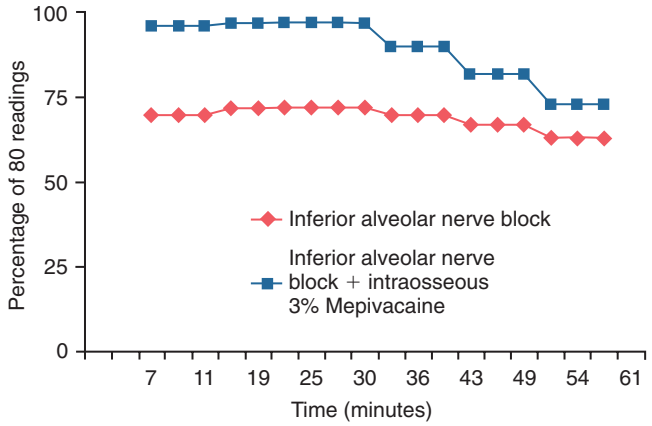

FIG. 4-12 **Incidence of first mandibular molar anesthesia: comparison of the combination intraosseous injection with 3% mepivacaine plus the inferior alveolar nerve block to the inferior alveolar nerve block alone.** Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The combination technique proved significantly better for approximately 30 minutes.

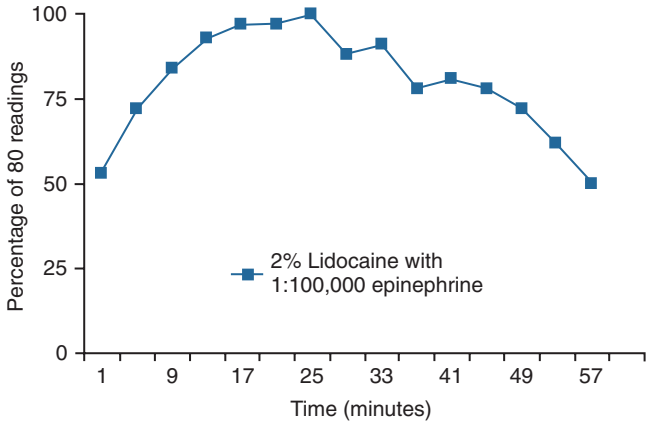

FIG. 4-13 Incidence of first maxillary molar anesthesia, as determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes.

Anesthetic Success

Infiltration results in a fairly high incidence of successful pulpal anesthesia (around 87% to 92%).* However, some patients may not be anesthetized because of individual variations in response to the drug administered, operator differences, and variations of anatomy and tooth position.

Onset of Pulpal Anesthesia

Pulpal anesthesia usually occurs in 3 to 5 minutes.†

Duration of Pulpal Anesthesia

The duration of pulpal anesthesia is a problem with maxillary infiltrations.‡ Pulpal anesthesia of the anterior teeth declines after about 30 minutes, with most losing anesthesia by 60 minutes.§ In premolars and first molars, pulpal anesthesia is good until about 40-45 minutes and then it starts to decline.‖ Additional local anesthetic should be administered depending on the duration of the procedure and the tooth group affected.

Time Course of Pulpal Anesthesia for the Maxillary First Molar

Figure 4-13 shows the time course for complete pulpal anesthesia for an asymptomatic first molar, as defined by the percentage of patients who do not respond at all to an EPT stimulus over time. Some patients had a slow onset of anesthesia until around 11 minutes. The overall success rate (no response at the device's highest setting) is 95% to 100%, with peak effects observed at around 30 minutes after injection.

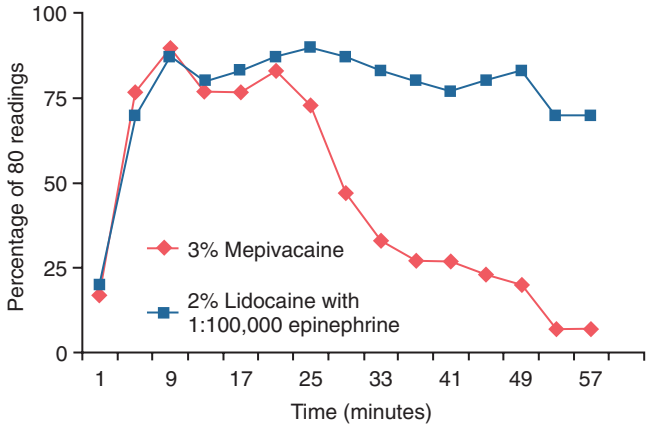

FIG. 4-14 **Incidence of first maxillary molar anesthesia: comparison of 3% mepivacaine to 2% lidocaine with 1:100,000 epinephrine.** Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The 3% mepivacaine showed a shorter duration of anesthesia than the lidocaine solution.

Significance of Lip Numbness

Soft tissue anesthesia (lip or cheek numbness) is not necessarily related to the duration of pulpal anesthesia. Pulpal anesthesia does not last as long as soft tissue anesthesia.[156,272,287]

ALTERNATIVE ANESTHETIC SOLUTIONS FOR INFILTRATIONS

Plain Solutions: 3% Mepivacaine (Carbocaine, Polocaine, Scandonest) and 4% Prilocaine (Citanest Plain)

Anesthesia duration is shorter with these solutions.[211,272] Therefore, use these for procedures of short duration (10 to 15 minutes) (Fig. 4-14). These agents are generally not as safe as solutions with vasoconstrictors if *large* volumes are administered because they are rapidly absorbed systemically, resulting in excessive plasma concentrations and possible toxic reactions.[262]

*References 48, 108, 156, 211, 246, 272, 287, 324, 334, 353, and 381.
†References 48, 108, 156, 211, 246, 272, 287, 324, 334, 353, and 381.
‡References 48, 108, 156, 211, 246, 272, 287, 324, 334, 353, and 381.
§References 48, 108, 156, 211, 246, 272, 287, 324, 334, 353, and 381.
‖References 48, 108, 156, 211, 246, 272, 287, 324, 334, 353, and 381.

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4% Prilocaine with 1:200,000 Epinephrine (Citanest Forte), 2% Mepivacaine with 1:20,000 Levonordefrin (Carbocaine with Neo-Cobefrin), and 4% Articaine with 1:100,000 Epinephrine (Septocaine, Articadent, Zorcaine)

These formulations are similar to 2% lidocaine with 1:100,000 epinephrine.[108,211,246]

0.5% Bupivacaine with Epinephrine (Marcaine)

Success rates (no response to EPT) with bupivacaine range from 80% to 95% in the maxillary lateral incisor, compared with 50% in the maxillary second premolars.[81,156,223,410] Although bupivacaine provides long-term anesthesia with the IAN block, it *does not* provide prolonged pulpal anesthesia with maxillary infiltration injection.[81,156,223] In the lateral incisor, bupivacaine has a shorter duration of pulpal anesthesia than lidocaine.[81,156] In the first molar, bupivacaine's duration of pulpal anesthesia is equivalent to that of lidocaine.[156] Neither agent provides pulpal anesthesia for an hour.[81,156]

EXTENDING THE DURATION OF PULPAL ANESTHESIA FOR MAXILLARY TEETH

Increasing the Solution Volume

A two-cartridge volume of 2% lidocaine with epinephrine extends the duration of pulpal anesthesia but not for 60 minutes.[287]

Increasing the Epinephrine Concentration

Increasing the epinephrine concentration to 1:50,000 epinephrine increases duration for the lateral incisor but not the first molar.[272] Duration was not 60 minutes in either teeth.[272]

Repeating the Infiltration

Adding another cartridge of 2% lidocaine with epinephrine at 30 minutes in anterior teeth and 45 minutes in posterior teeth significantly improves the duration of pulpal anesthesia and may be the best way to extend the duration of pulpal anesthesia (Fig. 4-15).[381]

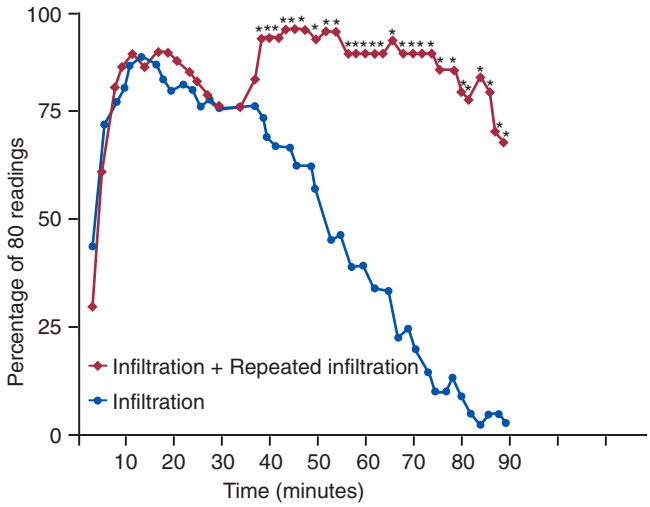

FIG. 4-15 Incidence of maxillary lateral incisor pulpal anesthesia using an initial infiltration and a repeated infiltration 30 minutes later (both infiltrations used 1.8 ml of 2% lidocaine with 1:100,000 epinephrine). Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings). The repeated infiltration injection significantly prolonged the duration of pulpal anesthesia.

ALTERNATIVE MAXILLARY INJECTION TECHNIQUES FOR RESTORATIVE DENTISTRY

Posterior Superior Alveolar (PSA) Nerve Block

The PSA nerve block anesthetizes the second molars and about 80% of first molars.[257,343] An additional mesial infiltration injection may be necessary to anesthetize the first molar. Generally, the PSA block injection is not advocated for routine restorative procedures. An infiltration of the molars is preferred.

Infraorbital Nerve Block

The infraorbital nerve block results in lip numbness but does not predictably anesthetize incisor pulps.[33,209] It usually anesthetizes the canines and premolars, but the duration is less than 1 hour.[33,209] Essentially, this injection technique is the same as an infiltration injection over the premolar teeth for pulpal anesthesia. Generally, the infraorbital injection is not advocated for routine restorative procedures. An infiltration of the individual maxillary teeth is preferred.

Second Division Nerve Block

The second division nerve block usually anesthetizes pulps of molars and some second premolars but does not predictably anesthetize first premolars, canines, or lateral and central incisors.[47,117] The high tuberosity technique is preferred to the greater palatine approach because it is easier and less painful.[47] Generally, the second division nerve block is not advocated for routine restorative procedures. An infiltration of the individual teeth is preferred.

Palatal–Anterior Superior Alveolar (P-ASA) Nerve Block

Traditionally, maxillary anterior teeth have been anesthetized with an infiltration injection near the apex of the target tooth. In the late 1990s, the P-ASA injection, a site-specific injection for maxillary anterior teeth, was introduced.[125,126] The P-ASA injection involves a palatal injection into the incisive canal; it derives its name from the injection's supposed ability to anesthetize both the right and left anterior superior alveolar nerves (Fig. 4-16). Unfortunately, the injection technique does not provide predictable pulpal anesthesia for the incisors and canines[50] and is often painful.[318]

Anterior Middle Superior Alveolar (AMSA) Nerve Block

The AMSA injection is another new technique for anesthetizing maxillary teeth.[123,124,126,130] The AMSA injection site is located palatally at a point that bisects the premolars and is approximately halfway between the midpalatine raphe and the crest of the free gingival margin (Fig. 4-17). The AMSA injection

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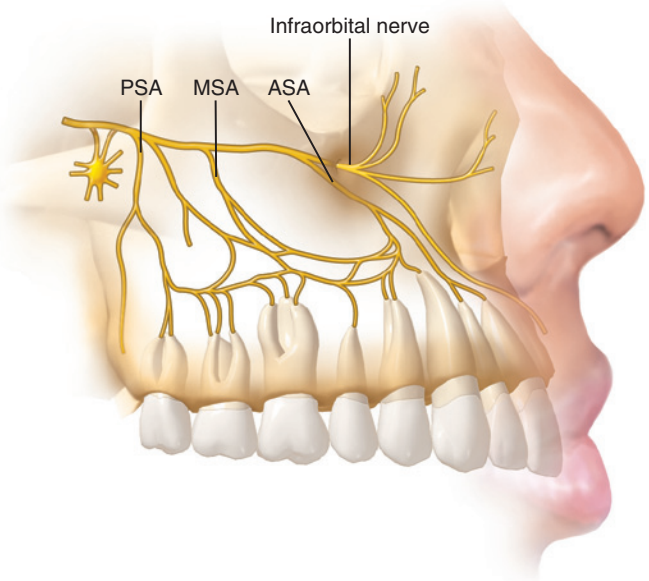

FIG. 4-16 Distribution of the maxillary division of the trigeminal nerve, showing the anterior superior alveolar (ASA) nerve, the middle superior alveolar (MSA) nerve, and the posterior superior alveolar (PSA) nerve.

FIG. 4-17 Palatal injection site for the anterior middle superior alveolar (AMSA) injection.

supposedly can anesthetize both the anterior and middle superior alveolar nerves[123,124,126] (see Fig. 4-16), resulting in unilateral anesthesia of the maxillary central and lateral incisors, canines, and first and second premolars. Unfortunately, the injection technique does not provide predictable pulpal anesthesia for these maxillary teeth[247] and is often painful.[321]

Pain, Inflammation, and Anxiety

Results differ from the normal when anesthesia is given to patients with any of these conditions.

SUPPLEMENTAL ANESTHESIA FOR RESTORATIVE DENTISTRY IN THE MANDIBLE AND MAXILLA

Indications

A supplemental injection is used if the standard injection is not effective. It is useful to repeat an initial injection only if the patient is not exhibiting the "classic" signs of soft tissue anesthesia (e.g., numbness of the lip). Generally, if the classic signs are present, reinjection is not very effective.[208] For example, after the inferior alveolar nerve block, the patient develops lip, chin, and tongue numbness and quadrant "deadness" of the teeth. A useful procedure is to pulp test the tooth with a cold refrigerant or an electrical pulp tester before the cavity preparation is begun.[56,100] If the patient feels pain to cold, a supplemental injection is indicated. To think that reinjection using the inferior alveolar nerve block approach will be successful is wishful thinking; failure the first time is usually followed by failure on the second attempt. Clinicians may think another injection is helpful because the patient sometimes achieves pulpal anesthesia after the second injection, but the patient may simply be experiencing slow onset of pulpal anesthesia from the first injection.

The clinician should go directly to a supplemental technique. Three such injections are *the infiltration injection; the IO injection; and the IL injection (formerly called the periodontal ligament [PDL] injection)*.

Infiltrations

Additional Infiltration of Lidocaine in the Maxilla

Because the duration of pulpal anesthesia for infiltration in the maxilla is not 60 minutes, adding an additional cartridge of 2% lidocaine with 1:100,000 epinephrine at 30 minutes in the anterior teeth and at about 45 minutes in premolar and molar teeth significantly improves the duration of pulpal anesthesia. This is an important clinical finding and may be the best way to extend the duration of pulpal anesthesia in maxillary teeth.[381]

Additional Infiltration of Articaine in the Mandible

Another important clinical finding is that an articaine infiltration of the first molar, premolars, and anterior teeth after an inferior alveolar nerve block should provide pulpal anesthesia for approximately 1 hour.[163,206,326] The second molar may require a supplemental IO or IL injection to achieve success.

Intraosseous Anesthesia

The IO injection is a supplemental technique that has been shown to be effective through substantial research and clinical usage. It is particularly useful in conjunction with a conventional injection when it is likely that supplemental anesthesia will be necessary (e.g., in mandibular second molar teeth).[103,137,158] The IO injection allows placement of a local anesthetic directly into the cancellous bone adjacent to the tooth. When a primary IO injection was compared with an infiltration injection, the IO technique showed a quicker onset and a shorter duration of anesthesia (Fig. 4-18).[324]

The Stabident IO system (Fairfax Dental Inc., Miami, Florida) is composed of a slow-speed, handpiece-driven perforator and a solid 27-gauge wire with a beveled end that drills a small hole through the cortical plate (Fig. 4-19). The anesthetic solution is delivered to cancellous bone through the 27-gauge, ultrashort injector needle placed into the hole made by the perforator (Fig. 4-20).

The X-Tip IO anesthetic delivery system (Dentpsly International, York, Pennsylvania) consists of an X-tip that separates into two parts, the drill and the guide sleeve (Fig. 4-21). The drill, a special hollow needle, leads the guide sleeve through the cortical plate; it then is separated from the guide sleeve and withdrawn. The remaining guide sleeve is designed to accept a 27-gauge needle for injection of the anesthetic

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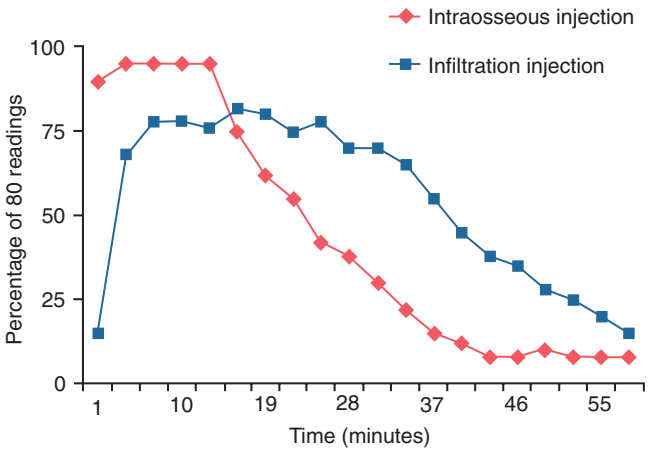

FIG. 4-18 **Incidence of anesthesia for intraosseous and infiltration injections.** Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The intraosseous injection showed a quicker onset and a shorter duration of anesthesia.

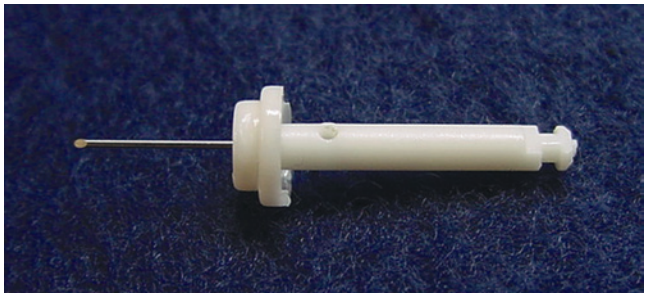

FIG. 4-19 The Stabident perforator, a solid, 27-gauge wire with a beveled end that is placed in a slow-speed handpiece.

FIG. 4-20 The anesthetic solution is delivered to the cancellous bone through the needle placed into the hole made by the perforator.

solution (Fig. 4-22). The guide sleeve is removed after the IO injection is complete.

The technique for IO injection of anesthetic using the Stabident or X-Tip system can be reviewed in the systems' instruction manuals or in published papers.[67,138,356-358]

Success

Supplemental IO injections of lidocaine and mepivacaine with vasoconstrictors allow quick onset and increase the success of

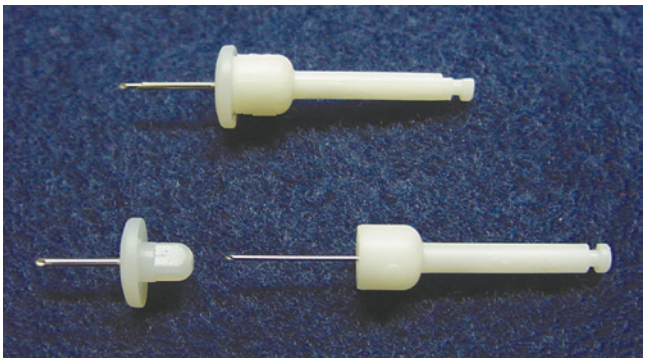

FIG. 4-21 The X-Tip anesthetic delivery system consists of an X-tip *(top)* that separates into two parts: the drill (a special hollow needle) and the guide sleeve component *(bottom)*.

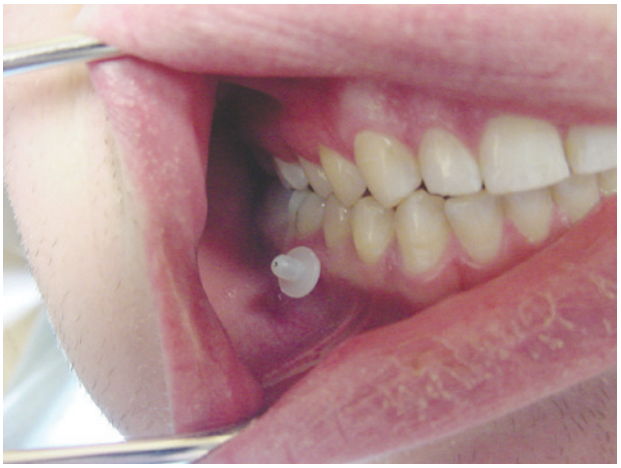

FIG. 4-22 Anesthetic solution is injected through the X-tip guide sleeve.

the inferior alveolar nerve block for approximately 60 minutes (see Fig. 4-11)[103,158] The addition of a supplemental IO injection reduced the incidence of slow onset of pulpal anesthesia to zero compared with the IAN block alone (18% incidence).[103] Using 3% mepivacaine plain results in pulpal anesthesia for approximately 30 minutes (see Fig. 4-12).[137]

Failure

If the anesthetic solution flows out of the perforation site (backflow), anesthetization will not occur.[139] Reperforation or use of another perforation site is a practical way to gain access to the cancellous bone in such cases. In fewer than 10% of cases, constricted cancellous spaces may limit the distribution of the anesthetic solution around the apices of the teeth.* In such cases, failure may result even if the anesthetic solution is delivered intraosseously.

Perforator Breakage

In about 1% of cases, the metal perforator separates from the plastic shank during use.[67,103,139,320,356-358] The metal wire is easily removed with a hemostat. This separation usually occurs during a difficult perforation (e.g., dense cortical bone); the wire probably is heated excessively, causing the plastic hub to melt. No perforator breakage (metal perforator breaking into parts) has been reported in numerous controlled clinical studies.[67,103,139,320,356-358] However, excessive torquing of the

*References 67, 103, 139, 158, 320, 323, 355-358, and 402.

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perforator laterally by an inexperienced operator may result in breakage.

Optimal Location for Injection Site

Injection at a site distal to the tooth to be anesthetized produces the best anesthesia.* Maxillary and mandibular second molars are an exception to this rule. A mesial site should be selected for these teeth because of the increased thickness of the cortical plate in the mandible and the difficulty with perforation and needle placement at a distal site.

Onset of Anesthesia

The onset of anesthesia is immediate, which eliminates the waiting period.†

Site Selection: Attached Gingiva or Alveolar Mucosa

Both the Stabident and X-Tip IO systems instruct the user to locate the perforation site in attached gingiva. This gingival site allows the perforation to be made through a minimal thickness of cortical bone and generally is equidistant between adjacent root structures. However, because the guide sleeve remains in place with the X-Tip system, two studies have successfully used it in alveolar mucosa at a more apical location.[139,320] The X-Tip system has a definite clinical advantage over the Stabident system because the X-Tip perforation may be made at an apical location in unattached gingiva. If the Stabident system is used apically in alveolar mucosa, the hole created for delivering the anesthetic solution is almost impossible to find. The clinician may want to consider using the X-Tip system in an apical location in specific clinical situations. For example, when periodontal pocketing does not allow perforation into cancellous bone through the more coronal attached gingiva or when interproximal space is lacking (i.e., roots are too close together), the X-Tip system can be used to achieve pulpal anesthesia. If the Stabident system fails, the clinician may want to consider using the X-Tip apically to achieve pulpal anesthesia.

Care must be taken during IO injection to avoid insertion in areas of the roots and other critical structures, such as the mental foramen.

Injection Discomfort

When the IO injection is used as a primary injection, pain is experienced about a fourth of the time.[67,138,356-358] When the IO injection is used as a supplemental injection, fewer patients experience pain.[103,137,158,356,402]

Duration

With a primary IO injection, the duration of pulpal anesthesia declines steadily over an hour.[67,358] There is an even shorter duration with 3% mepivacaine compared with 2% lidocaine with 1:100,000 epinephrine.[358] With a supplemental IO injection of lidocaine after the inferior alveolar nerve block in patients without pain, the duration of pulpal anesthesia is very good for an hour.[103,158] A solution of 3% mepivacaine used as a supplemental IO injection results in a shorter anesthetic duration.[137]

Repeating the Intraosseous Injection

Repeating the IO injection, using 1.4 ml of 2% lidocaine with 1:100,000 epinephrine 30 minutes after the initial IO injection, provides an additional 15 to 20 minutes of pulpal anesthesia, similar to the duration of the initial IO injection.[198]

Use of Bupivacaine for Intraosseous Anesthesia

In an attempt to increase the duration of pulpal anesthesia with IO injections, some clinicians may consider using long-acting anesthetics. Bupivacaine (Marcaine) is a long-acting anesthetic, but only for IAN blocks. Long-acting anesthetics do not show an extended duration of anesthesia when injected by the IO or maxillary infiltration routes.[81,156,185,402] Bupivacaine has cardiotoxic effects[28] and for IO anesthesia basically is equivalent to 2% lidocaine with epinephrine in terms of efficacy, duration, and heart rate effects. Therefore, bupivacaine offers no advantage clinically and should not be used for IO anesthesia.

Systemic Effects of Intraosseous Injection

A transient increase in heart rate has been reported in 46% to 93% of cases involving Stabident and X-Tip IO injection of solutions containing epinephrine or levonordefrin.* Four clinical trials using techniques such as objective electrocardiographic recordings and pulse oximetry have shown that subjects experienced a transient tachycardia (mean increase of 12 to 32 beats/min) after Stabident IO injection of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine, 1.8 ml of 2% mepivacaine with 1:20,000 levonordefrin, or 1.8 ml of 1.5% etidocaine with 1:200,000 epinephrine.[57,158,359,402] Another clinical trial reported transient tachycardia after IO injection, but not with infiltration injection, of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine in the maxillary anterior region.[444] Generally, all these studies showed that the heart rate returned to baseline readings within 4 minutes in most patients. Therefore, injection of anesthetic solutions containing vasoconstrictors, using either the Stabident or X-Tip system, results in a transient tachycardia. No significant change in diastolic, systolic, or mean arterial blood pressure has been observed with IO injection of 2% lidocaine with 1:100,000 epinephrine.[57,359]

An IO injection of 1.4 ml of 2% lidocaine with 1:100,000 epinephrine with the CCLAD (CompuDent, Milestone Scientific, Livingston, New Jersey) at a fast rate resulted in a significantly higher heart rate compared to a slow solution deposition using either the CCLAD or a traditional syringe.[408]

Clinical Significance of Increased Heart Rate

Although the patient is likely to notice the transient tachycardia that occurs after Stabident or X-Tip IO injection of 2% lidocaine with 1:100,000 epinephrine, it generally is not clinically significant in healthy patients.[359] The clinical significance, cardiovascular effects, and contraindications to the use of vasoconstrictors in IO injections have been reviewed.[359]

Lack of Heart Rate Effect of 3% Mepivacaine in Intraosseous Anesthesia

No significant tachycardia occurs when 3% mepivacaine is used for IO anesthesia.[137,359] Clinicians should keep in mind that this anesthetic is an alternative for IO injection in

*References 67, 103, 139, 158, 356-358, and 402.
†References 67, 103, 139, 158, 356-358, and 402.

*References 67, 103, 139, 158, 320, 323, 356-358, and 402.

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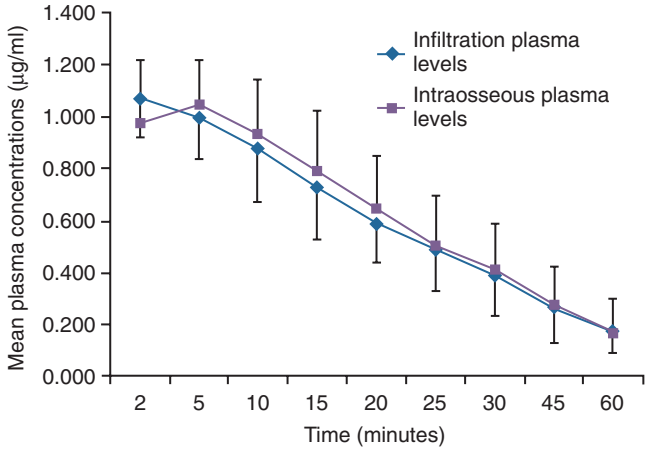

FIG. 4-23 Mean plasma concentrations of lidocaine for the intraosseous and infiltration injection techniques. No statistical differences were seen between the two techniques at any time period.

patients whose medical condition or drug therapy suggests caution in the use of solutions containing epinephrine or levonordefrin.[137,359]

Plasma Levels of Lidocaine After Intraosseous Injection

Some authors have cautioned that administration of an overly large volume of local anesthetic with an IO injection could lead to overdose reactions.[189] One experimental study using volunteers found that injection of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine produced venous plasma levels of lidocaine that were the same for maxillary anterior IO and infiltration injections (Fig. 4-23).[444] Although systemic concentrations of vasoconstrictors have a short-lived effect on the heart rate, the plasma concentration of lidocaine delivered with IO injection is no more than that delivered with infiltration, so the IO technique should not be considered an intravascular injection with regard to lidocaine. Also, if it were an intravascular injection, little or no anesthetic effect would be demonstrated; that is, all the local anesthetic solution would be carried into the vascular system, with none left for pulpal anesthesia. Obviously, clinical and experimental studies have demonstrated clinical anesthesia with IO techniques.* Therefore, the precautions for the maximum amount of lidocaine for an infiltration injection would seem to apply to an IO injection.

Postoperative Discomfort

When IO injections are given with the Stabident system, either as a primary or a supplemental technique, most patients report no or only mild pain; approximately 2% to 15% report moderate pain.[67,158,356-358] Less postoperative discomfort is reported for the Stabident IO injection than for IL injection.[379]

One study found that significantly more men experienced postoperative pain with the X-Tip system than with the Stabident system.[138] The authors interpreted these results as being due to the denser, more mineralized bone in the posterior mandible in men and to the fact that the diameter of the X-Tip perforating system is larger than that of the Stabident perforator, meaning the X-Tip system generates more frictional heat during perforation.

*References 67, 103, 139, 320, 323, and 355-358.

Postoperative Problems

With the Stabident system, fewer than 5% of patients develop swelling or exudate at the site of perforation.[67,158,356-358] The X-Tip system may show a higher incidence of postoperative swelling clinically.[138] With both systems, swelling or exudate (or both) may persist for weeks after the injection, but this resolves with time.[67,138,158,356-358] Slow healing of perforation sites may be the result of overheating of the bone caused by pressure during perforation.

With both the Stabident and X-Tip systems, approximately 4% to 15% of patients report that for a few days, the tooth "feels high" during chewing.[67,138,158,356-358] This feeling most likely is an increased awareness to biting that results from soreness in the area caused by damage from perforation or inflammation of the bone. The incidence of this feeling for the IO injection is lower than that reported for the IL injection (36% to 49%).[79,379]

Medical Contraindications

Patients taking antidepressants, nonselective beta-blocking agents, medicine for Parkinson disease, and cocaine should not receive IO injections of solutions containing epinephrine or levonordefrin.[353] Three percent mepivacaine plain is preferred.

Intraligamentary Anesthesia

The IL injection is another technique that is used if a conventional injection is unsuccessful.[397,428] The technique for IL injection of anesthesia is reviewed in a number of published papers and textbooks.

Success

For use as a primary injection, IL injections have a reported success rate of about 75% in mandibular and maxillary posterior teeth, with a duration of pulpal anesthesia of 10 to 15 minutes.[379,436] Success rates are low in anterior teeth.[280,379,436]

For use as a supplemental injection (standard techniques have failed to provide adequate anesthesia), good success rates are achieved, but the duration of pulpal anesthesia is approximately 23 minutes.[61]

Mechanism of Action

An IL injection forces anesthetic solutions through the cribriform plate into the marrow spaces around the tooth.[101,129,352,396,430] The primary route is *not* via the periodontal ligament, and unlike the intrapulpal (IP) injection,[38,423] the mechanism of action is not a pressure anesthesia.[105,296] The IL injection should be considered an intraosseous injection.

Back-Pressure

Studies have shown that the most important factor for anesthetic success with an IL injection is injection under *strong back-pressure*.[396,428] Pressure is necessary to force the solution into the marrow spaces.

Anesthetic Solutions

A vasoconstrictor significantly increases the efficacy of an IL injection.[155,213,228,278,379] Injection of a vasoconstrictor alone (1:100,000 epinephrine) does not produce pulpal anesthesia.[379] Anesthetic solutions with reduced vasoconstrictor concentrations (bupivacaine with 1:200,000 epinephrine) are not very effective with this technique.[155,200,213] Articaine is equivalent to lidocaine.[34]

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Amount of Solution Delivered

Usually about 0.2 ml of solution is deposited with each mesial and distal injection, using a traditional or pressure syringe. The exact amount is not always known because some of the anesthetic solution may escape from the sulcus during the injection.

Injection Discomfort

When the IL injection is used as a primary injection, needle insertion and injection have the potential to be painful about a third of the time.[296,379,436] The IL injection may be quite painful in maxillary anterior teeth[436] and should not be used in these teeth; infiltration is preferred. The potential for pain with the IL technique is greatly reduced when the injection is used as a supplemental injection after an inferior alveolar nerve block.[61]

Onset of Anesthesia

The onset of anesthesia is immediate with an IL injection,* which means no waiting period is required for the anesthesia to take effect. If anesthesia is still not adequate, reinjection is necessary.

Duration

Experimental studies with the EPT have shown that when the IL injection is given as a primary injection, the duration of profound pulpal anesthesia is approximately 10 to 20 minutes.[296,379,436] When the injection is used as a supplemental technique in asymptomatic teeth after an IAN block, the duration of pulpal anesthesia is approximately 23 minutes.[61]

New Technology for Intraligamentary Injections: CCLAD

A computer-assisted local anesthetic delivery system, introduced by Milestone Scientific, can be used to administer an IL injection. The CCLAD (also called *CompuDent* or *The Wand*) accommodates a standard local anesthetic cartridge that is linked by sterile microtubing to a disposable, penlike handpiece with a Luer-Lok needle (Fig. 4-24). The device is activated by a foot control, which automates the infusion of local anesthetic solution at a controlled rate. The fast rate delivers 1.4 ml of solution in 1 minute. The slow rate delivers 1.4 ml of solution in approximately 4 minutes, 45 seconds. The slow rate is used for the IL injection.

Anesthetic Success of the IL Injection Using the CCLAD

An experimental study compared the anesthetic efficacy of primary IL injection of 1.4 ml of 4% articaine with 1:100,000 epinephrine and 1.4 ml of 2% lidocaine with 1:100,000 epinephrine administered with a CCLAD in the mandibular first molar.[34] Successful pulpal anesthesia (two consecutive maximum EPT readings) was obtained 86% of the time with the articaine solution and 74% of the time with the lidocaine solution. No significant difference was seen between the articaine and lidocaine solutions. The duration of pulpal anesthesia ranged from 31 to 34 minutes, longer than the 10 minutes recorded in a similar study using a pressure syringe and 0.4 ml of a lidocaine solution.[436] Therefore, the CCLAD system offers the advantage of increasing the duration of pulpal anesthesia; however, the anesthesia slowly decreases over 60 minutes.

*References 79, 296, 379, 397, 428, and 436.

FIG. 4-24 Wand computer-assisted local anesthetic delivery unit (The Wand/CompuDent). The handpiece assembly and microtubing are also shown. (Courtesy Milestone Scientific, Livingston, NJ.)

Postoperative Discomfort

When the IL injection is used as a primary technique, postoperative pain occurs in a third to three quarters of patients, with a duration of 14 hours to 3 days.[79,296,379,397,428,436] There is no difference between articaine and lidocaine.[34] The discomfort is related to damage from needle insertion rather than to the pressure of depositing the solution.[79] About a fourth of patients report that their tooth feels "high" in occlusion.[379,436]

Systemic Effects

When a high-pressure syringe was used in dogs, IL injection of solutions containing epinephrine caused cardiovascular responses similar to those seen with IV injections.[395] Clinical studies using a high-pressure syringe in human beings found that IL injections of such solutions did not significantly change the heart rate, rhythm, or amplitude or the blood pressure.[53,317] These studies support the conclusion that IL injections do not cause significant changes in heart rate in human beings.

Other Factors

Different needle gauges (25, 27, or 30 gauge) are equally effective.[428] Special pressure syringes have been marketed but have not been proven more effective than a standard syringe.[379,428,436]

Safety of the Periodontium

Clinical and animal studies have demonstrated the relative safety of the IL injection technique.* Minor transient damage occurs only at the site of needle penetration, and the tissue subsequently undergoes repair. In rare cases, periodontal abscesses and deep pocket formation have occurred after IL injections.[61,436] A small clinical risk of periodontal abscess formation and bone loss exists with this technique, and although these effects are rare, the clinician should be aware of them. Localized areas of root resorption after IL injections have also been reported.[339,361]

*References 45, 128, 129, 135, 255, 296, 340, 379, 428, and 436.

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Safety of the Pulp

Clinical and animal studies have shown that IL injections have no permanent effect on the pulp.* However, IL injection of a solution with epinephrine produces a rapid, prolonged decrease in blood flow.[228] Some have suggested that using this injection technique during restorative procedures could result in accumulation of inflammatory mediators that would not be effectively removed because of the reduced blood flow.[228] This hypothesis was directly tested, and the IL injection of an anesthetic solution containing a vasoconstrictor in conjunction with a deep cavity preparation did *not* produce a more severe reaction than in controls (cavity preparation only).[344] Rather, the depth of the cavity preparation was the most important factor dictating pulpal responses. IL injections are therefore unlikely to cause pulpal necrosis.

Safety in Primary Teeth

One study has shown that IL injection of primary teeth may cause enamel hypoplasia of the developing permanent teeth.[45] The effect noted was not caused by the injection technique, but by the anesthetics used; that is, the cytotoxic anesthetic agents bound to the enamel matrix in the developing tooth germ. The same effect seemingly would be produced by an infiltration injection next to the developing tooth, so the recommendation that IL injections be used with great care on primary teeth close to developing permanent teeth[45] may not be correct.

Safety in Periodontally Involved Sites

IL injections have been shown to be safe in cases of mild to moderate gingival inflammation or incipient periodontitis.[76]

Avulsion

In a letter to the editor of the *Journal of the American Dental Association,* Nelson[311] reported on the avulsion of a tooth after IL injections. However, *no* clinical or experimental study has reported avulsion or loosening of teeth with this technique.[296,379,436] Avulsion should not be a concern with IL injections.

LOCAL ANESTHESIA FOR ENDODONTICS

For an emergency patient presenting with a tooth diagnosed with symptomatic irreversible pulpitis, anesthesia is administered with a standard IAN block. The patient reports classic signs of anesthesia (lip numbness and a dull feeling of the tooth or quadrant). After isolation, access preparation is begun. When the bur is in enamel, the patient feels nothing. Once the bur enters dentin (or possibly not until the pulp is exposed), the patient feels sharp pain. Obviously, pulpal anesthesia is not profound, and additional anesthetic is required. The following discussion outlines useful information for anesthetizing these endodontic patients.

Success of the Inferior Alveolar Nerve Block in Patients Presenting with Symptomatic Irreversible Pulpitis

Endodontic clinical studies of mandibular posterior teeth in patients with irreversible pulpitis have shown success occurs 14% to 57% of the time.* Articaine is not superior to lidocaine in this group of patients.[64,364,415] Nor is bupivacaine statistically superior to lidocaine.[376]

Some authors have suggested that a two-cartridge volume is better than a one-cartridge volume.[7] However, others did not find a statistically significant difference between one- and two-cartridge volumes.[336] In addition, a number of studies have used a two-cartridge volume, and success rates are similar to those for a one-cartridge volume.[122,253,330,390]

The epinephrine concentration in 2% lidocaine does not seem to make a difference in success in patients with symptomatic irreversible pulpitis.[8]

Success of Maxillary Molar Infiltration in Patients Presenting with Irreversible Pulpitis

Endodontic clinical studies of maxillary posterior buccal infiltrations in patients presenting with irreversible pulpitis have shown success occurs 54% to 88% of the time.[6,207,323] Although some authors have found a difference between articaine and lidocaine,[401] others have not.[27,207,370,384]

Asymptomatic Irreversible Pulpitis Versus Symptomatic Irreversible Pulpitis

Patients who have spontaneous pain and have moderate to severe pain at an emergency visit (symptomatic irreversible pulpitis) have less successful anesthesia after an inferior alveolar nerve block than patients who do not have spontaneous pain or only have pain when the tooth is stimulated (asymptomatic irreversible).[23] It is important to distinguish between these patients, because the success rates differ.

Supplemental Techniques

Infiltrations

Supplemental Buccal, Lingual, or Buccal Plus Lingual Infiltrations of Articaine

Although the infiltration of articaine is effective in restorative dentistry as a supplemental technique (after the inferior alveolar nerve block),[163] its use in endodontically involved teeth does not result in predictable pulpal anesthesia.[273,330,390] Success rates in studies have ranged from 38% to 84%, with most studies reporting success rates less than 60%.† A buccal infiltration of lidocaine is also not effective (65% success).[336]

Buccal infiltration alone, buccal plus lingual infiltrations alone (or after an inferior alveolar nerve block) do not result in complete pulpal anesthesia.‡ Therefore, IO and IL injections are the preferred techniques for supplemental anesthesia, and the IP injection is indicated when the IO or IL injection is not successful.

Intraosseous Anesthesia

Success in Symptomatic Irreversible Pulpitis

High success rates (around 90%) have been reported when the IO injection was used as a supplemental injection with

*References 251, 296, 342, 361, 379, and 436.

*References 3, 8, 26, 64, 68, 98, 131, 188, 197, 224, 226, 273, 295, 323, 330, 335, 355, 364, 391, 404, and 415.
†References 3, 4, 98, 273, 330, and 390.
‡References 3, 4, 10, 98, 109, and 346.

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irreversible pulpitis.[36,273,320,323,330] There is no difference between lidocaine and articaine.[36] Three percent mepivacaine has an 80% success rate, which increases to 98% with a second IO injection of 3% mepivacaine.[355]

An initial supplemental IO injection of 0.45 to 0.9 ml of 2% lidocaine with 1:100,000 epinephrine was successful in 79% of posterior mandibular teeth.[332] A second IO injection of the remaining cartridge increased the success rate to 91%. Therefore, initially giving a quarter to a half cartridge of 2% lidocaine with 1:100,000 epinephrine was less successful than initially giving a full cartridge.

Although some authors[338,354] have suggested an IO injection alone successfully anesthetizes patients presenting with irreversible pulpitis, it is very doubtful that this would be successful.[36,273,320,323,330]

Anesthetic Success in Symptomatic Teeth with Totally Necrotic Pulps and Radiolucent Areas

No study has investigated the use of IO anesthesia in patients with symptomatic teeth, totally necrotic pulps, and radiolucent areas. In a preliminary study performed at Ohio State, anesthetic solution deposition was very painful in these teeth, and the study had to be terminated.

Anesthetic Success in Partially Vital Teeth

The IO injection should work in teeth in which the chamber is necrotic, the canals are vital or partially vital, and widening of the periodontal ligament can be seen radiographically. A recent history of hot and cold sensitivity should differentiate this condition from that of a necrotic tooth experiencing an acute exacerbation (Phoenix abscess).

Injection Pain with Intraosseous Injection in Patients with Irreversible Pulpitis

Generally the Stabident system produces a very low incidence of moderate pain from perforation and solution deposition in asymptomatic patients.[67,138,356-358] A higher incidence was reported when the system was used to make IO injections in mandibular posterior teeth with symptomatic irreversible pulpitis. Up to 9% of patients reported moderate to severe pain after perforation, and 5% to 31% reported moderate to severe pain during deposition of the anesthetic solution.[323,355]

With the X-Tip system, 48% of patients with symptomatic irreversible pulpitis had moderate to severe pain with perforation, and 27% had moderate pain with solution deposition.[320] Patients with irreversible pulpitis may experience a transient but moderate to severe pain on perforation and solution deposition when either the Stabident or X-Tip system is used. The higher pain ratings, compared with those for asymptomatic teeth, are probably related to preexisting peripheral or central sensitization, which leads to increased pain responsiveness and preoperative anxiety.

Duration in Mandibular Teeth

In patients with irreversible pulpitis, supplemental IO injection using either the Stabident or X-Tip system provided anesthesia for the entire débridement appointment.[320,323,355]

Intraligamentary Anesthesia

Success

The overall success of supplemental IL injections in achieving pulpal anesthesia for endodontic procedures has been reported to be 50% to 96%.[68,260,397,428] As a primary anesthetic technique, IL injection produced a 50% to 79% success rate.[212,260] If a first IL injection fails, reinjection results in a success rate of 92%.[428] Similar results have been reported by other investigators.[397] IL injection is not successful in mandibular anterior teeth.[280,436] In addition, supplemental IL injections are not as successful as supplemental IO injections.[208,461]

Anesthetic Success of the IL Injection Using the CCLAD in Symptomatic Patients Presenting with Irreversible Pulpitis

A supplemental IL injection, administered with a CCLAD system in mandibular posterior teeth diagnosed with irreversible pulpitis after failure of an IAN injection, produced a 56% anesthesia success rate.[319] These results were somewhat disappointing because the CCLAD should have been capable of delivering approximately 1.4 ml of anesthetic solution with the IL injection by consistent maintenance of a precise flow rate.

Injection Discomfort

When the IL injection is given as a supplemental injection to anesthetize symptomatic vital teeth (i.e., in symptomatic irreversible pulpitis), the patient may have moderate pain.[100] Patients should be informed of this possibility.

Selective Anesthesia

Although some have reported that the IL injection can be used in the differential diagnosis of pulpally involved teeth,[256,386] experimental studies have shown that adjacent teeth may also become anesthetized with IL injection for a single tooth.[296,379,436] Therefore, the IL injection should *not* be used for differential diagnosis.

Duration

When the IL injection is used as a supplemental technique in endodontic therapy, the clinician must work fairly quickly and be prepared to reinject if profound anesthesia dissipates.

Intrapulpal Injection

In about 5% to 10% of mandibular posterior teeth with irreversible pulpitis, supplemental IO or IL injections, even when repeated, do not produce profound anesthesia; pain persists when the pulp is entered. This is an indication for an IP injection.

The major drawback of the IP injection technique is that the needle and injection are made directly into a vital and very sensitive pulp; this injection is often moderately to severely painful.[323] Because endodontic research offers more successful methods of supplemental anesthesia, the IP injection should be given only after all other supplemental techniques have failed. Another disadvantage of the technique is the possible duration of pulpal anesthesia (15 to 20 minutes). The bulk of the pulpal tissue must be removed quickly, at the correct working length, to prevent recurrence of pain during instrumentation. Yet another disadvantage is that the pulp obviously must be exposed to allow direct injection. Anesthetic problems frequently occur before exposure while the clinician is still working in dentin.*

*References 273, 319, 320, 323, 330, 355, and 390.

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The advantage of the IP injection is that it produces profound anesthesia if given under back-pressure.[38,423] The onset of anesthesia is immediate, and no special syringes or needles are required. The methods for this technique can be found in many excellent endodontic textbooks. As mentioned previously, strong back-pressure is a major factor in achieving successful IP anesthesia.[38,423] Depositing the anesthetic solution passively into the chamber is insufficient because the solution does not diffuse throughout the pulp.

MANAGEMENT OF ANESTHESIA IN ENDODONTIC CASES

Symptomatic Irreversible Pulpitis

When symptomatic irreversible pulpitis is a factor, the teeth that are most difficult to anesthetize are the mandibular molars, followed by the mandibular premolars, the maxillary molars and premolars, and the mandibular anterior teeth. The fewest problems arise in the maxillary anterior teeth.

In some teeth, irreversible pulpitis is the condition in the apical portion of the canals; the tissue in the chamber is necrotic and does not respond to pulp testing. The pulp chamber can be entered easily, but when attempts are made to place a file to length, severe pain results. IO or IL injections are helpful in these cases, and an IP injection may be used. However, irreversible pulpitis must be differentiated from a symptomatic necrotic tooth with a distinct apical abscess. In the latter condition, it is doubtful if IO or IL injections would be effective, and these injections may be quite painful, and bacteria may be forced into the periapical tissues by an IP injection.

Anesthetizing Mandibular Posterior Teeth

Until the 1980s, before supplemental IO and IL injections became popular, clinicians would administer the conventional IAN block and long buccal injection (molars). After signs of soft tissue anesthesia became evident, the pain abated and the patient relaxed. Local anesthesia produced the classic soft tissue signs and relieved the painful symptoms, but pain frequently resulted when the access opening was begun or the pulp was entered. Currently, this pain has been significantly reduced with supplemental techniques.

Timing of Supplemental IO Injection

Integration of the results of many clinical trials has changed the paradigm for anesthesia in patients with symptomatic irreversible pulpitis. Considering the high failure rate of the IAN block in patients with these teeth, now, after administering an anesthetic conventionally and observing signs of soft tissue anesthesia (which is required for a successful supplemental injection), the tooth is tested with cold refrigerant. If the result is negative, the clinician may proceed with access; if the result is positive, an IO injection is administered before the rubber dam is placed. The patient should be informed that the tooth is not as anesthetized as desired and that a little extra anesthetic will ensure his or her comfort. The clinician then explains that this extra anesthetic is placed next to the tooth and that the patient may feel some discomfort during the injection. Before the supplemental IO injection, a buccal infiltration of a cartridge of 4% articaine with 1:100,000 epinephrine is given over the tooth to reduce the pain of the IO injection. Because the supplemental IO injection is more successful than the supplemental IL injection for mandibular teeth with irreversible pulpitis,[208,461] the supplemental IO injection has replaced the IL injection. In addition, because negative testing with a cold refrigerant may not always indicate profound pulpal anesthesia, clinicians may proceed directly to an IO injection once soft tissue anesthesia is obtained. This technique significantly reduces pain and allows treatment to start sooner.

Many practitioners do not use this regimen because clinicians essentially do what they were taught in their initial clinical training, and sometimes change can be difficult. For example, a 1998 study in the *Journal of the American Medical Association* urged the use of anesthesia during circumcision[19]; nevertheless, up to 96% of baby boys don't receive anesthesia. Because many physicians were taught during their residencies not to administer anesthesia, changing their practice philosophy probably will be a slow process. This is a common problem in many health care disciplines, and it emphasizes the need for practitioners to stay current with recent advances.

Supplemental IO Injection: Use 3% Mepivacaine or 2% Lidocaine with 1:100,000 Epinephrine?

As a starting point, the authors recommend using 1.8 ml of 3% mepivacaine plain (e.g., 3% Carbocaine) for the IO injection. The basis for this recommendation is not the cardiovascular risks associated with anesthetic solutions containing a vasoconstrictor, but rather the results of clinical research, which indicate that 3% mepivacaine is reasonably effective and has no clinical side effect of increased heart rate.[137,359] A few patients may overreact to the heart rate increase produced by solutions containing epinephrine, making treatment difficult or time-consuming, because the patient must be calmed before treatment can begin. However, many clinicians also use 2% lidocaine with 1:100,000 epinephrine for IO anesthesia. Clinicians may want to experiment to determine which anesthetic solution (3% mepivacaine or 2% lidocaine with epinephrine) works best for them.

After anesthesia has been obtained, the rubber dam is placed, and the access preparation is slowly begun. The patient should be assured that the procedure will be stopped if he or she feels pain.

If the initial pain occurs in dentin, the rubber dam is removed and another cartridge of 3% mepivacaine is administered, which should be successful.[355] Again, the clinician should make sure that lip anesthesia has developed from the IAN block and that the anesthetic solution is deposited into medullary bone.

If the initial pain occurs when the pulp is entered, the rubber dam is removed and another cartridge of 3% mepivacaine is administered. If the patient still has pain, an IP injection is given. Some practitioners modify the access preparation under these conditions by creating a straight-line channel that directly opens the pulp tissue with little lateral access preparation (i.e., creating an entry hole the size of a #2 round bur). This provides quick access to the pulp, and the small size of the access facilitates IP injection with back-pressure if this technique is indicated. Usually, once the pulp is removed, further pain is minimal, owing to the longer duration of mandibular anesthesia.*

*References 60, 112, 277, 313, 332, and 424.

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Anesthetizing Mandibular Anterior Teeth

An inferior alveolar injection is given and the tooth is tested with cold refrigerant. If the result is negative, the clinician may proceed with access; if the result is positive, an IO injection is administered before access is begun (the IL injection does not work well in mandibular anterior teeth).[280,436] Before the supplemental IO injection, a labial infiltration of a cartridge of 4% articaine with 1:100,000 epinephrine is given over the tooth to reduce the pain of the IO injection. If pain is felt upon access, the IO injection is repeated. If this is unsuccessful, an IP injection is added.

Anesthetizing Maxillary Posterior Teeth

The initial anesthetic dose of 2% lidocaine with 1:100,000 epinephrine is doubled (to 3.6 ml) for the buccal infiltration.[287] A small amount of anesthetic solution is usually administered palatally for the rubber dam retainer. Although fewer anesthetic problems develop with the maxillary molars and premolars than with the mandibular posterior teeth, the clinician should be aware that they can occur.[6,323] The tooth is tested with cold refrigerant. If the result is negative, the clinician may proceed with access; if the result is positive, an IO or IL injection is administered before access is begun. If pain is felt during the access, the IO or IL injection is repeated. In some cases, an IP injection may be needed. Occasionally, pain is experienced in the palatal canal of molars. Infiltration over the palatal apex with 0.5 ml of anesthetic solution enhances pulpal anesthesia and may prove helpful.[157]

The duration of anesthesia in the maxilla is less than that in the mandible.[156,287,381] Therefore, if pain is experienced during instrumentation or obturation, additional primary and/or supplemental injections are necessary.

Anesthetizing Maxillary Anterior Teeth

Anesthetic is administered initially as a labial infiltration and occasionally as a palatal infiltration for the rubber dam retainer. The tooth is tested with cold refrigerant. If the result is negative, the clinician may proceed with access; if the result is positive, an IO injection is administered before access is begun. Rarely is an IO injection needed. The duration of anesthesia may be less than 1 hour. An additional infiltration injection may be necessary if the patient experiences pain during the later stages of instrumentation or obturation.[156,287,381]

Anesthetizing Symptomatic Teeth with Total Pulpal Necrosis and Periapical Abscess

Symptomatic teeth with total pulp necrosis and periapical radiolucencies are an indicator of pain in the periapical tissue. These teeth may be painful to manipulation and movement during treatment and require extra care. For mandibular teeth, the IAN block (and long buccal injection for molar teeth) is given in all situations. For maxillary teeth with no swelling, anesthetic is administered with conventional infiltration. If soft-tissue swelling is present (i.e., cellulitis or abscess), infiltration should be done on either side of the swelling, or a block could be administered (second division nerve block, PSA nerve block, or infraorbital nerve block). These provide some degree of bone and soft tissue anesthesia. After signs of anesthesia are observed, the rubber dam is placed, and the access is begun *slowly*. The pulp chamber usually can be entered without causing the patient discomfort if the tooth is not torqued excessively. Hand and rotary file shaping can be performed without causing much pain if instruments are used with gentleness and care.

Occasionally, the conventional injections do not provide profound anesthesia, particularly in the maxillary teeth. Although results from clinical trials are lacking for treatment of this region, anecdotal experience suggests that careful consideration of a supplemental injection, consisting of an additional palatal infiltration injection, may be helpful.[348]

In patients with severe preoperative pain, a long-acting anesthetic (e.g., bupivacaine) may help control postoperative pain in mandibular teeth but is not very successful in maxillary teeth.[81,156] However, the duration of analgesia in the mandible is usually not so long to preclude prescribing oral analgesics.[112]

Anesthetizing Asymptomatic Teeth with Total Pulp Necrosis and Periapical Radiolucencies

Asymptomatic teeth are the easiest to anesthetize. Although it may be tempting to proceed without anesthesia, vital sensitive tissue (ingrowth of periapical tissue into canal) may be encountered in the apical portion of canals or placement of files may cause pressure and extrusion of fluid periapically. The conventional injections are administered: IAN block and long buccal injection (for molars), infiltration injections for maxillary teeth. The clinician then proceeds with access and file placement. The patient usually is comfortable in such cases. Rarely, some discomfort may be felt during canal preparation, requiring an IO or IL injection. An IP injection should not be given, because bacteria and debris may be forced from the canal into the periapical tissue. Additional infiltration may be necessary in the maxilla if anesthesia begins to wear off.

Interim Treatment for Irreversible Pulpitis Using Pulpotomy

Patients often have to decide between extraction and endodontic treatment for irreversible pulpitis. Because of financial circumstances, patients may choose extraction if they are only presented with two choices. One study performed pulpotomies in patients with irreversible pulpitis and restored the teeth with intermediate restorative material (IRM) or IRM base/glass ionomer core.[274] They found pain was present in 10% of patients at 6 months and in 22% at 12 months. Although not ideal, the third option of pulpotomy and temporary restoration may allow the patient time to find the means to finance complete endodontic treatment.[87,218,274]

Pain Reduction in Irreversible Pulpitis When Endodontic Treatment Is Impossible

Endodontic débridement (pulpectomy or pulpotomy) most predictably relieves the pain of irreversible pulpitis.[240,329] When débridement is not possible, clinicians may prescribe strong analgesics and antibiotics in an attempt to relieve the pain. Unfortunately, the pain persists, and antibiotics have no effect on the pain of untreated irreversible pulpitis.[309] One clinical trial evaluated pain reduction in untreated irreversible pulpitis

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by giving an IO injection of Depo-Medrol (a long-acting methylprednisolone).[136] Clinically, the authors found that Depo-Medrol reduced the pain to manageable levels for up to 7 days before the patient received endodontic treatment, supporting this as a method for controlling a patient's pain until definitive endodontic treatment can be performed.

Oral Conscious Sedation with Triazolam (Halcion) and Alprazolam (Xanax)

Patients who are anxious have reduced pain tolerance[253] and may be harder to anesthetize. Oral conscious sedation is beneficial in reducing patient's anxiety. However, as shown by two studies, triazolam and alprazolam are *not* a way to reduce pain during endodontic or dental treatment.[226,253] Profound local anesthesia is still required to control the patient's pain.

Conscious Sedation with Nitrous Oxide

Nitrous oxide provides conscious sedation for apprehensive dental patients. Moreover, nitrous oxide provides a mild analgesic effect. A recent study showed administration of 30% to 50% nitrous oxide resulted in a statistically significant increase in the success of the IAN block in patients presenting with symptomatic irreversible pulpitis.[404]

Preemptive Nonsteroidal Antiinflammatory Drugs (NSAIDs)

Rationale for Preemptive NSAID Use in Patients Presenting with Irreversible Pulpitis

Administering NSAIDs 1 hour before anesthetic administration has been proposed to increase the success of the IAN block in patients presenting with irreversible pulpitis. The rationale is that prostaglandin induces sensitization of peripheral nociceptors.[174,327] Interventions with NSAIDs reduce the overall concentration of prostaglandins and lead to reduced activation of these receptors.[327] It has been shown that prostaglandin E_2 (PGE_2) is reduced in the pulps of patients presenting with irreversible pulpitis when NSAIDs were taken.[341]

Preemptive NSAID Use in Mandibular Posterior Teeth with Symptomatic Irreversible Pulpitis

An early study[291] of preemptive ibuprofen use found higher success rates of the IAN block using tooth sensitivity levels (TSLs). However, the assumption of pulpal anesthesia was not tested by accessing the endodontically involved teeth. Preemptive lornoxicam (8 mg) significantly improved the efficacy of the IAN block, but diclofenac (50 mg) did not.[347] The use of preemptive ketorolac had no significant effect on the success rate of the IAN block.[5,197,284] One study used preemptive oral ketamine and found that the requirements for the number of anesthetic cartridges were reduced when compared to a placebo.[216] However, the assumption of pulpal anesthesia was not tested by accessing the involved teeth.

A number of other studies[5,131,187,330,390] have demonstrated that preemptive administration of 600 mg or 800 mg ibuprofen, a combination of 800 mg ibuprofen and 1000 mg acetaminophen, or a combination of acetaminophen/hydrocodone did not statistically improve the success of the IAN block in patients presenting with symptomatic irreversible pulpitis.

However, preemptive NSAIDs have been shown to improve anesthesia in other studies.[316,333] In addition, a meta-analysis seemed to indicate an effect from preemptive NSAIDs in the treatment of irreversible pulpitis.[248] A simple explanation for these disparate findings is lacking, and more studies are needed on this topic.

Preemptive NSAID Use in Mandibular Posterior Teeth with Asymptomatic Irreversible Pulpitis

In one study[333] of patients presenting with no spontaneous pain at the endodontic appointment (asymptomatic irreversible pulpitis), preoperative administration of 600 mg ibuprofen or 75 mg indomethacin increased the success of the IAN block (78% and 62%, respectively) over a placebo (32%). However, as shown by Argueta-Figueroa et al.,[23] success rates are higher in patients with asymptomatic irreversible pulpitis. Another study of patients who presented with asymptomatic irreversible pulpitis[383] found that 400 mg of ibuprofen, 0.5 mg dexamethasone, and a placebo resulted in success rates of 25%, 38%, and 13%, respectively. However, the success rates with either ibuprofen or dexamethasone would not ensure profound pulpal anesthesia without supplemental injection of a local anesthetic.

Preemptive NSAID Use in Maxillary First Molars with Symptomatic Irreversible Pulpitis

One study[351] compared preemptive NSAIDs in maxillary teeth and found success rates of 93% for an 800-mg dose of ibuprofen, 90% for a 100-mg dose of aceclofenac, and 73% for 1000 mg of paracetamol. All these were significantly better than a placebo (26% success).

Patient Satisfaction with Painful Dental Procedures

Studies[131,253,404] have shown that patients were moderately or completely satisfied with endodontic treatment even though moderate to severe pain was experienced. Patient satisfaction may be related to the "bedside manner" of the dentist or to satisfaction with the emergency procedure, which they hoped would abate their discomfort. This is an important finding clinically because it helps explain why patients accept painful dental and medical procedures.

ANESTHESIA FOR SURGICAL PROCEDURES

Incision for Drainage

Clinicians should always attempt to achieve some degree of anesthesia before performing an incision and drainage procedure because patients tolerate such procedures better. In the mandible, a conventional IAN injection and long buccal injection are given. In the maxilla, 1.8 ml of 2% lidocaine with 1:100,000 epinephrine is infiltrated on both sides of the facial swelling. As an alternative, because we are mostly concerned with soft tissue anesthesia, a PSA or second division nerve block can be used for premolar and molar teeth and an infraorbital injection given for anterior teeth. For palatal swelling, 0.5 ml of 2% lidocaine with 1:100,000 epinephrine is infiltrated over the greater palatine foramen (molar and premolar teeth) or nasopalatine foramen (anterior teeth). These injections should not be given if swelling is present over the foramen; infiltration should be done on either side of the swelling. The Wand system reduces the pain of palatal injections. Generally, the computer-assisted injection system causes less

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pain than the conventional syringe technique for AMSA, palatal, and IAN block injections.*

Profound anesthesia is usually difficult to achieve in these cases,[29] and this must be explained to the patient. The swelling is not injected because traditionally it has been believed that injection directly into a swelling is contraindicated. Reasons for this included the possible spread of infection and the contention that the inflamed tissue's lower pH would make the anesthetic solution less effective. These ideas may have some merit, but swellings are not injected basically because it is *very* painful and relatively ineffective. An area of cellulitis has an increased blood supply, and an anesthetic injected into this area is largely carried away into the systemic circulation, which diminishes the anesthetic's local effectiveness. Also, edema and purulence may dilute the solution. (See Chapter 18 for additional information on incision and drainage procedures.)

Periapical Surgery

Most periapical surgery should be performed by an endodontist because he or she has received advanced training in the surgical procedures, periapical bone anatomy of the mandible and maxilla, use of magnification technologies, complex canal anatomy, and advanced microsurgical techniques for retrograde preparation and filling.

In the mandible, the inferior alveolar and long buccal injections are reasonably effective. Additional buccal infiltration injections in the vestibule may be useful to achieve vasoconstriction, particularly in the mandibular anterior region. In the maxilla, infiltration injections are generally effective; usually larger volumes are necessary to provide anesthesia over the surgical field. If the area of operation is inflamed, anesthesia may not be totally successful.

After the flap has been reflected, if anesthesia is inadequate, attempts to enhance or regain anesthesia (through additional infiltrations or injecting the sensitive area) are not particularly effective. The effectiveness of surgical anesthesia is decreased by half compared to anesthesia for nonsurgical procedures because when the clinician reflects a flap and opens into bone, the anesthetic solution is diluted by bleeding and removed by irrigation.[454]

Use of a long-acting anesthetic has been advocated for surgery.[84,371] In the mandible, this is reasonably effective. In the maxilla, long-acting agents have a shorter duration of anesthesia and decreased epinephrine concentrations, which result in more bleeding during surgery.[78,454]

Administration of a long-acting anesthetic after periapical surgery has been suggested.[262] However, postsurgical pain is usually not severe and can be managed by analgesics.[279]

SUMMARY AND FUTURE DIRECTIONS FOR EFFECTIVE ANESTHESIA

The emergence of evidence-based dentistry has rapidly drawn interest across all fields of dentistry. Fortunately, the large number of high-quality, randomized, controlled studies in anesthesia and analgesia provide a wealth of information supporting evidence-based recommendations. The practitioner must focus on high-quality research when making clinical decisions. This section of the chapter adopted the evidence-based approach in providing a comprehensive summary of local anesthetic clinical trials for the treatment of patients with endodontic pain.

The prudent practitioner knows that there is no "magic bullet" in pain control. Rather, the clinician must be knowledgeable about the advantages and disadvantages of several anesthetic-vasoconstrictor combinations and the various routes of injection. Clearly, judicious selection of local anesthetics, delivered through multiple sites of injection, is likely to provide predictable anesthesia with minimal exposure to side effects.

Research continues in the development of new or improved anesthetics. Investigations on extended-duration anesthetic formulations suggest that local anesthetics may prove useful for treating postprocedural pain.[141,462] In the future, clinicians may be able to provide anesthesia of a specific area for a day or maybe a number of days using liposomal-encapsulated anesthetic agents. The timing of anesthesia would be tailored by how the liposome is formulated to dissolve and the amount of drug in the liposome.

In addition, capsaicin and transient receptor potential vanilloid-1 (TRPV-1) agonists and antagonists may in the future be used in the clinical management of pain associated with inflammation.[144,232,237]

New modes of delivery of drugs are also being studied. Microneedles may be used in the future to deliver topical or local anesthetics across mucosal surfaces painlessly.[14,331,451]

II. ANALGESICS AND THERAPEUTIC RECOMMENDATIONS

NON-NARCOTIC ANALGESICS

Management of endodontic pain is multifactorial and directed at reducing the peripheral and central components of hyperalgesia through combined endodontic procedures and pharmacotherapy. A major class of drugs for managing endodontic pain is non-narcotic analgesics, which include both NSAIDs and acetaminophen. NSAIDs have been shown to be very effective in managing pain of inflammatory origin and, by virtue of their binding to plasma proteins, actually exhibit increased delivery to inflamed tissue via the extravasation of plasma proteins.[49,92,168,171] Although these drugs classically are thought to produce analgesia through peripheral mechanisms, the CNS is believed to be an additional site of action.[267,409] NSAIDs inhibit the synthesis of prostaglandins by blocking the enzyme cyclooxygenase (COX) which has two known isoforms, COX-1 and COX-2. Some researchers have proposed that a splice variant of COX-1 (i.e., COX-3) is expressed predominantly in the CNS and is the major site of action of acetaminophen.[59,231,325,380] However, recent studies[177] indicate that the antipyretic and analgesic effects of acetaminophen do not involve inhibition of COX-3; they are more likely exerted through effects of an active metabolite on CNS cannabinoid receptors,[18] and this metabolite appears to act by blocking a calcium channel ($Ca_V3.2$).[225]

Numerous NSAIDs are available for management of pain and inflammation (Table 4-4). Unfortunately, comparatively few studies (particularly for endodontic pain) directly compare one NSAID to another for analgesia and side-effect liability. The lack of comprehensive comparative studies in endodontic models means that only general recommendations can be made, and clinicians are encouraged to

*References 145, 181, 349, and 456.

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TABLE 4-4
Summary of Selected Non-Narcotic Analgesics

Analgesic	Trade Name	Dose Range (mg)	Daily Dose (mg)
Acetaminophen	Tylenol and others	325-650	4000
Aspirin	Many	325-1000	4000
Diclofenac potassium	Cataflam	50-100	150-200
Diflunisal	Dolobid	250-1000	1500
Etodolac	Lodine	200-400	1200
Fenoprofen	Nalfon	200	1200
Flurbiprofen	Ansaid	50-100	200-300
Ibuprofen	Motrin et al.	200-800	2400 (Rx)
Ketoprofen	Orudis	25-75	300 (Rx)
Ketorolac*	Toradol Sprix	30-60 (oral) 31.5	60 126 mg
Naproxen	Naprosyn	250-500	1500
Naproxen Na	Anaprox and others	220-550	1650 (Rx)

Modified from Cooper SA: Treating acute dental pain, *Postgrad Dent* 2:7, 1995.
Note: Cyclooxygenase-2 (COX-2) inhibitors are not included (see text).
Rx, Prescription strength.
*A new package insert for ketorolac tablets includes the instructions that the drug should be used only as a transition from injectable ketorolac and for no longer than 5 days. The package insert for intranasal ketorolac states that for patients 65 years of age or older, renally impaired patients, and patients weighing less than 50 kg, the dose should be limited to 15.75 mg, and the maximum daily dose should be limited to 63 mg.

familiarize themselves with several of these drugs. Ibuprofen generally is considered the prototype of contemporary NSAIDs and has a well-documented efficacy and safety profile.[89] Other NSAIDs may offer certain advantages over ibuprofen. For example, etodolac (i.e., Lodine) has reduced gastrointestinal (GI) irritation,[25] and ketoprofen (i.e., Orudis) has been shown in some studies to be somewhat more analgesic than ibuprofen.[73] An intranasal formulation of ketorolac tromethamine (Sprix, Regency Therapeutics, Shirley, New York) is now available and provides significant pain relief within 30 minutes of administration in patients with endodontic pain.[420] Recent studies suggest that in addition to inhibiting cyclooxygenase, ketorolac and diclofenac inhibit peripheral *N*-methyl-D-aspartate (NMDA) receptors, which may contribute to their analgesic effects.[54,96] The advantages of NSAIDs include their well-established analgesic efficacy for inflammatory pain. Many of the NSAIDs listed in Table 4-4 have been shown to be more effective than traditional acetaminophen and opioid combinations such as acetaminophen with codeine.[72,89,417]

A 2002 paper represents the first systematic review comparing all endodontic pain studies evaluating oral NSAIDs.[184] This study also provides a framework for other investigators interested in conducting systematic reviews for endodontic research. The authors concluded that NSAIDs combined with other drugs (e.g., flurbiprofen with tramadol[97]) or pretreatment and posttreatment application of NSAIDs provides effective pain control. Although relatively new to endodontic research, systematic reviews of analgesic drugs in inflammatory pain models have been conducted for several years. Table 4-5 lists the results of a large ongoing systematic review of the relative efficacy of analgesics in inflammatory pain conditions. Importantly, the data are generated based on postoperative patients having moderate to severe pain, and the number needed to treat (NNT) is based on the relative superiority of the analgesic over placebo for producing a 50% relief in pain. These data, therefore, constitute important, clinically relevant information for clinicians who want to compare the relative efficacy of posttreatment analgesics. Of course, other issues, such as the potential adverse effects of drugs and the patient's medical history, must be considered when a treatment plan for postendodontic pain is developed.

The introduction of selective inhibitors of COX-2 offered the potential for both analgesic and antiinflammatory benefits and reduced GI irritation.[91,227] Oral surgery pain studies evaluating COX-2 inhibitors have indicated that rofecoxib (i.e., Vioxx) has significant analgesic efficacy in this model.[106] In one study, a 50-mg dose of rofecoxib produced analgesia equivalent to 400 mg of ibuprofen, with the two drugs displaying similar times for onset of analgesia.[106] Expression of COX-2 is increased in inflamed human dental pulp,[310] and a COX-2 inhibitor (rofecoxib) is analgesic in patients with endodontic pain. However, most selective COX-2 inhibitors have been withdrawn from the market due to prothrombic adverse effects, and the only coxib still available, celecoxib, has not received approval by the U.S. Food and Drug Administration (FDA) for treatment of acute inflammatory pain. Concern has been raised that the COX-2 inhibitors may also display at least some GI irritation in patients with preexisting GI disease.[426]

Another major concern centers on the recognized prothrombic adverse effects of the COX-2 inhibitors. This debate originally started when patients randomized to 50 mg/day of rofecoxib in the VIGOR study had a fivefold increase in thromboembolic cardiovascular (CV) events compared to 1000 mg/day of naproxen.[40] The debate continued until the demonstration of an increased risk for prothrombic events after long-term administration of rofecoxib, which led to the withdrawal of this drug from the market in 2004.[110] Two meta-analyses have examined the CV safety of traditional NSAIDs and COX-2 inhibitors. Kearney et al.[217] conducted a meta-analysis of 138 randomized trials, and McGettigan and Henry[276] conducted a meta-analysis of 23 controlled observational studies.[217,276] Kearney et al.[217] estimated a relative risk for CV events associated with COX-2 to be 1.42 (95% CI, 1.64 to 2.91). Naproxen was found to have no significant adverse effects on the CV system in both meta-analyses. Diclofenac (Voltaren) is a relatively COX-2–selective drug and seems to have a degree of COX-2 selectivity similar to that of celecoxib. Diclofenac was associated with increased CV events. In the randomized trial analysis, there was an increase in CV risk with high-dose ibuprofen. Based on the available data, the FDA has requested that manufacturers of all prescription products containing nonselective NSAIDs revise their product labeling to include (1) a boxed warning regarding the potential serious adverse CV events and the serious, potentially life-threatening GI adverse events associated with the use of this class of drugs; (2) a contraindication to use in patients who have recently undergone coronary artery bypass surgery; and (3) a medication guide for patients regarding the potential for CV and GI adverse events associated with the use of this class of drugs.

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TABLE 4-5

Oxford League Table of Analgesic Efficacy*

Analgesic†	Number of Patients in Comparison	Percentage with at Least 50% Pain Relief	NNT	Lower Confidence Interval	Higher Confidence Interval
Ibuprofen (800 mg)	76	100	1.6	1.3	2.2
Ketorolac (60 mg) (IM)	116	56	1.8	1.5	2.3
Diclofenac (100 mg)	548	69	1.8	1.6	2.1
Oxycodone IR (5 mg) + acetaminophen (500 mg)	150	60	2.2	1.7	3.2
Diclofenac (50 mg)	738	63	2.3	2	2.7
Naproxen (440 mg)	257	50	2.3	2	2.9
Oxycodone IR (15 mg)	60	73	2.3	1.5	4.9
Ibuprofen (600 mg)	203	79	2.4	2	4.2
Ibuprofen (400 mg)	5456	55	2.5	2.4	2.7
Aspirin (1200 mg)	279	61	2.4	1.9	3.2
Oxycodone IR (10 mg)	315	66	2.6	2	3.5
Ketorolac (10 mg) + acetaminophen (650 mg)	790	50	2.6	2.3	3.1
Ibuprofen (200 mg)	3248	48	2.7	2.5	2.9
Naproxen (500/550)	784	52	2.7	2.3	3.3
Diclofenac (50 mg)	1296	57	2.7	2.4	3.1
Diclofenac (25 mg)	204	54	2.8	2.1	4.3
Demerol (100 mg) (IM)	364	54	2.9	2.3	3.9
Tramadol (150 mg)	561	48	2.9	2.4	3.6
Morphine (10 mg) (IM)	946	50	2.9	2.6	3.6
Naproxen (500/550 mg)	169	46	3	2.2	4.8
Naproxen (220/250 mg)	202	45	3.4	2.4	5.8
Ketorolac (30 mg) (IM)	359	53	3.4	2.5	4.9
Acetaminophen (500 mg)	561	61	3.5	2.2	13.3
Acetaminophen (600/650 mg) + codeine (60 mg)	1123	42	4.2	3.4	5.3
Acetaminophen (650 mg) + dextropropoxyphene (65 mg hydrochloride or 100 mg napsylate)	963	38	4.4	3.5	5.6
Aspirin (600/650 mg)	5061	38	4.4	4	4.9
Acetaminophen (600/650 mg)	1886	38	4.6	3.9	5.5
Tramadol (100 mg)	882	30	4.8	3.8	6.1
Tramadol (75 mg)	563	32	5.3	3.9	8.2
Aspirin (650 mg) + codeine (60 mg)	598	25	5.3	4.1	7.4
Oxycodone IR (5 mg) + acetaminophen (325 mg)	149	24	5.5	3.4	
Ketorolac (10 mg) (IM)	142	48	5.7	3	53
Acetaminophen (300 mg) + codeine (30 mg)	379	26	5.7	4	9.8
Tramadol (50 mg)	770	19	8.3	6	13
Codeine (60 mg)	1305	15	16.7	11	48
Placebo	>10,000	18	N/A	N/A	N/A

Modified from http://www.medicine.ox.ac.uk/bandolier/booth/painpag/acutrev/analgesics/lftab.html (Accessed August 10, 2015.)
IM, Intramuscular; NA, not applicable; NNT, number needed to treat.
*Analgesics are listed in descending order from most to least effective based on the number needed to treat (NNT). The NNT reflects the superiority of an analgesic over a placebo treatment; therefore an analgesic with a lower NNT has greater efficacy than an analgesic with a higher NNT. The NNT is calculated for the proportion of patients with at least 50% pain relief over 4 to 6 hours compared with placebo in randomized, double-blind, and single-dose studies in patients with moderate to severe pain. The 95% confidence interval contains the upper and lower estimates of the NNT with a 95% chance of accuracy.
†Drugs were given orally except where noted.

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The available data do not suggest an increased risk of serious CV events for the short-term, low-dose use of NSAIDs available over the counter, but the FDA has requested changes to the label to better inform consumers about the safe use of these products. Given this situation and reasonable alternative NSAIDs, we recommend against the use of COX-2 inhibitors for the treatment of patients with routine endodontic pain.

Limitations and Drug Interactions

Clinicians should educate themselves not only on the efficacy of the non-narcotic analgesics, but also on their limitations and interactions with other drugs.[52] For example, NSAIDs exhibit an analgesic ceiling that limits the maximal level of analgesia and induces side effects, including those affecting the GI system (3% to 11% incidence) and the CNS (1% to 9% incidence of dizziness and headache). NSAIDs are contraindicated in patients with ulcers and aspirin hypersensitivity.[63,65,125,396] They also are associated with severe GI complications, and the risk of adverse effects increases with an increasing lifetime-accumulated dose of these drugs and concurrent intake of aspirin, steroids, or coumadin.[24,93,242,442] A strategy for avoiding GI bleeding associated with NSAIDs is to use a proton pump inhibitor (PPI). A combination of a PPI (esomeprazole magnesium) with naproxen (Vimovo, Patheon/Astrozenica, Wilmington, Delaware) is now available. Although this is a cost-effective approach, it does not protect the lower GI tract.[400] Another approach is to use a histamine receptor H_2 antagonist in combination with an NSAID. The combination of famotidine (an H_2-receptor antagonist) and ibuprofen (Duexis, Horizon Pharma, Northbrook, Illinois) was recently approved by the FDA. The NSAIDs have been reported to interact with a number of other drugs (Table 4-6).

Acetaminophen and opioid combination drugs are alternatives for patients unable to take NSAIDs.[71] Further information is available on the pharmacology and adverse effects of this important class of drugs.[52,69,93,133,452] Other resources are also available for evaluation of drug interactions, including Internet drug search engines, such as *rxlist.com*, *Epocrates.com*, and *Endodontics.UTHSCSA.edu*.

TABLE 4-6

Summary of Selected Drug Interactions with Nonsteroidal Antiinflammatory Drugs (NSAIDs)

Drug	Possible Effect
Angiotensin-converting enzyme (ACE) inhibitors	Reduced antihypertensive effectiveness of captopril (and especially indomethacin)
Anticoagulants	Increase in prothrombin time or bleeding with anticoagulants (e.g., coumarins)
Beta-blockers	Reduced antihypertensive effects (e.g., propranolol, atenolol, pindolol)
Cyclosporine	Increased risk of nephrotoxicity
Digoxin	Increase in serum digoxin levels (especially ibuprofen, indomethacin)
Dipyridamole	Increased water retention (especially indomethacin)
Hydantoins	Increased serum levels of phenytoin
Lithium	Increased serum levels of lithium
Loop diuretics	Reduced effectiveness of loop diuretics (e.g., furosemide, bumetanide)
Methotrexate	Increased risk of toxicity (e.g., stomatitis, bone marrow suppression)
Penicillamine	Increased bioavailability (especially indomethacin)
Sympathomimetics	Increased blood pressure (especially indomethacin with phenylpropanolamine)
Thiazide diuretics	Reduced antihypertensive effectiveness

Data from Facts and Comparisons: *Drug facts and comparisons,* ed 54, St Louis, 2000, Facts and Comparisons; Gage T, Pickett F: *Mosby's dental drug reference,* ed 5, St Louis, 2000, Mosby; and Wynn R, Meiller T, Crossley H: *Drug information handbook for dentistry,* Hudson, Ohio, 2000, Lexi-Comp.

Acetaminophen

Acetaminophen (*N*-acetyl *p*-aminophenol) is one of the most commonly used analgesic and antipyretic drugs. Acetaminophen alone is not comparable to ibuprofen in relieving moderate to severe pain; however, a combination of ibuprofen and acetaminophen may provide greater pain relief than either drug alone. In a randomized, double-blind, placebo-controlled study, patients who had undergone pulpectomy were administered a single dose of either a combination of acetaminophen (1000 mg) and ibuprofen (600 mg) or ibuprofen (600 mg) alone. The combination of the two analgesics provided greater pain relief in the immediate postoperative period (8 hours) than ibuprofen (600 mg) alone.[285]

Acetaminophen is one of the most common drugs found in combination products for the relief of pain and symptoms of cold or flu. It is considered safe when taken at normal doses, but in higher doses, acetaminophen causes liver toxicity and is associated with nearly half of the cases of acute liver failure in the United States.[244] Most acetaminophen is conjugated in the liver to form inactive metabolites. A small portion is metabolized by the cytochrome P450 system to form *N*-acetyl-*p*-benzoquinone imine (NAPQI), which is very toxic but is generally detoxified by glutathione and converted into nontoxic compounds. Large doses of acetaminophen saturate the main route of metabolism, causing more acetaminophen to be converted to NAPQI. Liver injury occurs once glutathione becomes depleted and NAPQI is allowed to accumulate. To minimize this risk, it has been recommended that healthy adults not take more than 3 g (3000 mg) of acetaminophen in a 24-hour period (*www.tylenolprofessional.com/extra-strength-tylenol-dosage-faq.html*). The FDA mandated that by 2014, manufacturers of oral prescription acetaminophen combination medicines limit the maximum amount of acetaminophen to 325 mg per tablet. The FDA also requires a boxed warning to be added to the label of all oral prescription drug products that contain acetaminophen. (A boxed warning, the strongest warning that the FDA requires, indicates that the drug carries a significant risk of serious adverse effects).

OPIOID ANALGESICS

Opioids are potent analgesics and are often used in dentistry in combination with acetaminophen, aspirin, or ibuprofen. Most clinically available opioids activate mu opioid receptors at several important sites in the brain and on afferent neurons. Studies indicate that they activate peripheral opioid receptors in dental pulp and that IL injection of morphine significantly

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TABLE 4-7
Selected Opioid Combination Analgesic Drugs

Formulation	Trade Name*	Possible Rx
APAP 300 mg and codeine 30 mg	Tylenol with codeine no. 3	2 tabs q4h
APAP 500 mg and hydrocodone 5 mg	Vicodin, Lortab 5/500	1-2 tabs q6h
APAP 325 mg and oxycodone 5 mg	Percocet	1 tab q6h
APAP 500 mg and oxycodone 5 mg	Tylox	1 tab q6h
ASA 325 mg and codeine 30 mg	Empirin with codeine no. 3	2 tabs q4h
ASA 325 mg and oxycodone 5 mg	Percodan	1 tab q6h

APAP, Acetaminophen; *ASA*, aspirin; *Rx*, prescription.
*Several generics are available for most formulations.

TABLE 4-8
Analgesic Doses of Representative Opioids

Opioid	Dose Equivalent to Codeine 60 mg
Codeine	60 mg
Oxycodone	5-6 mg
Hydrocodone	10 mg
Dihydrocodeine	60 mg
Propoxyphene HCl	102 mg
Propoxyphene-N	146 mg
Meperidine	90 mg
Tramadol	50 mg

Modified from Troullos E, Freeman R, Dionne RA: The scientific basis for analgesic use in dentistry, *Anesth Prog* 33:123, 1986.
HCl, Hydrochloride; *N*, napsylate.

reduces pain in endodontic patients and in other inflammatory pain states.[94,111,167] Gender-dependent differences appear to exist in responsiveness to at least the kappa opioid agonists.[142] In a randomized, controlled clinical trial, women taking a combination of pentazocine and naloxone had significantly less postoperative endodontic pain than men taking the same medications.[374]

Although opioids are effective as analgesics for moderate to severe pain, their use is generally limited by their adverse side effects, which can include nausea, emesis, dizziness, drowsiness, and the potential for respiratory depression and constipation. Chronic use is associated with tolerance and dependence. Because the dosage is limited by side effects, these medications are almost always used in combination drugs to manage dental pain. A combination formulation is preferred because it permits a lower dose of the opioid, thereby reducing side effects (Table 4-7).

Codeine is often considered the prototype opioid for orally available combination drugs. Most studies have found that the 60-mg dose of codeine (the amount in two tablets of Tylenol with codeine no. 3) produces significantly more analgesia than placebo, although it often produces less analgesia than either aspirin 650 mg or acetaminophen 600 mg.[71,72,168] In general, patients taking only 30 mg of codeine report about as much analgesia as those taking a placebo.[31,417] Table 4-8 provides comparable doses of other opioids equivalent to 60 mg of codeine.

CORTICOSTEROIDS

Posttreatment pain or flare-up after endodontic treatment can be attributed to inflammation, infection, or both in the periradicular tissues. Establishing patency and subsequently débriding and shaping the root canal system may irritate the periradicular tissues and inadvertently introduce bacteria, bacterial products, necrotic pulp tissue, or caustic irrigating solution through apical foramina.

In response to this irritation, inflammatory mediators (e.g., prostaglandins [PGs], leukotrienes, bradykinin, platelet-activating factor, substance P) are released into the tissues surrounding the apical area of the tooth. As a result, pain fibers are directly stimulated or sensitized, and an increase in vascular dilation and permeability results in edema and increased interstitial tissue pressure.

Glucocorticosteroids are known to reduce the acute inflammatory response by suppressing vasodilation, migration of polymorphonuclear (PMN) leukocytes, and phagocytosis and by inhibiting formation of arachidonic acid from neutrophil and macrophage cell membrane phospholipids, thus blocking the COX and lipoxygenase pathways and respective synthesis of PGs and leukotrienes. It is not surprising that a number of investigations have evaluated the efficacy of corticosteroids (administered via either intracanal or systemic routes) in the prevention or control of postoperative endodontic pain or flare-ups.[268]

Intracanal Administration

Several studies have evaluated intracanal administration of steroids. In 50 consecutive patients requiring nonsurgical root canal treatment of vital teeth, one investigator alternately placed a dexamethasone solution or saline placebo as intracanal medicaments after the root canals had been cleaned and shaped.[307] Pretreatment pain ratings were collected, as were ratings at 24, 48, and 72 hours after treatment. Results indicated a significant reduction in pain at 24 hours but no significant difference at 48 and 72 hours. In a similar double-blind clinical trial, intracanal placement of a 2.5% steroid solution or saline placebo on completion of instrumentation resulted in a significant reduction of the incidence of postoperative pain in teeth in which the pulp was vital.[58] When the pulp was necrotic, however, there was no significant difference between the steroid and placebo in reducing postoperative discomfort.

Another study found no significant difference in the flare-up rate when either formocresol, Ledermix (a corticosteroid antibiotic paste), or calcium hydroxide was placed as an intracanal medicament in strict sequence, irrespective of the presence or absence of symptoms or radiographic signs of apical periodontitis.[416] However, a large-scale clinical trial of 223 patients reported significantly less posttreatment pain in patients after intracanal administration of Ledermix compared with either calcium hydroxide or no intracanal dressing.[107] Intracanal steroids appear to have significant effects for reducing postoperative pain.[365]

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Systemic Administration

Some studies have evaluated the systemic route of administration of corticosteroids on posttreatment pain or flare-ups. In one double-blind, randomized, placebo-controlled study, dexamethasone (4 mg/ml) or saline was injected intramuscularly at the conclusion of a single-visit endodontic appointment or at the first visit of a multivisit procedure.[269] The results indicated that the steroid significantly reduced the incidence and severity of pain at 4 hours when compared with the placebo. No difference in pain was seen at the 24- and 48-hour time points.

In a similar study, 106 patients with irreversible pulpitis and acute periradicular periodontitis were given an intraoral intramuscular injection of dexamethasone at different doses, either on completion of a single-visit endodontic treatment or after the first visit of a multivisit procedure.[250] Systemic administration of dexamethasone was shown to significantly reduce the severity of pain at 4 and 8 hours, with an optimum dose between 0.07 and 0.09 mg/kg. However, no significant reduction in the severity of pain was noted at 24, 48, and 72 hours, and no overall effect was seen on the incidence of pain. Another study compared the effect an IL injection of methylprednisolone, mepivacaine, or placebo in preventing posttreatment endodontic pain.[215] The results showed that methylprednisolone significantly reduced postoperative pain within a 24-hour follow-up period.

In a double-blind placebo-controlled study, patients with irreversible pulpitis were given 4 mg of dexamethasone or placebo by means of a supraperiosteal injection at the apex of the treated tooth after pulpectomy.[283] This is an injection technique that most clinicians would be familiar with (as opposed to intramuscular injection). Posttreatment pain was significantly reduced in the steroid group during the first 24 hours. There was no difference at 48 hours.

Yet another study evaluated the effect of IO injection of methylprednisolone or placebo in patients with irreversible pulpitis. Highly significant pain reduction in the steroid group was maintained for 7 days after a single injection.[143]

Animal studies have histologically evaluated the antiinflammatory effects of corticosteroids on inflamed periradicular tissues. In one study, after an acute inflammatory reaction was induced in the molar teeth of rats by overextending endodontic instruments, sterile saline or dexamethasone was infiltrated supraperiosteally into the buccal vestibule adjacent to the treated teeth. Dexamethasone significantly reduced the number of neutrophils present and thus had an antiinflammatory effect on the periradicular tissues of the treated teeth.[315]

Other studies of systemic administration have evaluated the effect of oral administration of corticosteroids on the incidence and severity of posttreatment endodontic pain. In a double-blind, controlled clinical trial, 50 patients randomly received either 0.75 mg of dexamethasone or a placebo tablet orally after initial endodontic treatment.[238] Oral dexamethasone significantly reduced posttreatment pain after 8 and 24 hours compared with placebo. A follow-up study evaluated the effect a larger oral dose of dexamethasone (i.e., 12 mg given every 4 hours) on the severity of posttreatment endodontic pain.[147] Results showed that dexamethasone was effective in reducing posttreatment endodontic pain up to 8 hours after the treatment was completed; no effect on the severity of pain was seen at 24 and 48 hours after treatment. In a double-blind study comparing etodolac and dexamethasone, the perioperative administration of oral dexamethasone was comparable to that of etodolac in reducing pain after endodontic surgery. In a randomized clinical trial,[252] 40 patients received a single dose of prednisolone (30 mg) or placebo 30 minutes before initiation of endodontic treatment. Prednisolone significantly reduced posttreatment pain at 6, 12, and 24 hours compared to placebo.[194]

Collectively, these studies on systemic steroid administration indicate that corticosteroids reduce the severity of posttreatment endodontic pain compared with placebo treatment. However, given the relative safety/efficacy relationship between steroids and NSAIDs, most investigators choose an NSAID as the drug of first choice for postoperative pain control.

ANTIBIOTICS

Because bacteria are involved in endodontic cases with apical periodontitis, the incidence of a posttreatment infection or flare-up is a concern to clinicians providing endodontic treatment. Prescribing an antibiotic to prevent such an occurrence might make sense, but use of antibiotics is controversial for several reasons.[119] First, overprescribing of antibiotics, especially when these drugs are not indicated, has led to increased bacterial resistance and patient sensitization. Second, antibiotics have been mistakenly prescribed for patients with severe pain who have a vital tooth (i.e., when bacteria are unlikely to be a causative factor in periradicular pain).[457] Third, even when bacteria are likely to be present, data from controlled clinical trials provide little or no support for the hypothesis that antibiotics reduce pain.[218]

A series of clinical studies evaluated the efficacy of prophylactically administered systemic antibiotics for preventing posttreatment endodontic flare-ups. Working on the premise that the incidence of infectious flare-ups after endodontic treatment is 15%, Morse et al.[305] randomly prescribed a prophylactic dose of either penicillin or erythromycin to patients after endodontic treatment of teeth with a diagnosis of necrotic pulp and chronic periradicular periodontitis (no placebo was used). The results showed that the overall incidence of flare-ups was 2.2%, with no difference between penicillin and erythromycin. Similar results were obtained in a study in which dental students (instead of private practitioners) provided the endodontic treatment.[1] Outcomes found a 2.6% incidence of flare-up, with no statistically significant differences between penicillin and erythromycin. However, neither the original nor the follow-up study was a randomized, placebo-controlled clinical trial. This observation appears to be highly significant for clinical recommendations because in general, randomized controlled studies fail to detect any analgesic benefit to antibiotics, whereas open-label or historical control studies often report profound effects.[119]

To determine whether the timing of administration of an antibiotic altered the occurrence of flare-ups and pain unassociated with flare-ups, researchers analyzed components of two separate prospective studies of patients undergoing endodontic treatment for teeth with necrotic pulps and chronic periradicular periodontitis. In the first study, prophylactic penicillin was provided; in the second study, patients were instructed to take penicillin (or erythromycin if they were allergic to penicillin) at the first sign of swelling.[304,305] The authors concluded that prophylactic administration of antibiotics is preferable to having the patient take antibiotics at the first sign of an infection.

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Another study of similar design compared the incidence of flare-up when a cephalosporin or erythromycin was given prophylactically.[306] When the data from previous studies were pooled and retrospectively compared, the authors concluded that prophylactically administered antibiotics, including cephalosporins, significantly reduced the incidence of flare-ups in endodontic cases involving necrotic pulp and chronic periradicular periodontitis. However, these studies have been questioned because of the lack of concurrent placebo-treated groups and the use of historical controls.

In a multicenter, two-part clinical study, 588 consecutive patients received one of nine medications or placebos and were monitored for 72 hours after treatment.[414] The results showed that ibuprofen, ketoprofen, erythromycin, penicillin, and penicillin plus methylprednisolone significantly reduced the severity of pain within the first 48 hours after treatment. The second part of the study then evaluated the incidence of posttreatment pain after obturation of the same teeth in the first phase of the study.[414] Although only 411 of the original 588 patients participated in this phase, they were randomly given the same medications or placebo after completion of the obturation appointment. The incidence of posttreatment pain was lower after obturation (5.83%) than after cleaning and shaping (21.76%) of the root canal system, and no significant difference was found in the effectiveness of the various medications and placebo in controlling posttreatment pain after obturation.

Walton and Chiappinelli[429] were concerned that previous studies were uncontrolled, retrospective, or carried out on different patient groups at different times and with different treatment modalities. They conducted a randomized, prospective, double-blind clinical trial to test the hypothesis that an antibiotic (e.g., penicillin) can prevent a posttreatment endodontic flare-up.[429] Eighty patients with teeth that had necrotic pulp and chronic periradicular periodontitis were randomly divided into three groups. In the first two groups, either penicillin or a placebo was administered 1 hour before and 6 hours after the individual appointments on a double-blind basis. Upon completion of the individual appointments, which included débridement, shaping, and possibly obturation of the root canal system, the patients completed questionnaires at 4, 8, 12, 24, and 48 hours. No significant difference was found among the three groups in the incidence of flare-ups, pain, or swelling. The authors concluded that the use of prophylactic penicillin offers no benefit for postoperative pain or flare-up, and prophylactic administration of penicillin should not be used routinely in patients undergoing endodontic treatment of necrotic teeth and chronic periradicular periodontitis.

In another randomized, prospective, placebo-controlled clinical study, a group of investigators examined whether supplemental penicillin reduced symptoms or shortened the course of recovery of emergency patients diagnosed with necrotic pulp and acute apical abscess.[120] Patients were randomly given penicillin, a placebo, or no medication. Using a visual analog scale, the subjects then evaluated their postoperative pain and swelling for up to 72 hours. No significant difference was found among the three groups. Recovery occurred as a result of endodontic treatment alone.

It is well recognized that antibiotics may be indicated for the management of infections of endodontic origin. However, a review of the available literature indicates that prophylactic use is contraindicated in immunocompetent patients with no systemic signs of infection and with swelling localized to the vestibule. Controlled clinical studies indicate that antibiotics offer little or no benefit for pain reduction under these circumstances, but they may be indicated for immunocompromised patients and for patients with systemic signs and symptoms of an infection or an infection that has spread into the fascial spaces of the head and neck.

PAIN MANAGEMENT STRATEGIES

When managing pain in an individual patient, the skilled clinician must customize the treatment plan, balancing the general principles of endodontics, mechanisms of hyperalgesia, and pain management strategies with the particular factors of the individual patient (e.g., medical history, concurrent medications).[171,219,220,369,393]

Effective management of endodontic pain starts with the "three Ds": *diagnosis*, *definitive* dental treatment, and *drugs* (Box 4-1). Comprehensive reviews on *diagnosis* and *definitive* dental treatment (e.g., incision and drainage, pulpectomy) are provided elsewhere in this text (Chapters 1, 6, 9, and 18). As described earlier in this chapter, the management of endodontic pain should focus on the removal of peripheral mechanisms of hyperalgesia and allodynia (see Box 4-1). This generally requires treatment that removes and reduces causative factors (e.g., bacterial and immunologic factors). For example, both pulpotomy and pulpectomy have been associated with substantial reduction in patient reports of pain compared to pretreatment pain levels.[97,173,274,337] However, pharmacotherapy often is required to reduce continued nociceptor input (e.g., NSAIDs, local anesthetics) and suppress central hyperalgesia (e.g., NSAIDs, opioids).

Pretreatment

Treatment with an NSAID before a procedure has been shown to have a significant benefit in many studies[89,191] but not all.[314] The rationale for pretreatment is to block the development of hyperalgesia by reducing the input from peripheral nociceptors. For patients who cannot take NSAIDs, pretreatment with acetaminophen also has been shown to reduce postoperative pain.[298] Patients can be pretreated 30 minutes before the procedure with either an NSAID (e.g., ibuprofen 400 mg or flurbiprofen 100 mg) or with acetaminophen 1000 mg.[97,191,298]

Long-Acting Local Anesthetics

A second pharmacologic approach for pain management is the use of long-acting local anesthetics, such as bupivacaine and ropivacaine. Clinical trials indicate that long-acting local anesthetics not only provide anesthesia during the procedure, but

BOX 4-1

Considerations for Effective "Three D" Pain Control

1. Diagnosis
2. Definitive dental treatment
3. Drugs
   - Pretreat with nonsteroidal antiinflammatory drugs (NSAIDs) or acetaminophen when appropriate.
   - Use long-acting local anesthetics when indicated.
   - Use a flexible prescription plan.
   - Prescribe "by the clock" rather than as needed.

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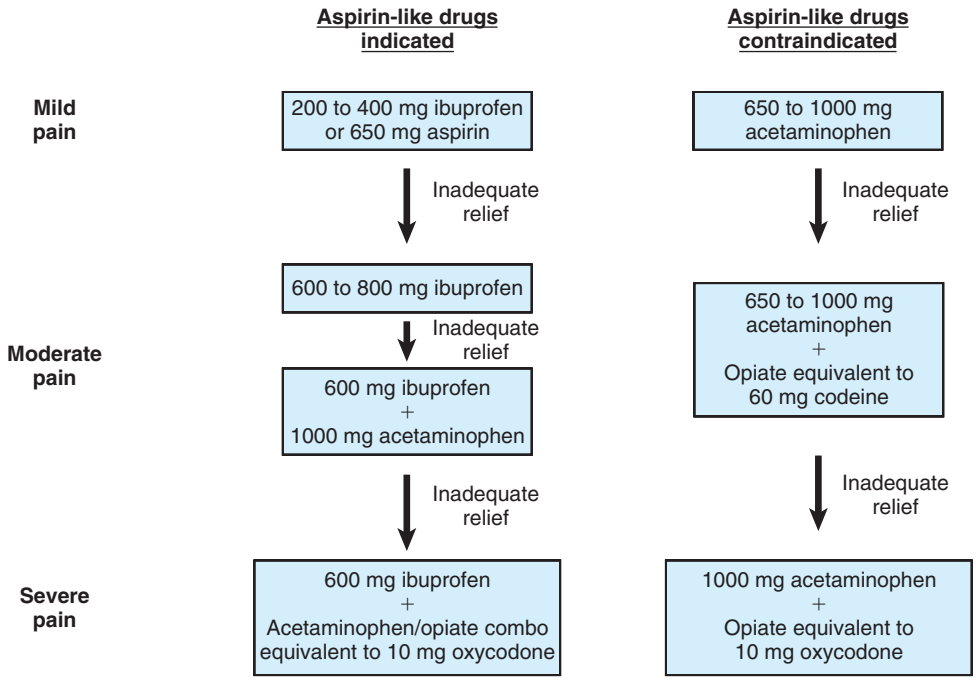

FIG 4-25 A flexible analgesic strategy.

also significantly delay the onset of posttreatment pain compared with local anesthetics that contain lidocaine.[77,89,152,153,196] Administration of long-acting local anesthetics by block injection has been shown to reduce posttreatment pain for 2 to 7 days after the oral procedure[152,153,196] because an afferent barrage of nociceptors can induce central hyperalgesia.[445-447] The analgesic benefit of long-acting local anesthetics is more prominent with block injections than infiltration injections, but the clinician should also be aware of adverse effects attributed to these agents.[28,297]

Flexible Plan

A third pharmacologic approach is the use of a flexible plan for prescribing analgesics (Fig. 4-25).* A flexible prescription plan serves to minimize both postoperative pain and side effects. Given these goals, the clinician's strategy is twofold: (1) to achieve a maximally effective dose of the non-narcotic analgesic (either an NSAID or acetaminophen for patients who cannot take NSAIDs); and (2) in the rare cases in which the patient still has moderate to severe pain, to consider adding drugs that increase the NSAID's analgesia. Because of its predictive value, the presence of preoperative pain or mechanical allodynia may be an indicator that such NSAID combinations should be considered.

Most, but not all, studies report that combining an NSAID with acetaminophen 1000 mg alone (i.e., no opioid) produces nearly twice the analgesic response as just the NSAID.[46,74,285,434] Administration of ibuprofen 600 mg with acetaminophen 1000 mg produced significant relief of posttreatment endodontic pain compared to ibuprofen alone or placebo (Fig. 4-26). However, in a recent study, the analgesic effect of a combination of ibuprofen 600 mg with acetaminophen 1000 mg did not differ from that of acetaminophen alone.[434] Studies have also shown that concurrent administration of an NSAID and an acetaminophen-opioid combination drug provided significantly greater analgesia than an NSAID alone.[46,403] The concurrent administration of acetaminophen and NSAIDs appears to be well tolerated, with no detectable increase in side effects or alterations in pharmacokinetics.[46,243,403,448]

In cases of moderate to severe pain, an NSAID may need to be administered with an opioid. Two general methods are used to combine NSAIDs and opioids to achieve the analgesic benefits of both. The first involves an alternating regimen of an NSAID followed by an acetaminophen-opioid combination.[17,72] Aspirin and opioid combinations are not used in this alternating schedule because of the potential for NSAID/aspirin interactions. The second method involves administration of a single drug consisting of an NSAID-opioid combination. For example, a tablet of Vicoprofen contains both ibuprofen (200 mg) and hydrocodone (7.5 mg). Postoperative pain studies have shown that this combination was about 80% more effective for analgesia than ibuprofen (200 mg) alone, with about the same incidence of side effects.[438] Other opioids can be added to an NSAID for increased analgesia. Ibuprofen 400 mg with a 10-mg oxycodone tablet produces significantly greater analgesia than ibuprofen alone.[90] A study on posttreatment endodontic pain demonstrated short-term benefits of the combination of flurbiprofen and tramadol.[97] Other NSAID and opioid combinations have also been evaluated.[93] However, the results of clinical trials on the use of NSAIDs alone and combined with acetaminophen (see Fig. 4-26) suggest that opioid combinations may be required only rarely.

Not all patients require concurrent use of NSAIDs with acetaminophen-opioid combinations or combinations of an NSAID and opioid. The basic premise of a flexible prescription plan is that the analgesic prescribed is matched to the patient's need. The major advantage of a flexible plan is that the clinician is prepared for those rare cases when additional pharmacotherapy is indicated to increase the efficacy of pain control. As mentioned, preoperative hyperalgesia may be an indicator

*References 17, 72, 166, 168, 171, 219, 221, and 417.

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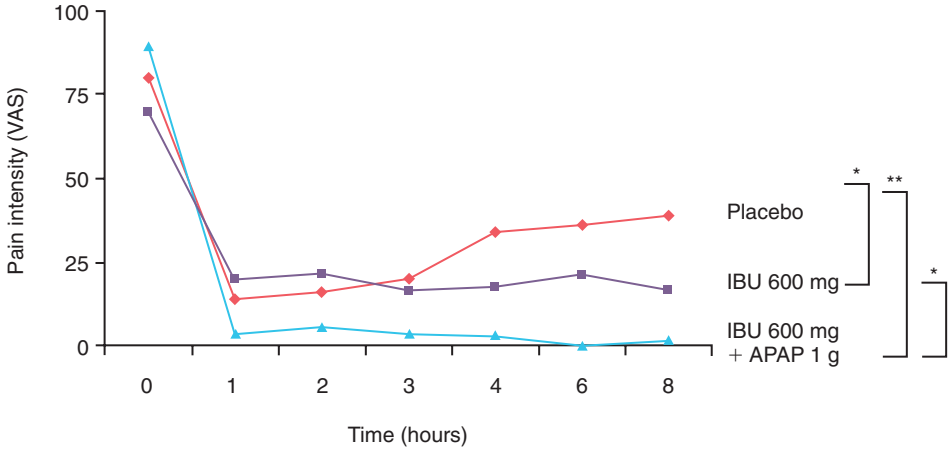

FIG. 4-26 Comparison of ibuprofen 600 mg with acetaminophen 1000 mg to ibuprofen alone or to placebo treatment in postendodontic pain patients. (From Menhinick KA, Gutmann JL, Regan JD, et al: The efficacy of pain control following nonsurgical root canal treatment using ibuprofen or a combination of ibuprofen and acetaminophen in a randomized, double-blind, placebo-controlled study, *Int Endod J* 37:531, 2004.)

for more comprehensive pharmacotherapy. When considering the combination of various analgesics, the clinician must make sure to use dosing regimens that do not exceed any of the drugs' maximum daily dosage.

FUTURE DIRECTIONS

COX-inhibiting nitric oxide (NO) donators (CINODs), a new class of NSAIDs, have an NO-donating moiety. These drugs, also known as NO-NSAIDs, were developed with the idea that the released NO would lead to improved vascular tone and mucosal blood flow, thus attenuating the adverse effects of NSAIDs on GI mucosa and blood pressure. Although none of the CINODS have been approved by the FDA yet, they may be available in the near future.

Current use of analgesics has been driven by clinical trials to arrive at doses that obtain the desired level of pain relief with acceptable side-effect profiles. After administration, drugs are absorbed and distributed to their site of action, where they interact with functional targets. They then undergo metabolism and eventual excretion. All of the steps along the way are influenced by a variety of environmental and genetic factors. The ability to predict how a patient's genome may affect the efficacy of a given analgesic drug is being discovered in the field of pain pharmacogenomics (for a review, see Rollason et al.[366]).

One example relevant to pain control in dentistry is the variable efficacy of codeine in a specific population. Many analgesic drugs are metabolized by the cytochrome P450 (CYP) family of hepatic enzymes, and the genes that encode for their biosynthesis have been identified. Codeine is a prodrug that is demethylated to produce morphine, which is responsible for its analgesic action. This demethylation is catalyzed by the enzyme cytochrome P402D6. It has been estimated that 6% to 7% of the Caucasian population has a nonfunctional CYP2D6 mutant allele, making them poor metabolizers of codeine to morphine.[88] These patients may be aware that codeine is ineffective in their systems from past experience and may request a different (usually more potent) form of narcotic. The clinician may suspect drug-seeking behavior, when in fact there is a biochemical reason for their request. Given the current state of clinical DNA analysis, it is not incomprehensible to envision a time when rapid (chairside) genomic evaluation will lead to specific recommendations for analgesic prescription.

SUMMARY

The information and recommendations provided in this chapter were selected to aid the clinician in the management of acute endodontic pain. Clinical judgment must also take into account other sources of information—the patient history, concurrent medications, the nature of the pain, and the overall treatment plan—when determining the best means of alleviating patient pain. An effective approach to managing endodontic pain demands integration of general principles of pain mechanisms and management with thorough, individualized clinical assessment.

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CHAPTER 5

Tooth Morphology, Isolation, and Access

JAMES L. GUTMANN | BING FAN

CHAPTER OUTLINE

Components of the Root Canal System
Root Canal Anatomy
Clinical Determination of the Root Canal Configuration
Objectives and Guidelines for Access Cavity Preparation
Objectives
Key Steps to Consider in Access Preparation
Visualization of the Likely Internal Anatomy
Evaluation of the Cementoenamel Junction and Occlusal Tooth Anatomy
Preparation of the Access Cavity Through the Lingual and Occlusal Surfaces
Removal of All Defective Restorations and Caries Before Entry into the Pulp Chamber
Removal of Unsupported Tooth Structure
Preparation of Access Cavity Walls that Do Not Restrict Straight- or Direct-Line Passage of Instruments to the Apical Foramen or Initial Canal Curvature
Inspection of the Pulp Chamber Walls and Floor
Tapering of Cavity Walls and Evaluation of Space Adequacy for a Coronal Seal
Mechanical Phases of Access Cavity Preparation
Magnification and Illumination
Handpieces
Burs
Endodontic Explorer and Spoon
Ultrasonic Unit and Tips
Access Cavity Preparations
Anterior Teeth
Posterior Teeth
Challenging Access Preparations
Teeth with Minimal or No Clinical Crown
Heavily Restored Teeth (Including Those with Full Coronal Coverage)
Access in Teeth with Calcified Canals
Crowded or Rotated Teeth
Errors in Access Cavity Preparation
Morphology of and Access Cavity Preparations for Individual Teeth
Maxillary Central Incisor
Maxillary Lateral Incisor
Maxillary Canine
Maxillary First Premolar
Maxillary Second Premolar
Maxillary First Molar
Maxillary Second Molar
Maxillary Third Molar
Mandibular Central and Lateral Incisors
Mandibular Canine
Mandibular First Premolar
Mandibular Second Premolar
Mandibular First Molar
Mandibular Second Molar
Mandibular Third Molar
Teeth with C-Shaped Root Canal Systems

The dental pulp presents with a variety of configurations and shapes throughout the dentition. Therefore, a thorough knowledge of tooth morphology, careful interpretation of radiographic documentation, and adequate access to and exploration of the pulpal space are prerequisites for all root canal procedures, whether nonsurgical or surgical.[182] To enhance this exploration and interpretation, magnification and illumination are indispensable. This chapter describes and illustrates tooth morphology and explains the techniques necessary to achieve unobstructed, direct access to the root canal system. The clinician is challenged to perform adequate enlarging, shaping, cleaning, disinfection, and obturation of the pulpal space to achieve predictable outcomes with root canal procedures. However, the optimal result is difficult to achieve if the access is not prepared properly. Therefore, knowledge of the complexity of the root canal system is essential to understand the principles and problems encountered in access preparation.

The complexity of the root canal system is best understood by the integration of formative knowledge of tooth anatomy and the interpretation of radiographic documentation. Careful evaluation of two or more periapical radiographs, exposed at different horizontal angulations of the x-ray cone, is essential.

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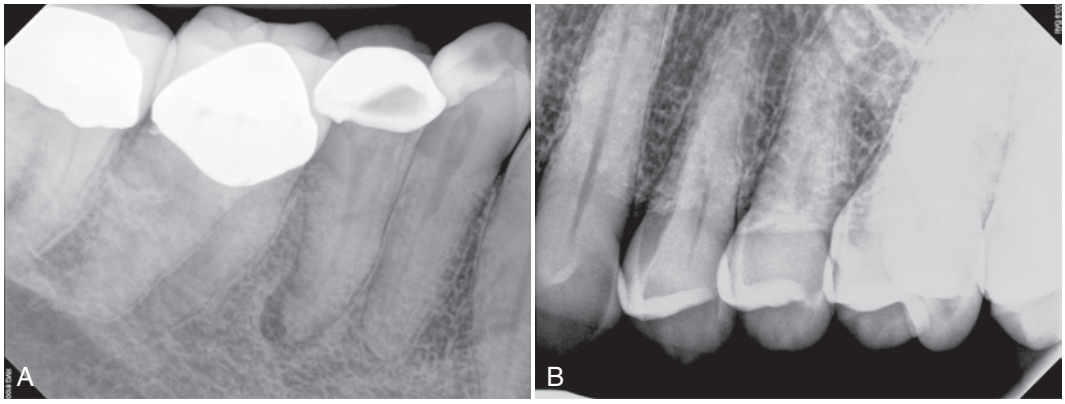

FIG. 5-1 A, Abrupt disappearance of the large canal in the mandibular first premolar indicates a canal bifurcation. B, The same is true for the maxillary first premolar.

These divergently exposed radiographs along with cone-beam computed tomography (CBCT) scans in some clinical situations (see Chapter 2) provide important information about root canal morphology. However, the inclination of the x-ray tube significantly influences the ability to detect variable root canal systems present in many teeth. For example, in premolars, if the horizontal angle is varied by either 20 or 40 degrees, the number of root canals seen in the maxillary first and second premolars and the mandibular first premolars coincides with the number of canals actually present.[134] However, in the mandibular second premolar, only the 40-degree horizontal angle correctly identifies the root canal morphology. The careful reading and interpretation of each radiograph before and during root canal procedures is necessary because many teeth present with unusual canal morphology. Unfortunately, the interpretation of traditional radiographs may not always result in the correct morphologic assessment, particularly when only a buccolingual view is taken. In one study, 790 extracted mandibular incisors and premolars were radiographed to assess the incidence of canal bifurcation in a root.[147] When the *fast break* guideline was used (i.e., interpreting a sudden disappearance or narrowing of a canal as a sign of canal division, such as bifurcation [Fig. 5-1]), the result was failure to identify one third of these divisions from a single radiographic view. Thus, evaluation of the root canal system is most accurate when the information from several radiographic views is integrated with a thorough clinical exploration of the interior and exterior of the tooth. Alternatively, the recent development of micro-computed tomography (μCT) scanning of teeth has greatly increased clinical assessment of these complexities and three-dimensional (3D) relationships found in root canal systems.

The main objectives of root canal procedures are adequate enlargement, shaping, cleaning, and disinfection of all pulpal spaces, along with obturation of these spaces with an acceptable filling material. At times a root canal or its elaborate, complex system may go undetected, which results in failure to achieve the stated objectives. Therefore, the use of a vast array of tools, in particular magnification and illumination, is essential to accomplish these objectives on a more predictable basis.

Initially important aids for determining pulp space morphology, in particular the pulp chamber and location of root canal orifices, include multiple pretreatment radiographs, CBCTs, examination of the pulp chamber floor with a sharp explorer, visual assessment of color changes in the dentin,

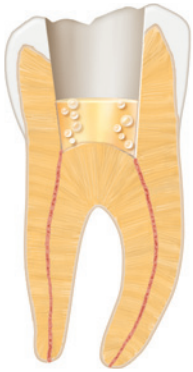

FIG. 5-2 Allowing sodium hypochlorite (NaOCl) to remain in the pulp chamber may help locate a calcified root canal orifice. Tiny bubbles may appear in the solution, indicating the position of the orifice. This is best observed through magnification.

troughing of anatomic grooves with ultrasonic tips, staining the chamber floor with 1% methylene blue dye, performing a sodium hypochlorite "champagne bubble" test (Fig. 5-2), visualizing the pulp chamber anatomy from established documents,[107] and root canal bleeding points. Sequential application of 17% aqueous ethylene diaminetetraacetic acid (EDTA) and 95% ethanol has been recommended for effective cleaning and drying of the pulp chamber floor before visual inspection.[207]

Specifically, the use of the dental operating microscope (DOM), which is intended to provide superior magnification, increased lighting, and enhanced visibility,[182] is recommended to determine the location of root canal orifices in the properly prepared coronal access (Fig. 5-3). Removal of dentin that may obscure the location of these orifices is also enhanced with better visualization with the DOM. The DOM also improves the identification of extra canals (e.g., the mesiopalatal canal found in many first and second maxillary molars) and has been shown to be superior to the use of the naked eye and magnifying loupes for this assessment.[14,187] Additional studies have noted that use of the DOM improves the detection of mesiopalatal canals to more than 90% in maxillary first molars and 60% in maxillary second molars.[108,207] These evaluative studies demonstrate that magnification and illumination greatly enhance the identification of the pulp chamber morphology and ultimately enable the clinician to achieve better outcomes within each of the stated objectives for root canal procedures.

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As with all advances in technology, however, there are varying points of concurrence and disagreement. For example, one group of investigators determined that dental loupes and the DOM were equally effective for locating mesiopalatal canals in maxillary molars,[29] whereas other studies determined that the DOM did not significantly enhance the ability to locate canals.[80] However, there appears to be consensus that the DOM enhances the locating of canals by magnifying and illuminating the grooves in the pulpal floor and by distinguishing the color differences in the dentin of the floor and walls.[41,107]

COMPONENTS OF THE ROOT CANAL SYSTEM

The dental pulp is often referred to as the root canal system, as opposed to a simple tube or circular space, due to its complexity (Fig. 5-4). The outline of this system generally corresponds to the external contour of the tooth. However, factors such as physiologic aging, pathosis, trauma, and occlusion all can modify its dimensions through the production of dentin or reparative (irregular secondary, irritation, and tertiary) dentin (Fig. 5-5).[199] The root canal system is divided into two portions: the pulp chamber, located in the anatomic crown of the tooth, and the pulp or root canal (or canals), found in the anatomic root. Other notable features are the pulp horns; accessory, lateral, and furcation canals; canal orifices; apical deltas; and apical foramina. The pulp horns are important because the pulp in them is often exposed by caries, trauma, or mechanical invasion, which usually necessitates vital pulp or root canal procedures. Also, the pulp horns undergo rapid mineralization, along with reduction of the size and shape of the pulp chamber because of the formation of reparative dentin over time.

The root canal begins as a funnel-shaped canal orifice, generally at or just apical to the cervical line, and ends at the apical foramen, which opens onto the root surface at or within 3 mm of the center of the root apex.* Nearly all root canals are curved, particularly in a faciolingual direction. These curves may pose problems during enlargement and shaping procedures because they are not evident on a standard two-dimensional (2D) radiograph. Angled views are often necessary to determine their presence, direction, and severity. A curvature may be a gradual curve of the entire canal or a sharp curvature near the apex. Double S-shaped canal curvatures also can occur. In most cases, the number of root canals corresponds to the number of roots; however, an oval root may have more than one canal.

Accessory canals are minute canals that extend in a horizontal, vertical, or lateral direction from the pulp space to the periodontium. In 74% of cases they are found in the apical third of the root, in 11% in the middle third, and in 15% in the cervical third.[223] Accessory canals contain connective tissue and vessels but do not supply the pulp with sufficient circulation to form a collateral source of blood flow. They are formed by the entrapment of periodontal vessels in Hertwig's epithelial root sheath during calcification.[46] They may play a significant role in the communication of disease processes, serving as avenues for the passage of irritants, primarily from the pulp to

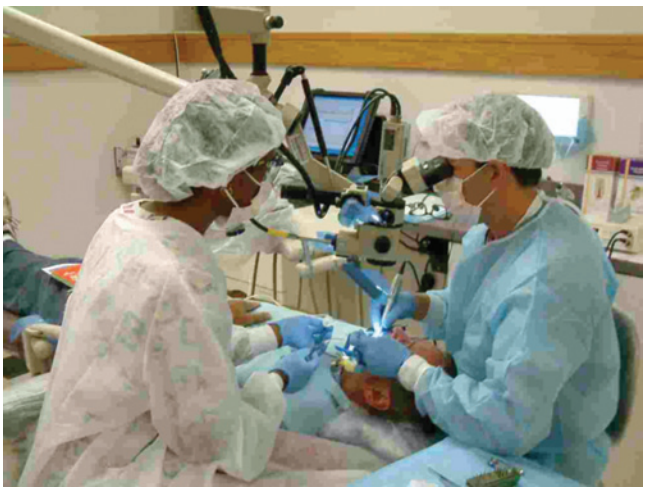

FIG. 5-3 The dental operating microscope (DOM) has vastly improved locating the position of the coronal canal anatomy.

*References 30, 81, 83, 170, 223, and 227.

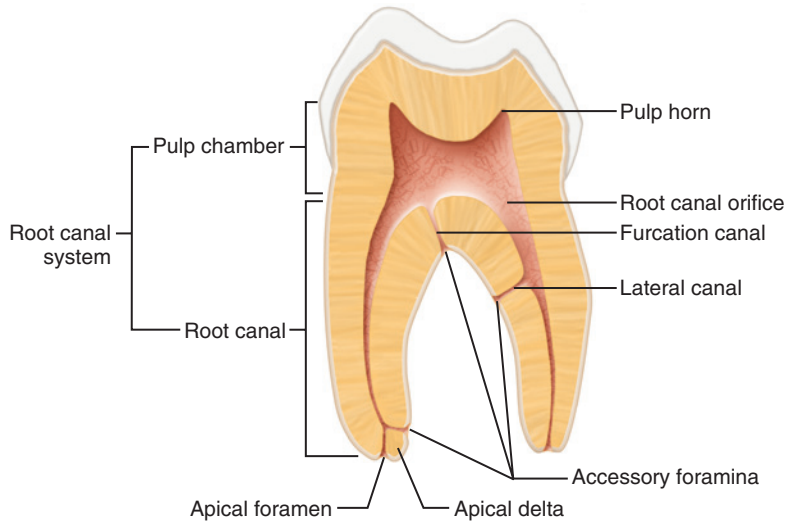

FIG. 5-4 Major anatomic components of the root canal system.

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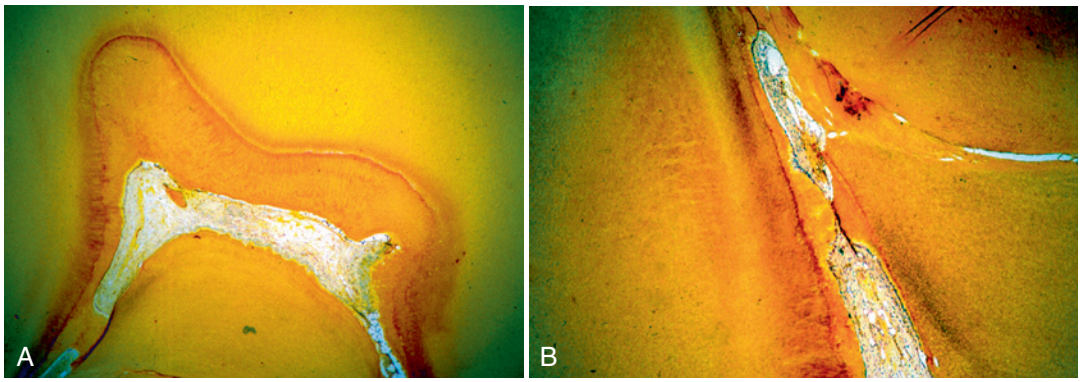

FIG. 5-5 A, Formation of reparative dentin and reduction of the pulp chamber space. B, Calcific narrowing and closing of a root canal as it leaves the pulp chamber. (From Gutmann JL, Lovdahl PE: *Problem solving in endodontics,* ed 5, St Louis, 2011, Elsevier.)

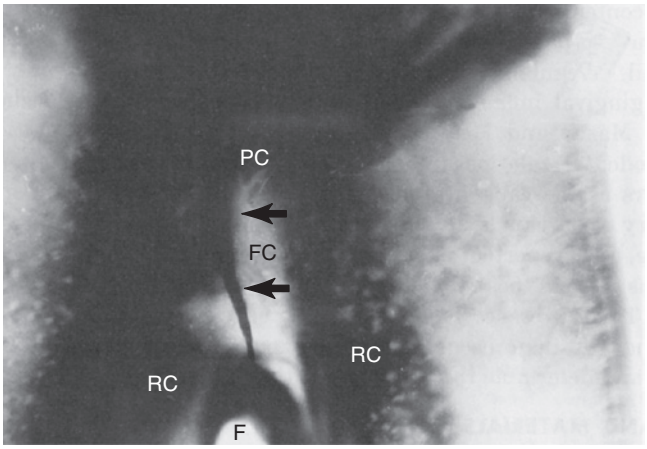

FIG. 5-6 Mandibular first molar showing a furcation canal *(FC, arrows). F,* Furcation; *PC,* pulp chamber floor; *RC,* root canal.

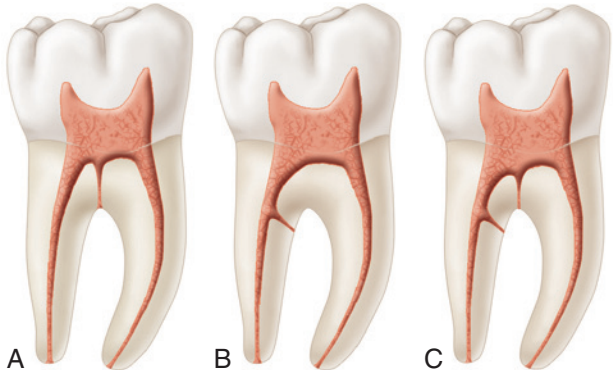

FIG. 5-7 Accessory canals occur in three distinct patterns in the mandibular first molars. A, In 13% a single furcation canal extends from the pulp chamber to the intraradicular region. B, In 23% a lateral canal extends from the coronal third of a major root canal to the furcation region (80% extend from the distal root canal). C, About 10% have both lateral and furcation canals.

the periodontium, although communication of inflammatory processes may occur from either tissue.

Accessory canals that are present in the bifurcation or trifurcation of multirooted teeth are referred to as *furcation canals* (Fig. 5-6).[223,226] These channels form as a result of the entrapment of periodontal vessels during the fusion of the diaphragm, which becomes the pulp chamber floor.[46] In mandibular molars these canals occur in three distinct patterns (Fig. 5-7). Tables 5-1 and 5-2 present the incidence of furcation canals for each tooth.

Based on scanning electron microscopy (SEM) studies, the diameter of furcation openings in mandibular molars varies from 4 to 720 m.[224] The number of furcation canals ranges from none to more than 20 per specimen. Foramina on both the pulp chamber floor and the furcation surface were found in 36% of maxillary first molars, 12% of maxillary second molars, 32% of mandibular first molars, and 24% of mandibular second molars (Fig. 5-8). Mandibular teeth have a higher incidence of foramina involving both the pulp chamber floor and the furcation surface (56%) than do maxillary teeth (48%). No relationship was found between the incidence of accessory foramina and the occurrence of pulp chamber calcification or the distance from the chamber floor to the furcation. Radiographs usually fail to show the presence of furcation and lateral canals in the coronal portion of these roots. In one study involving 200 permanent molars, the pulp chamber floor was stained with 0.5% basic fuscin dye. Patent furcation canals were detected in 24% of maxillary and mandibular first molars, 20% of mandibular second molars, and 16% of maxillary second molars.[87] Pulpal inflammation can communicate to the periodontium via these canals, and the result is furcation lesions in the absence of demonstrable periodontal disease. Likewise, the long-term presence of periodontal furcation lesions may influence the viability of the coronal or radicular pulp tissue when these aberrant channels are present.[84]

ROOT CANAL ANATOMY

The knowledge of common root canal morphology and its frequent variations is a basic requirement for success during root canal procedures. The significance of canal anatomy has been underscored by studies that demonstrated that the natural variations in canal geometry had a greater effect on the changes that occurred during enlargement and shaping than did the instrumentation techniques used to achieve these objectives.[167-169]

From the early works of Preiswerk[175] in 1912, Fasoli and Arlotta[67] in 1913, and Hess and Zurcher[90] in 1917, to more recent studies[33,82,100,190] demonstrating the anatomic complexities of the root canal system, data indicate that a root with a

TABLE 5-1

Morphology of the Maxillary Permanent Teeth*

Tooth	Root	Number of Teeth	Canals with Lateral Canals	Position of Lateral Canals				Transverse Anastomosis Between Canals	Position of Transverse Anastomosis			Position of Apical Foramen		Apical Deltas
				Cervical	Middle	Apical	Furcation		Cervical	Middle	Apical	Central	Lateral	
Central	—	100	24	1	6	93	—	—	—	—	—	12	88	1
Lateral	—	100	26	1	8	91	—	—	—	—	—	22	78	3
Canine	—	100	30	0	10	90	—	—	—	—	—	14	86	3
First premolar	—	400	49.5	4.7	10.3	74	11	34.2	16.4	58	25.6	12	88	3.2
Second premolar	—	200	59.5	4	16.2	78.2	1.6	30.8	18.8	50	31.2	22.2	77.8	15.1
First molar	MB	100	51	10.7	13.1	58.2	↑	52	10	75	15	24	76	8
	DB	100	36	10.1	12.3	59.6	18	0	0	0	0	19	81	2
	P	100	48	9.4	11.3	61.3	↓	0	0	0	0	18	82	4
Second molar	MB	100	50	10.1	14.1	65.8	↑	21	8	72	20	12	88	3
	DB	100	29	9.1	13.3	67.6	10	0	0	0	0	17	83	2
	P	100	42	8.7	11.2	70.1	↓	0	0	0	0	19	81	4

From Vertucci FJ: Root canal anatomy of the human permanent teeth, *Oral Surg Oral Med Oral Pathol* 58:589, 1984.
DP, Distobuccal; *MB*, mesiobuccal; *P*, palatal.
*Figures represent percentage of the total.

TABLE 5-2

Morphology of the Mandibular Permanent Teeth*

Tooth	Root	Number of Teeth	Canals with Lateral Canals	Position of Lateral Canals				Transverse Anastomosis Between Canals	Position of Transverse Anastomosis			Position of Apical Foramen		Apical Deltas
				Cervical	Middle	Apical	Furcation		Cervical	Middle	Apical	Central	Lateral	
Central	—	100	20	3	12	85	—	—	—	—	—	25	75	5
Lateral	—	100	18	2	15	83	—	—	—	—	—	20	80	6
Canine	—	100	30	4	16	80	—	—	—	—	—	30	70	8
First premolar	—	400	44.3	4.3	16.1	78.9	0.7	32.1	20.6	52.9	26.5	15	85	5.7
Second premolar	—	400	48.3	3.2	16.4	80.1	0.3	30	0	66.7	33.3	16.1	83.9	3.4
First molar	Mesial	100	45	10.4	12.2	54.4	↑ 23	63	12	75	13	22	78	10
	Distal	100	30	8.7	10.4	57.9	↓	55	10	72	18	20	80	14
Second molar	Mesial	100	49	10.1	13.1	65.8	↑ 11	31	10	77	13	19	81	6
	Distal	100	34	9.1	11.6	68.3	↓	16	11	74	15	21	79	7

From Vertucci FJ: Root canal anatomy of the human permanent teeth, *Oral Surg Oral Med Oral Pathol* 58:589, 1984.
*Figures represent percentage of the total.

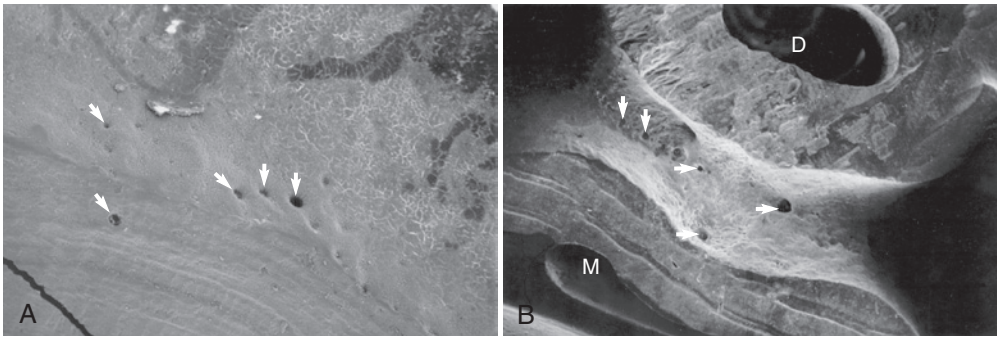

FIG. 5-8 A, Electron photomicrograph of the pulp chamber floor of a mandibular first molar. Multiple accessory foramina can be seen *(arrows),* ranging from 20 to 140 μm. (×20.) B, Electron photomicrograph of the furcation surface of a mandibular first molar. Multiple accessory foramina can be seen on the furcation surface. (×30.). *D,* Distal canal; *M,* mesial canals.

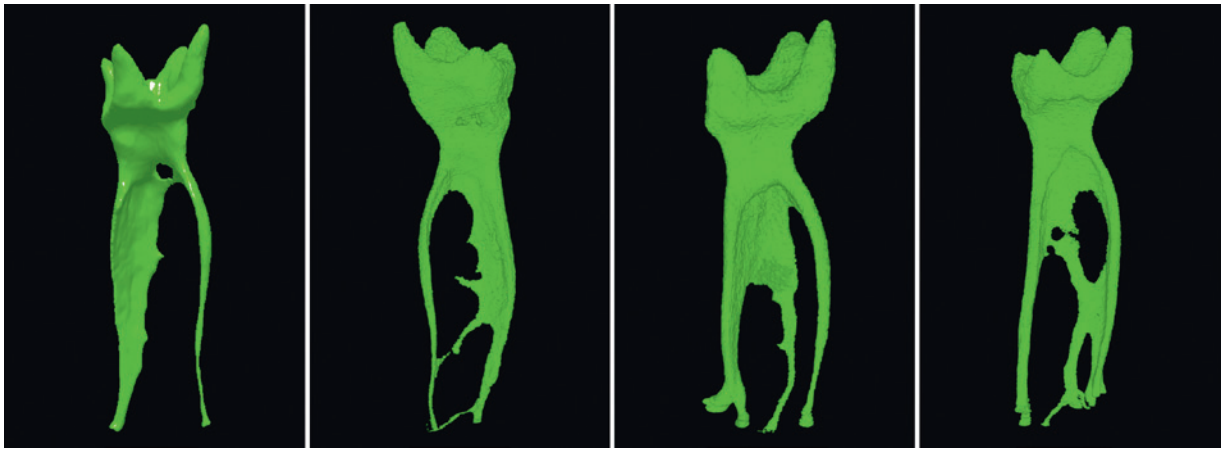

FIG. 5-9 Microcomputed tomography (μCT) scans show multiple canal configurations, depicting the complexity of the root canal system.

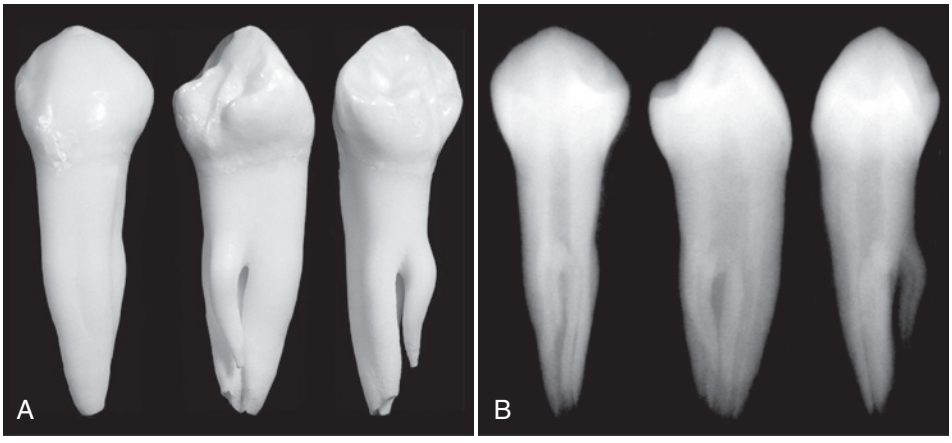

FIG. 5-10 A, Mandibular first premolar with three separate roots trifurcating in the midroot. B, Radiograph of the three views. Small canals diverging from the main canal create a configuration that is difficult to prepare and obturate biomechanically.

tapering canal and a single foramen is the exception rather than the rule. Investigators have shown multiple foramina, additional canals, fins, deltas, intercanal connections, loops, C-shaped canals, and furcation and lateral canals in most teeth (Fig. 5-9).* Consequently, complex anatomy must be considered the norm. The first premolar in Fig. 5-10, *A*, is a good example of complex anatomy. The extra root is not obvious in a pretreatment radiograph (Fig. 5-10, *B*). Figure 5-11 shows a cross-section of a similar tooth. This tooth has a fine, ribbon-shaped canal system instead of two distinct canals. Both these teeth present challenges for locating the canal and for achieving the previously stated objectives for root canal procedures.

Typically, root canals take variable pathways throughout, coursing from the orifice to the apex. The pulp canal system is complex, and canals may branch, divide, and rejoin. Weine[238]

*References 48, 53, 154, 170, 196, and 223.

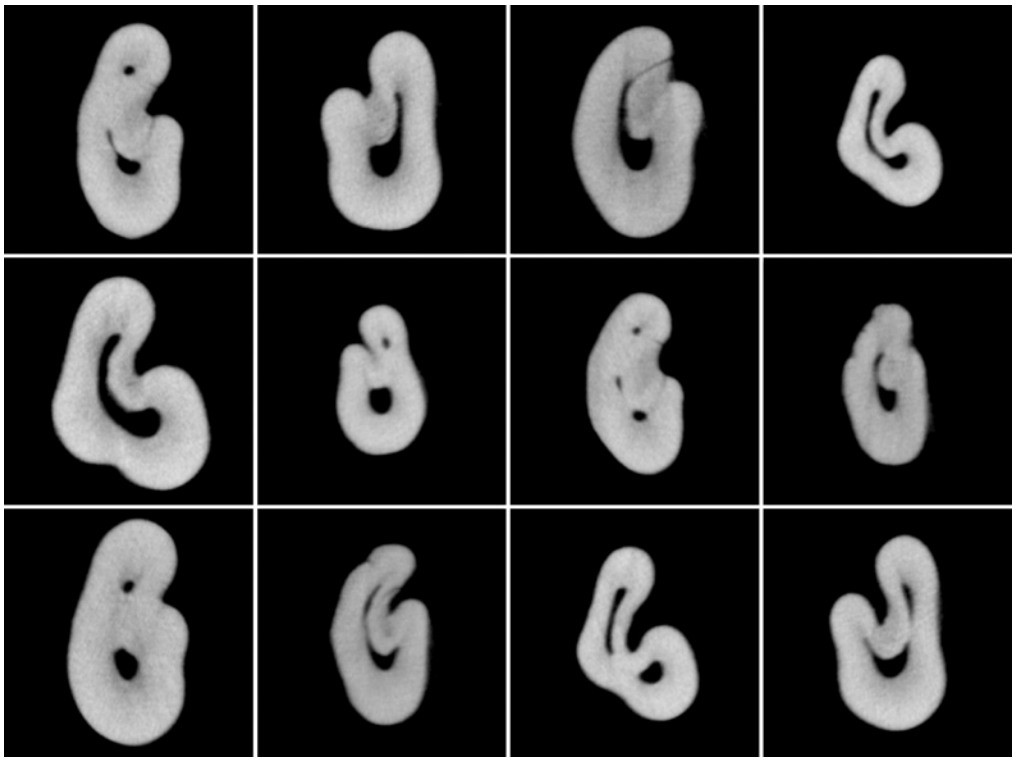

FIG. 5-11 Cross sections of teeth similar to those seen in Fig. 5-10 amplify the unique nature of the root canal system, particularly its ribbon and C-shaped configurations.

categorized the root canal systems in any root into four basic types. Other studies,[225] using cleared teeth in which the root canal systems had been stained with hematoxylin dye, found a much more complex canal system. In doing so the authors identified eight pulp space configurations, which can be briefly described as follows (Fig. 5-12):

Type I: A single canal extends from the pulp chamber to the apex (1).

Type II: Two separate canals leave the pulp chamber and join short of the apex to form one canal (2-1).

Type III: One canal leaves the pulp chamber and divides into two in the root; the two then merge to exit as one canal (1-2-1).

Type IV: Two separate, distinct canals extend from the pulp chamber to the apex (2).

Type V: One canal leaves the pulp chamber and divides short of the apex into two separate, distinct canals with separate apical foramina (1-2).

Type VI: Two separate canals leave the pulp chamber, merge in the body of the root, and separate short of the apex to exit as two distinct canals (2-1-2).

Type VII: One canal leaves the pulp chamber, divides and then rejoins in the body of the root, and finally separates into two distinct canals short of the apex (1-2-1-2).

Type VIII: Three separate, distinct canals extend from the pulp chamber to the apex (3).

The anatomic variations present in these teeth are listed in Tables 5-1 and 5-2. The only tooth that showed all eight possible configurations was the maxillary second premolar.

The percentages of human permanent teeth with these variable canal configurations are presented in Tables 5-3 and 5-4.

Similar observations have been described in large population studies with the exceptions that one canal was found in 23% of maxillary laterals, 55% of mesiobuccal roots of maxillary second molars, and 30% of distal roots of mandibular second molars.[33,242] Differences in these studies, from the types described by Weine, may be due to variations in the ethnic and racial populations studied. Other studies that were ethnic, racial, or gender-specific based have indicated wide variations in canal morphology, which sometimes appeared more often in specific teeth.* These authors concluded that gender, racial, and ethnic aspects should be considered in the pretreatment evaluation for root canal procedures (Fig. 5-13).

In addition to in vitro morphologic studies, a large number of case reports has described a variety of complex canal configurations (see Tables 5-8 to 5-27 online at the Expert Consult site). Reports of complex anatomy from both in vitro and in vivo investigations contribute to the adage that it is easier to recognize an anatomic feature if one is already prepared to see it. With the availability of CBCT, many more irregular and challenging root canal anatomies have been identified.[168,217]

One well-recognized ethnic variant is the higher incidence of single-rooted and C-shaped mandibular second molars in Native American and Asians populations (Fig. 5-14).[62,63,130,131] However, this is not always the case, as the occurrence of two canals in the mesiobuccal root of maxillary first molars in Japanese patients is similar to that described for other ethnic groups.[239] All this information makes it clear that the clinician is confronted daily with highly complex and variable root canal systems.

*References 82, 100, 190, 219, 229-231, and 236.

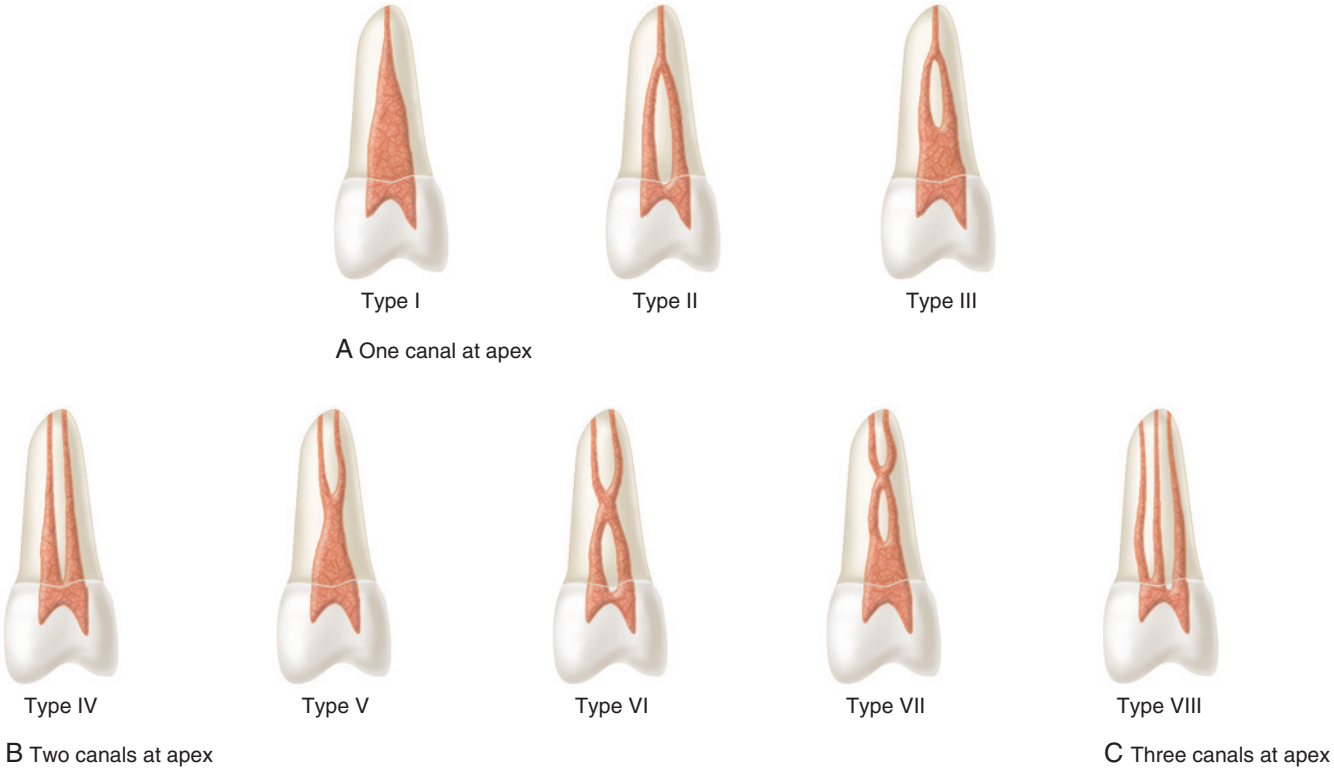

FIG. 5-12 Diagrammatic representation of canal configurations based on the work of Vertucci.

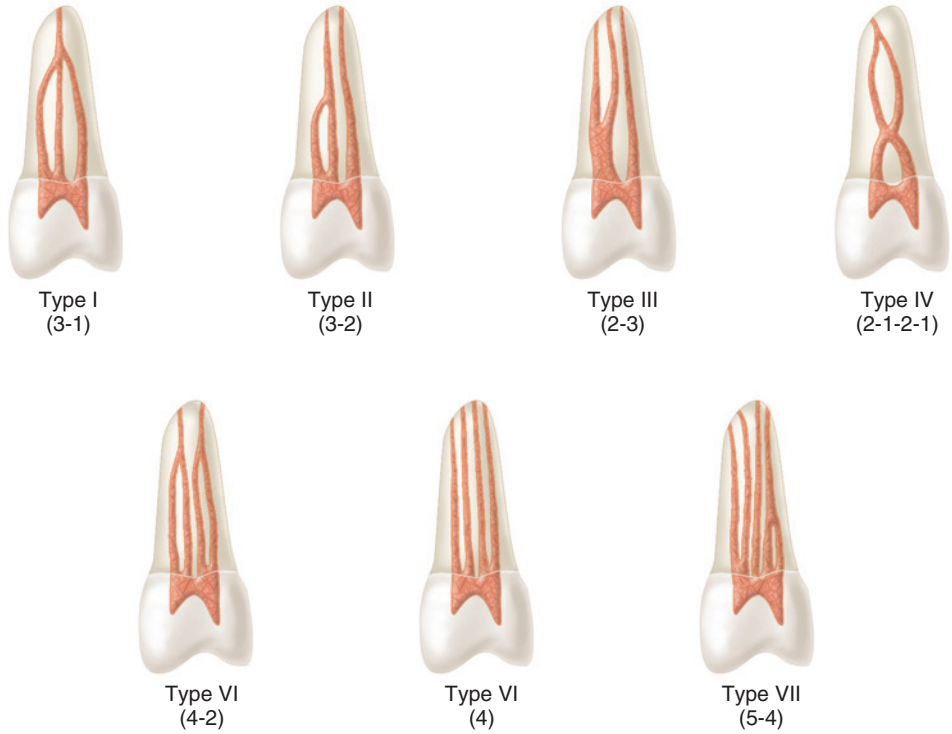

FIG. 5-13 Diagrammatic representation of supplemental canal configurations based on the work of Gulabivala and colleagues on a Burmese population.

TABLE 5-3

Classification and Percentage of Root Canals in the Maxillary Teeth

Tooth	Number of Teeth	Type I (1) Canals	Type II (2-1) Canals	Type III (1-2-1) Canals	Total with One Canal at Apex	Type IV (2) Canals	Type V (1-2) Canals	Type VI (2-1-2) Canals	Type VII (1-2-1-2) Canals	Total with Two Canals at Apex	Type VIII (3) Canals	Total with Three Canals at Apex
Maxillary central	100	100	0	0	100	0	0	0	0	0	0	0
Maxillary lateral	100	100	0	0	100	0	0	0	0	0	0	0
Maxillary canine	100	100	0	0	100	0	0	0	0	0	0	0
Maxillary first premolar	400	8	18	0	26	62	7	0	0	69	5	5
Maxillary second premolar	200	48	22	5	75	11	6	5	2	24	1	1
Maxillary First Molar												
Mesiobuccal	100	45	37	0	82	18	0	0	0	18	0	0
Distobuccal	100	100	0	0	100	0	0	0	0	0	0	0
Palatal	100	100	0	0	100	0	0	0	0	0	0	0
Maxillary Second Molar												
Mesiobuccal	100	71	17	0	88	12	0	0	0	12	0	0
Distobuccal	100	100	0	0	100	0	0	0	0	0	0	0
Palatal	100	100	0	0	100	0	0	0	0	0	0	0

From Vertucci FJ: Root canal anatomy of the human permanent teeth, *Oral Surg Oral Med Oral Pathol* 58:589, 1984.

TABLE 5-4

Classification and Percentage of Root Canals in the Mandibular Teeth

Tooth	Number of Teeth	Type I (1) Canals	Type II (2-1) Canals	Type III (1-2-1) Canals	Total with One Canal at Apex	Type IV (2) Canals	Type V (1-2) Canals	Type VI (2-1-2) Canals	Type VII (1-2-1-2) Canals	Total with Two Canals at Apex	Type VIII (3) Canals	Total with Three Canals at Apex
Mandibular central incisor	100	70	5	22	97	3	0	0	0	3	0	0
Mandibular lateral incisor	100	75	5	18	98	2	0	0	0	2	0	0
Mandibular canine	100	78	14	2	94	6	0	0	0	6	0	0
Mandibular first premolar	400	70	0	4	74	1.5	24	0	0	25.5	0.5	0.5
Mandibular second premolar	400	97.5	0	0	97.5	0	2.5	0	0	2.5	0	0
Mandibular first molar												
Mesial	100	12	28	0	40	43	8	10	0	59	1	1
Distal	100	70	15	0	85	5	8	2	0	15	0	0
Mandibular second molar												
Mesial	100	27	38	0	65	26	9	0	0	35	0	0
Distal	100	92	3	0	95	4	1	0	0	5	0	0

From Vertucci FJ: Root canal anatomy of the human permanent teeth, *Oral Surg Oral Med Oral Pathol* 58:589, 1984.

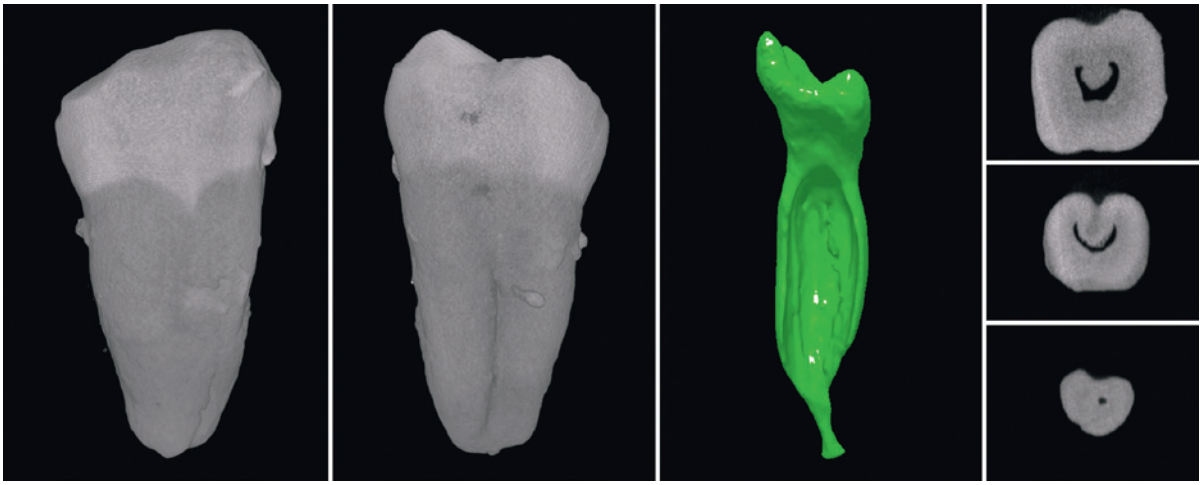

FIG. 5-14 Common variant of a C-shaped canal anatomy found in Native Americans and Asian populations.

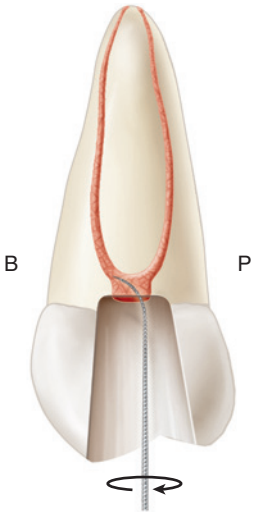

FIG. 5-15 An oval orifice must be explored with apically curved small instruments. When trying to locate the buccal canal, the clinician should place the file tip in the orifice with the tip curved to the buccal side. To explore for the palatal canal, a curved file tip is placed toward the palate. *B,* Buccal; *P,* palatal.

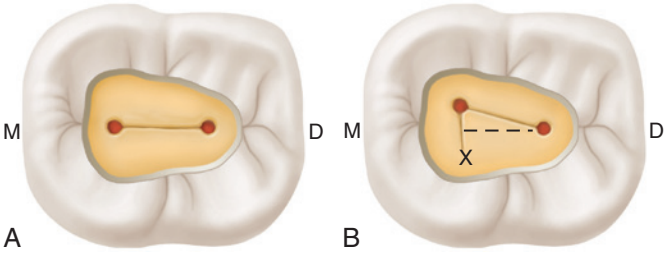

FIG. 5-16 A, In a mandibular second molar with two canals, both orifices are in the mesiodistal midline. B, If two orifices are not directly in the mesiodistal midline, a search should be made for another canal on the opposite side in the area of "X." *D,* Distal; *M,* mesial.

Clinical Determination of the Root Canal Configuration

Coronal Considerations

Examination of the pulp chamber floor can reveal clues to the location of orifices and to the type of canal system present. It is important to note that if only one canal is present, it usually is located in the center of the access preparation. All such orifices, particularly if oval shaped, should be explored thoroughly with small, stiff K-files that have a smooth to sharp bend in the apical 1 to 2 mm (C files and C+ files, Dentsply Tulsa Dental Specialties, Tulsa, Oklahoma, and Dentsply Maillefer, Ballaigues, Switzerland, respectively). If only one orifice is found and it is not in the center of the root, another orifice probably exists, and the clinician should search for it on the opposite side (Fig. 5-15). The relationship of the two orifices to each other is also significant (Fig. 5-16). The closer they are, the greater the chance the two canals join at some point in the body of the root. As the distance between orifices in a root increases, the greater is the chance the canals will remain separate. The more separation between orifices, the less the degree of canal curvature.[40] The direction a file takes when introduced into an orifice is also important. If the first file inserted into the distal canal of a mandibular molar points either in a buccal or lingual direction, a second canal is present. If two canals are present, they will be smaller than a single canal. (See μCTs and cross sections of all tooth groups later in this chapter. In addition, rotational videos of these groups can be found online as Videos 5-1 through 5-16 at the Expert Consult site.)

Midroot Considerations

As the canal leaves the coronal portion of the root and blends into the midroot portion, many changes can occur, including fins, webs, culs-de-sac, and isthmuses (also called *anastomoses*). These structures are narrow, ribbon-shaped communications between two root canals that contain pulp or pulpally derived tissue, or they may represent a communication between two canals that split in the midroot portion of the canal. These structures contain variable amounts of tissue, and when the pulp is infected, they often contain bacteria and their byproducts. In one study, isthmuses in the mesiobuccal root of maxillary first molars were found most often 3 to 5 mm from the root apex.[244] A complete or partial isthmus was found at the 4-mm level 100% of the time. In another study, partial isthmuses were found more often than complete ones.[213]

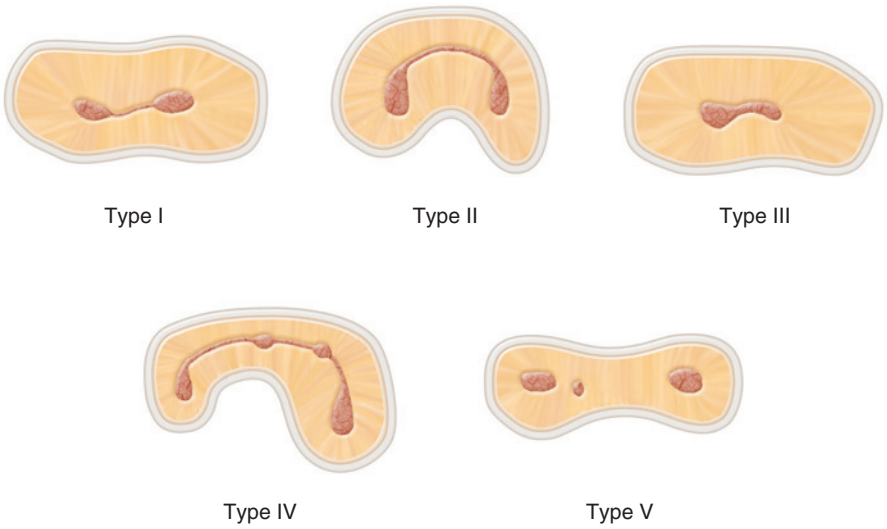

FIG. 5-17 Schematic representation of isthmus classifications described by Kim and colleagues. Type I is an incomplete isthmus; it is a faint communication between two canals. Type II is characterized by two canals with a definite connection between them (complete isthmus). Type III is a very short, complete isthmus between two canals. Type IV is a complete or incomplete isthmus between three or more canals. Type V is marked by two or three canal openings without visible connections. (From Kim S, Pecora G, Rubinstein R, Dorscher-Kim J: *Color atlas of microsurgery in endodontics,* Philadelphia, 2001, Saunders.)

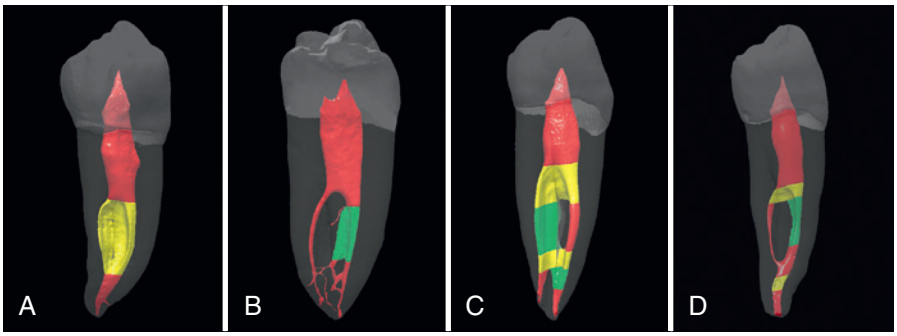

FIG. 5-18 μCT scans of mandibular premolars reflect a wide variation in canal morphology, moving from coronal to apical sections The red color indicates a non–C-shaped canal space; yellow indicates a semilunar canal shape; and green indicated a continuous C-shaped configuration.

Isthmuses are found in 15% of anterior teeth; in maxillary premolar teeth, they are found in 16% at the 1-mm level from the apex and in 52% at the 6-mm level, which puts them primarily in the middle third of the canal (Fig. 5-17). The prevalence of an isthmus increases in the mesiobuccal root of the maxillary first molar, from 30% to 50% near the junction of middle and apical thirds of the root. Eighty percent of the mesial roots of mandibular first molars have these communications at the apical to middle third junction, with the distal root display more in the apical third.

Another change that often occurs in the midroot portion is the splitting of a single canal into two or more canals, along with a wide variation in canal morphology. Likewise, canals often join in this area, starting as coronally two separate canals (Fig. 5-18).

Whenever a root contains two canals that join to form one, the lingual/palatal canal generally is the one with direct access to the apex, although this may require radiographic verification. When one canal separates into two, the division is buccal and palatal/lingual, and the lingual canal generally splits from the main canal at a sharp angle, sometimes nearly a right angle (Fig. 5-19). One study[197] recommended visualizing this configuration as a lower case letter "h". The buccal canal is the straight-line portion of the h; the lingual canal exists about midroot at a sharp angle from the buccal canal. In these situations modification of the access to develop an unobstructed passage of instruments into the lingual canal is indicated.

Although the splitting of one canal into two presents challenges for the clinician, of greater concern is the very small canal that may split off, at times at almost a 90 degree angle, and cannot be located tactilely or even under magnification with the DOM. Although these canals may join the main canal at some point apically, some of them exit separately. This is not uncommon in teeth with C-shaped canals.

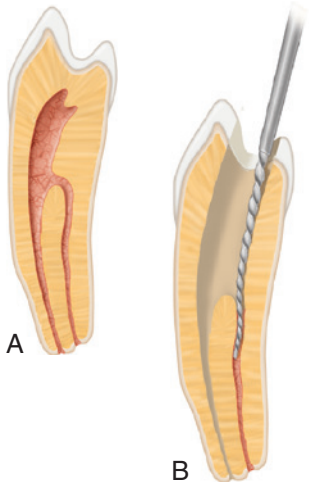

FIG. 5-19 A, Mesial view of a mandibular premolar with a type V canal configuration. The lingual canal separates from the main canal at nearly a right angle. B, This anatomy requires widening of access in a lingual direction to achieve straight-line access to the lingual canal. This should be done with a DOM.

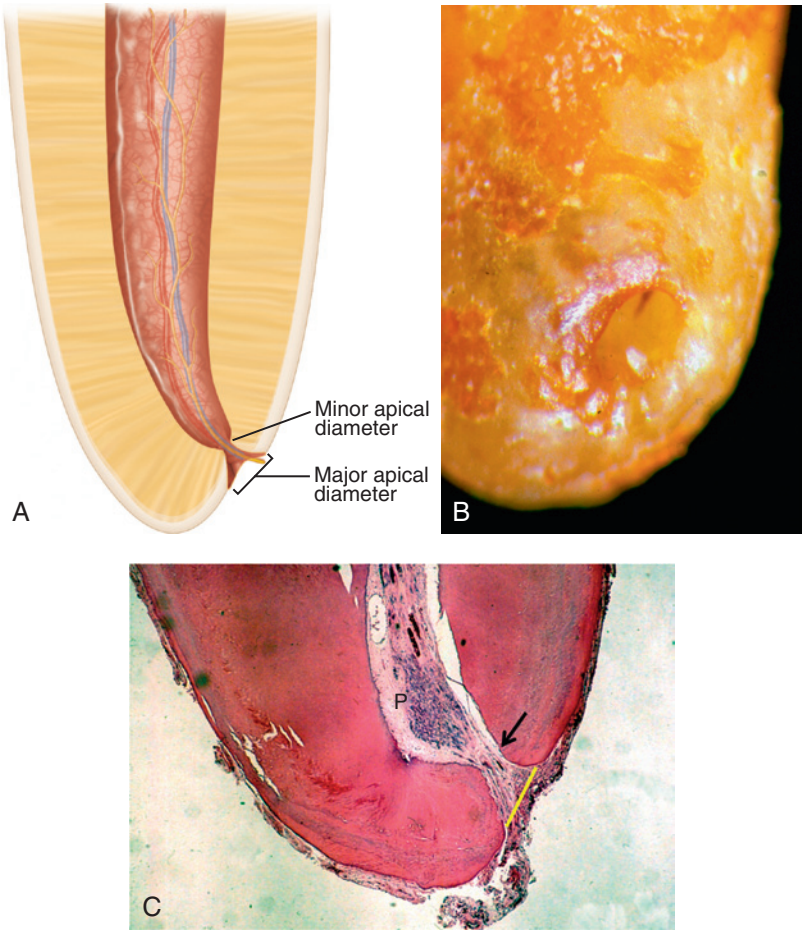

FIG. 5-20 A, Morphology of the root apex. From its orifice the canal tapers to the apical constriction, or minor apical diameter, which generally is considered the narrowest part of the canal. From this point the canal widens as it exits the root at the apical foramen, or major apical diameter. The space between the minor and major apical diameters is funnel shaped. B, Clinical view of major apical foramen. C, Histologic view of the canal constriction and canal foramen. (B and C from Gutmann JL, Lovdahl PE: *Problem solving in endodontics,* ed 5, St Louis, 2011, Elsevier.)

Apical Considerations

The classic concept of apical root anatomy is based on three anatomic and histologic landmarks in the apical region of a root: the apical constriction (AC), the cementodentinal junction (CDJ), and the apical foramen (AF). Kuttler's description of the anatomy of the root apex has the root canal tapering from the canal orifice to the AC, which generally is 0.5 to 1.5 mm coronal to the AF (Fig. 5-20).[109] The AC generally is considered the part of the root canal with the smallest diameter; it also is the reference point clinicians use most often as the apical termination for enlarging, shaping, cleaning, disinfecting, and filling. Violation of this area with instruments or filling materials is not recommended for long-term, successful outcomes.

The CDJ is the point in the canal where cementum meets dentin; it is also the point where pulp tissue ends and periodontal tissues begin. The location of the CDJ in the root canal varies considerably. It generally is not in the same area as the AC, and estimates place it approximately 1 mm from the AF.[184,200]

From the AC, or minor apical diameter, the canal widens as it approaches the AF, or major apical diameter. The space between the major and minor diameters has been described as funnel shaped or hyperbolic, or as having the shape of a morning glory. The mean distance between the major and minor apical diameters is 0.5 mm in a young person and 0.67 mm in an older individual.[109] The distance is greater in older individuals because of the buildup of cementum.

The AF is the "circumference or rounded edge, like a funnel or crater, that differentiates the termination of the cemental canal from the exterior surface of the root."[109] The diameter of the foramen is 502 µm in individuals 18 to 25 years of age and 681 µm in those over age 55, which demonstrates the growth of the AF with age.[109] By comparison, these sizes are larger than the cross-sectional diameter of #50 and #60 root canals files, respectively. The AF does not normally exit at the anatomic apex, but rather is offset 0.5 to 3 mm. This variation is more marked in older teeth through cementum apposition. Studies have shown that the AF coincides with the apical root vertex in 17% to 46% of cases.*

The location and diameter of the CDJ differ from those of the AF in maxillary anterior teeth.[174] Extension of cementum from the AF into the root canal differs considerably, even when opposite canal walls are compared. Cementum reaches the same level on all canal walls in only 5% of cases. The greatest extension generally occurs on the concave side of the canal curvature. This variability confirms that the CDJ and the AC generally are not in the same area and that the CDJ should be considered just a variable junction at which two histologic tissues meet in the root canal (Fig. 5-21). The diameter of the canal at the CDJ varies considerably; it was determined to be 353 µm for the central incisors, 292 µm for the lateral incisors, and 298 µm for the canines.[167] These measures approximate the size of #30 to #35 root canal files.

In maxillary anterior teeth, the root apex and main AF coincided in 17% of examined central incisors and canines and

*References 30, 81, 83, 170, 182, 208, 223, and 227.

FIG. 5-21 Root apex following root canal filling *(RCF)* short of the actual root length. Histologic evidence shows that hard tissue *(black arrows)* has formed from the cells of the periodontal ligament *(PDL)* adjacent to the root filling material. Cementum formation on the internal aspect of the apical foramen is indicated by the yellow arrows. These findings accentuate the variable nature of the apical tissues. (From Gutmann JL, Lovdahl PE: *Problem solving in endodontics,* ed 5, St Louis, 2011, Elsevier.)

TABLE 5-5

Mean Perpendicular Distance from Root Apex to Apical Constriction with Mesiodistal and Labiolingual Diameters at Constriction

Tooth	Mesiodistal (mm)	Labiolingual (mm)	Vertical (mm)
Central incisor	0.37	0.428	0.863
Lateral incisor	0.307	0.369	0.825
Canine	0.313	0.375	1.01

From Mizutani T, Ohno N, Nakamura H: Anatomical study of the root apex in the maxillary anterior teeth, *J Endod* 18:344, 1992.

in 7% of lateral incisors.[139] Both the root apex and the AF of the central incisors and canines were displaced distolabially, whereas those of the lateral incisors were displaced distolingually. The perpendicular distance from the root apex to the AC and both mesiodistal and labiolingual root canal diameters at the AC are shown in Table 5-5. The labiolingual diameter in all maxillary anterior teeth is approximately 50 µm more than the mesiodistal diameter. This has definite implications for intracanal root canal procedures because only the mesiodistal diameter is evident on radiographs.

Scanning electron microscopy has been used to determine the number and size of main apical foramina, their distance from the anatomic apex, and the size of accessory foramina. Morfis et al.[145] observed that more than one main foramen was found in all teeth except the palatal root of maxillary molars

TABLE 5-6

Size of Main Apical Foramina

Teeth	Mean Value (µm)
Maxillary incisors	289.4
Mandibular incisors	262.5
Maxillary premolars	210
Mandibular premolars	268.25
Maxillary molars	
Palatal	298
Mesiobuccal	235.05
Distobuccal	232.2
Mandibular molars	
Mesial	257.5
Distal	392

From Morfis A, Sylaras SN, Georgopoulou M, et al: Study of the apices of human permanent teeth with the use of a scanning electron microscope, *Oral Surg Oral Med Oral Pathol* 77:172, 1994.

and the distal root of mandibular molars. No main foramen was seen in 24% of maxillary premolars and 26% of maxillary incisors. The mesial roots of mandibular molars (50%) and maxillary premolars (48%) and the mesial roots of maxillary molars (42%) had the highest percentage of multiple main foramina. This finding is consistent with observations that blunted roots usually have more than one root canal. The mean values for the size of the main foramen are listed in Table 5-6. Sizes ranged from 210 µm for the maxillary premolars to 392 µm for the distal roots of the mandibular molars. All groups of teeth had at least one accessory foramen. The maxillary premolars had the most and the largest accessory foramina (mean value, 53 µm) and the most complicated apical morphologic makeup. The mandibular premolars had strikingly similar characteristics, a possible reason root canal procedures may fail in premolar teeth.

The morphology of the apical third of the root reflects multiple anatomic variations, including numerous accessory canals; areas of resorption and repaired resorption; attached, embedded, and free pulp stones; varying amounts of reparative dentin; and varying root canal diameters (Table 5-7).[140,141] Primary dentinal tubules are found less often than in the coronal dentin and are more or less irregular in direction and density. Some areas are completely devoid of tubules. Fine tubular branches (300 to 700 µm in diameter) that run at a 45-degree angle to the main tubules and microbranches (25 to 200 µm in diameter) that run at a 90-degree angle to the main tubules are often present (Fig. 5-22). This variable nature of the apical structure and significant absence of dentinal tubules may lead to reduced chances of bacterial invasion into the dentinal walls; however, it also presents challenges for all root canal procedures, from cleaning and disinfection to obturation.

Clinically, considerable controversy exists over the exact termination point for root canal procedures in the apical third of the root; clinical determination of apical canal morphology is difficult at best.[84,193] The existence of an AC may be more conceptual than real. Several studies have reported that a traditional single AC was present less than half the time, particularly when apical root resorption and periradicular pathosis were factors.[44,55,192,238] The apical portion of the root canal often

TABLE 5-7

Median Canal Diameter 1, 2, and 5 mm from Apex

Tooth (Canal) Position	Buccal/Lingual			Mesial/Distal		
	1 mm	2 mm	5 mm	1 mm	2 mm	5 mm
Maxillary						
Central incisor	0.34	0.47	0.76	0.3	0.36	0.54
Lateral incisor	0.45	0.6	0.77	0.33	0.33	0.47
Canine	0.31	0.58	0.63	0.29	0.44	0.5
Premolar						
Single canal	0.37	0.63	1.13	0.26	0.41	0.38
Buccal	0.3	0.4	0.35	0.23	0.31	0.31
Palatal	0.23	0.37	0.42	0.17	0.26	0.33
Molar						
Single mesiobuccal	0.43	0.46	0.96	0.22	0.32	0.29
First mesiobuccal	0.19	0.37	0.46	0.13	0.27	0.32
Second mesiobuccal	0.19	0.31	0.38	0.16	0.16	0.16
Distobuccal	0.22	0.33	0.49	0.17	0.25	0.31
Palatal	0.29	0.4	0.55	0.33	0.4	0.74
Mandibular						
Incisor	0.37	0.52	0.81	0.25	0.25	0.29
Canine	0.47	0.45	0.74	0.36	0.36	0.57
Premolar						
Single	0.35	0.4	0.76	0.28	0.32	0.49
Buccal	0.2	0.34	0.36	0.23	0.29	0.41
Palatal	0.13	0.32	0.37	0.18	0.21	0.17
Molar						
Single M	0.45	0.8	2.11	0.22	0.3	0.29
Mesiobuccal	0.4	0.42	0.64	0.21	0.26	0.32
Mesiolingual	0.38	0.44	0.61	0.28	0.24	0.35
Distal	0.46	0.5	1.07	0.35	0.34	0.59

From Wu M-K, R'oris A, Barkis D, Wesselink P: Prevalence and extent of long oval canals in the apical third, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 89:739, 2000.

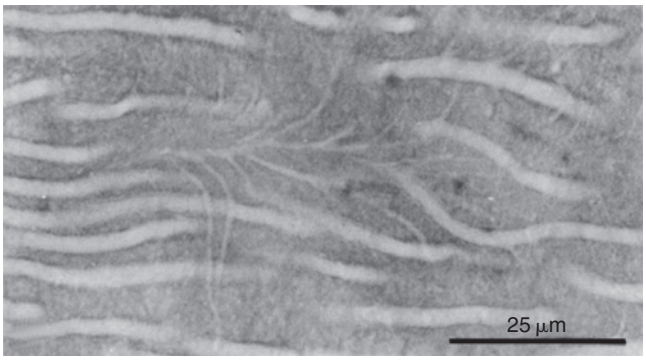

FIG. 5-22 Fine tubules and microbranches can be seen in the apical part of the root. (From Mjör IA, Nordahl I: The density and branching of dentinal tubules in human teeth, *Arch Oral Biol* 41:401, 1996.)

is tapered, or the walls are parallel to each other, or the canal has multiple constrictions.[55] Weine[238] recommended the following termination points: 1 mm from the apex when no bone or root resorption has occurred; 1.5 mm from the apex when only bone resorption has occurred; and 2 mm from the apex when both bone and root resorption are present.

Locating the AC and AF is difficult clinically; for this reason, some researchers contend that the radiographic apex is a more reliable reference point.[250] These authors recommend that root canal procedures terminate at or within 3 mm of the radiographic apex, depending on the pulpal diagnosis. For vital cases, clinical and biologic evidence indicates that a favorable point at which to terminate therapy is 2 to 3 mm short of the radiographic apex.[103,195] This leaves an apical pulp stump, which prevents extrusion of irritating filling materials into the periradicular tissues. However, what is not recognized commonly among clinicians is that this so-called pulp stump is actually not pulp tissue, but rather periodontal tissue that can ensure apical healing with cementum over the root end (see Fig. 5-21). On the other hand, pulp necrosis, bacteria and their byproducts, and biofilms may be present in the apical root canal, which may negate healing if they are not removed thoroughly during root canal procedures. Studies have shown that in these cases, a better success rate is achieved when therapy ends at or within 2 mm of the radiographic apex.[102,103,195] When the intracanal procedures ended short of the 2-mm point or extended past the radiographic apex, the success rate declined by 20%. For revision of procedural failures, apical procedures should extend to or preferably 1 to 2 mm short of the radiographic apex to prevent overextension of instruments and filling materials into the periradicular tissues (see Chapters 6 and 8).

Many investigators who have evaluated apical and periradicular tissues after root canal procedures concluded that the most favorable prognosis was obtained when procedures were terminated at the AC, and the worst prognosis was produced by treatment that extended beyond the AC.[113-116,180] Procedures terminated more than 2 mm from the AC had the second worst prognosis. These findings occurred with root canal procedures in teeth with both vital and necrotic pulps and when bacteria were present beyond the AF. Sealer or gutta-percha (or both) in the periradicular tissues, lateral canals, and apical ramifications may cause a severe inflammatory reaction. However, it is difficult to locate the AC clinically, which is why some studies direct clinicians to terminate all procedures at or beyond the radiographic apex, thereby filling all apical ramifications and lateral canals.[186] This dictate is empirically based, and CBCT evaluation of procedures previously thought to be successful has allowed identification of more posttreatment disease.[158]

Although the apical limit of instrumentation and obturation during root canal procedures continues to be the subject of major controversy, modern electronic apex locators can help the clinician determine the approximate working length of the root canal with greater assuredness. The ultimate challenges are that the two hallmarks of the apical region are its variability and unpredictability. The tremendous variation in canal shapes and diameters complicates enlargement and shaping procedures in all dimensions. Success in these goals depends on the anatomy of the root canal system, the dimensions of the canal walls, use of the appropriate instruments within these confines, and the operator's skill and experience.

OBJECTIVES AND GUIDELINES FOR ACCESS CAVITY PREPARATION

Objectives

Access to the complex root canal system is the first and arguably the most important phase of any nonsurgical root canal procedure.[84,212] The objectives of access cavity preparation are to (1) remove all caries when present, (2) conserve sound tooth structure, (3) unroof the pulp chamber completely, (4) remove all coronal pulp tissue (vital or necrotic), (5) locate all root canal orifices, and (6) achieve straight- or direct-line access to the apical foramen or to the initial curvature of the canal. If done properly, a thorough assessment of the restorative needs of every tooth can be made (e.g., the need for crown lengthening, a post, or simply a bonded core or composite to ensure the structural integrity of the tooth after root canal procedures).

A properly prepared access cavity creates a smooth, straight-line path to the canal system and ultimately to the apex or position of the first curvature (Fig. 5-23, *A*). Straight-line access provides the best chance of débridement of the entire canal space; it reduces the risk of instrument breakage[143]; and it results in straight entry into the canal orifice, with the line angles forming a funnel that drops smoothly into the canal (or canals). Projection of the canal center line to the occlusal surface of the tooth indicates the location of the line angles (Fig. 5-23, *B*). Connection of the line angles creates the outline form. Modifications of the access outline form may be needed to facilitate location of canals and to create a convenient form for the planned procedures.

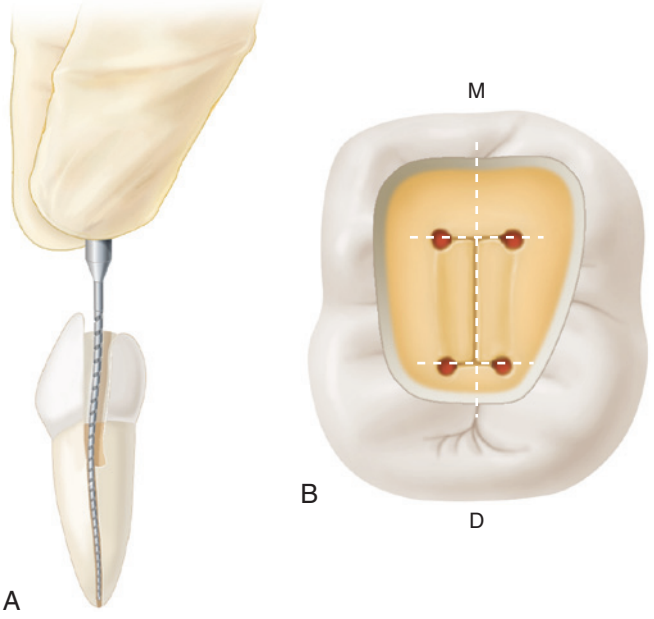

FIG. 5-23 A, Straight-line access to a canal. The instrument should not be deflected until it reaches the initial canal curvature. In some cases coronal tooth structure must be sacrificed to obtain direct access to the pulp chamber. B, Diagrammatic representation of centrality and concentricity of symmetry and location of canal orifices. *D,* Distal; *M,* mesial.

KEY STEPS TO CONSIDER IN ACCESS PREPARATION

Visualization of the Likely Internal Anatomy

Internal tooth anatomy dictates access shape; therefore, the first step in preparing an access cavity is visualization of the position of the pulp space in the tooth. This visualization requires evaluation of angled periapical radiographs and examination of the tooth anatomy at the coronal, cervical, and root levels. Although only two-dimensional, diagnostic radiographs help the clinician estimate the position of the pulp chamber, the degree of chamber calcification, the number of roots and canals, and the approximate canal length. Palpation along the attached gingiva may aid in the determination of root location and direction. This information, when considered together, guides the clinician in the direction of the bur that is chosen to begin cutting the access cavity.

Evaluation of the Cementoenamel Junction and Occlusal Tooth Anatomy

Traditionally, access cavities have been prepared in relation to the occlusal anatomy. However, complete reliance on the occlusal/lingual anatomy is dangerous because this morphology can change as the crown is destroyed by caries and reconstructed with various restorative materials. Likewise, the root may not be perpendicular to the occlusal surface of the tooth; thus complete dependence on the occlusal or lingual anatomy may explain the occurrence of some procedural errors, such as coronal perforations along the cervical line or into the furcation. Krasner and Rankow[107] found that the cementoenamel junction (CEJ) was the most important anatomic landmark for determining the location of pulp chambers and root canal orifices. Their study demonstrated the existence of a specific and consistent anatomy of the pulp chamber floor. These authors proposed guidelines, or spatial concepts, that apply to the 3D assessment of the pulp chamber anatomy and that can help the clinician determine the number and location of orifices on the chamber floor (see Fig. 5-23, *B*).

- *Centrality:* The floor of the pulp chamber is always located in the center of the tooth at the level of the CEJ.
- *Concentricity:* The walls of the pulp chamber are always concentric to the external surface of the tooth at the level of the CEJ; that is, the external root surface anatomy reflects the internal pulp chamber anatomy.
- *Location of the CEJ:* The distance from the external surface of the clinical crown to the wall of the pulp chamber is the same throughout the circumference of the tooth at the level of the CEJ, making the CEJ the most consistent repeatable landmark for locating the position of the pulp chamber.
- *Symmetry:* Except for the maxillary molars, canal orifices are equidistant from a line drawn in a mesiodistal direction through the center of the pulp chamber floor. Except for the maxillary molars, canal orifices lie on a line perpendicular to a line drawn in a mesiodistal direction across the center of the pulp chamber floor.
- *Color change:* The pulp chamber floor is always darker in color than the walls.
- *Orifice location:* The orifices of the root canals are always located at the junction of the walls and the floor; the

orifices of the root canals are always located at the angles in the floor-wall junction; and the orifices of the root canals are always located at the terminus of the roots' developmental fusion lines.

More than 95% of the teeth these investigators examined conformed to these spatial relationships.[107] Slightly less than 5% of mandibular second and third molars did not conform because of the occurrence of C-shaped anatomy.

Preparation of the Access Cavity Through the Lingual and Occlusal Surfaces

Access cavities on anterior teeth usually are prepared through the lingual tooth surface, and those on posterior teeth are prepared through the occlusal surface. These approaches are the best for achieving straight-line access while reducing esthetic and restorative concerns. Some authors have recommended that the traditional anterior access for mandibular incisors be moved from the lingual surface to the incisal surface in selected cases[135]; this allows better access to the lingual canal and improves canal débridement (Fig. 5-24). In teeth that are lingually inclined or rotated, this is often the preferred choice for access and is performed before the dental dam is placed, or careful alignment of the root is determined before a bur is used.[84] Likewise, initial access through crowns may be better achieved without a dental dam in place so that the inclination of the root can be visualized; this information can be used as an indicator of the direction of the long axis of the treated tooth. Furthermore, canal identification and partial enlargement before dental dam placement may be beneficial in some cases. The Micro-Opener (Dentsply Maillefer) (Fig. 5-25, *A*) and EndoHandle with Find and File instruments (Venta Innovative Dental Products, Logan, Utah) (Fig. 5-25, *B*) are excellent instruments for locating canal orifices and developing the canal pathway when a dental dam has not been placed. These flexible, stainless steel hand instruments have variably tapered tips and permit not only canal identification, but also initial pathway formation when indicated. The former has offset handles that provide enhanced visualization of the pulp chamber, whereas the latter permits the use of a wide variety of instruments of different sizes.

FIG. 5-24 An incisal access cavity on mandibular anterior teeth may allow for improved straight-line access and canal débridement.

Removal of All Defective Restorations and Caries before Entry Into the Pulp Chamber

Removal of all defective restorations and caries before entering the root canal system is essential for many reasons.[84] Hidden caries or fractures are often identified, and the ability to determine the restorability of the tooth is enhanced. In this respect it is not uncommon, before entering the pulp chamber, to stop with the access opening preparation in favor of a crown lengthening procedure or adding a bonded buildup. Subsequently, the tooth will be much easier to isolate before entry into the pulp chamber, and sound restorative margins will have been identified. In some cases, an extraction may be indicated because of tooth fracture or unrestorability (Fig. 5-26). Ultimately, with an open preparation, canals are much easier to locate, especially under enhanced illumination and magnification, and intracanal procedures are facilitated. Studies have indicated that clinicians were about 40% more likely to miss fractures, caries, and marginal breakdown if restorations were not completely removed.[1] Working through a clean access also

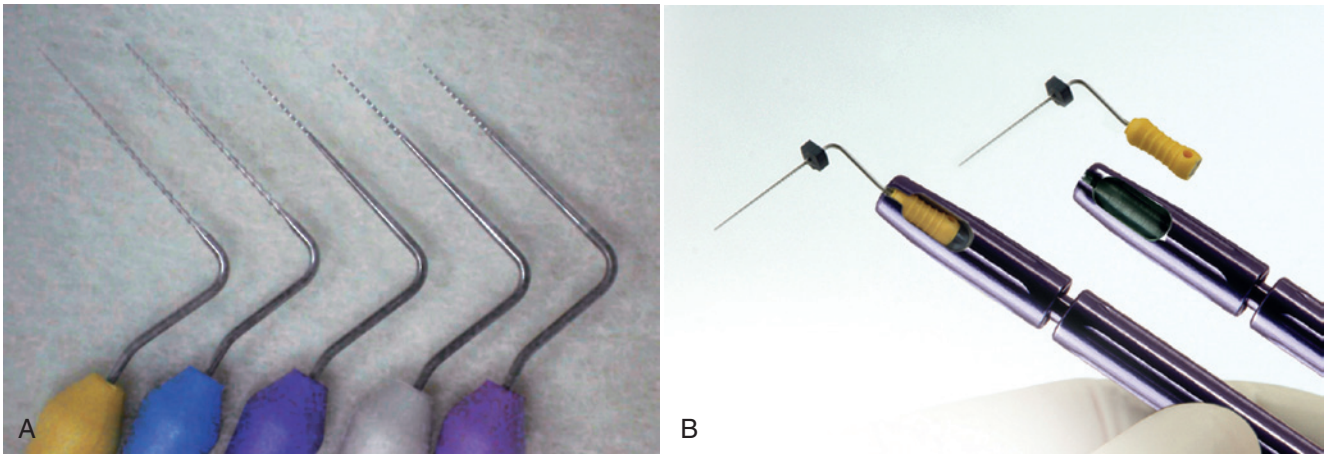

FIG. 5-25 A, Set of Micro-Openers (Dentsply Maillefer) for canal identification and enlargement. B, Similar tool but with changeable instruments that are placed in the EndoHandle (Courtesy Venta Endo) and can be positioned straight or at different angles.

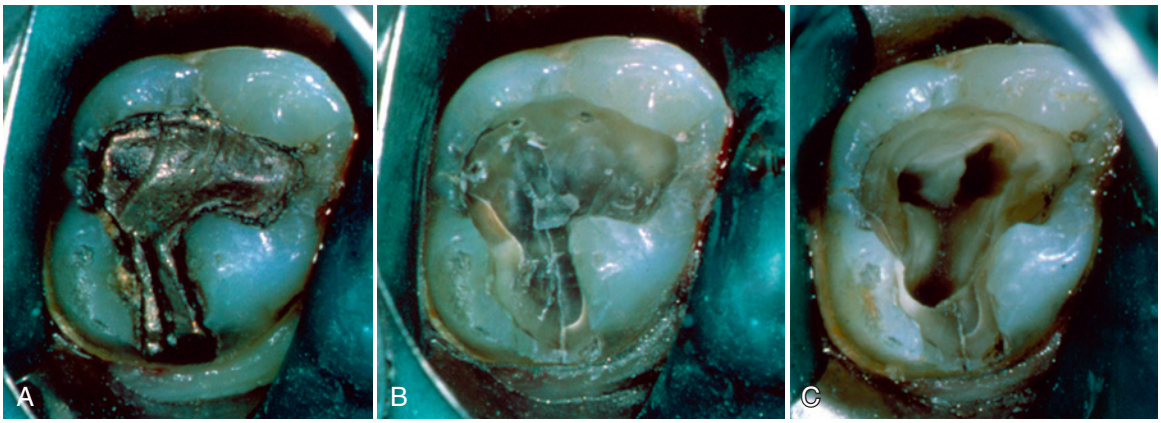

FIG. 5-26 A, Maxillary molar requiring root canal procedure. B, Removal of the amalgam reveals a vertical fracture on the palatal margin. C, Complete cleaning and shaping of the canals. Fracture lines are still visible, but no probings are present. (From Gutmann JL, Lovdahl PE: *Problem solving in endodontics,* ed 5, St Louis, 2011, Elsevier.)

prevents restorative debris from becoming dislodged and inadvertently pushed into the canal system.

Removal of Unsupported Tooth Structure

Along with the removal of caries and restorations, unsupported tooth structure should be removed to prevent tooth fracture during or between procedures. Although unnecessary removal of sound tooth structure should be avoided, oftentimes the access shape may have to be modified to facilitate canal location and intracanal procedures.

Preparation of Access Cavity Walls that Do Not Restrict Straight- or Direct-Line Passage of Instruments to the Apical Foramen or Initial Canal Curvature

Sufficient tooth structure must be removed to allow instruments to be placed easily into each canal orifice without interference from canal walls, particularly when a canal curves severely or leaves the chamber floor at an obtuse angle. Hence, access design depends not only on the orifice location, but also on the position and curvature of the entire canal. The walls of the root canal, rather than the walls of the access preparation, must guide the passage of instruments into the canal. Failure to follow this guideline results in treatment errors, including root perforation, misdirection of an instrument from the main canal (ledge formation), instrument separation, or creation of an incorrect canal shape (apical transportation).

Inspection of the Pulp Chamber Walls and Floor

Magnification is particularly important during initial root canal procedures, especially for determining the location of canals and removing tissue and calcifications from the pulp chamber. Illumination provided by the use of many magnification tools, in particular the DOM, aids in the initial negotiation of constricted, curved, and partially calcified canals. Enhanced vision allows the clinician to see internal dentin color changes and subtle landmarks that may not be visible to the unaided eye, including previously hidden cracks and decay. Surgical loupes and endodontic endoscopes[12] are also available to help in locating intricate root canal systems. In most cases, after dental dam placement, in addition to magnification, a sharp endodontic explorer (DG-16) is used to locate canal orifices and to determine their angle of departure from the pulp chamber.

Tapering of Cavity Walls and Evaluation of Space Adequacy for a Coronal Seal

A proper access cavity generally has tapering walls, with its widest dimension at the occlusal surface. In such a preparation, occlusal forces do not push the temporary restoration into the cavity and disrupt the seal. A minimum 3.5 mm of temporary filling material (e.g., Cavit; 3M ESPE, St. Paul, Minnesota) is needed to provide an adequate coronal seal for a short time.[237] Recently, canal orifice plugs of composite, glass ionomer, and mineral trioxide aggregate (ProRoot MTA, Dentsply Tulsa Dental Specialties) have shown promise in reducing the risk of bacterial contamination of the canal system when microleakage occurs at the coronal-restorative margins.[96]

MECHANICAL PHASES OF ACCESS CAVITY PREPARATION

The preparation of an access cavity requires the following equipment:

- Magnification and illumination
- Handpieces
- Burs
- Endodontic explorers
- Endodontic spoon
- Ultrasonic unit and tips

Magnification and Illumination

The access cavity is best prepared with the use of magnification and an appropriate light source. In lieu of a DOM, surgical loupes with an auxiliary light source are highly recommended.

Handpieces

Good tactile awareness is essential to perform most phases of access preparation with a high-speed handpiece. In many cases,

FIG. 5-27 Access burs: #2, #4, and #6 round carbide burs.

the use of a slow-speed handpiece is indicated, which is especially helpful in the careful excavation of caries located in the area of the anticipated access opening. For challenging access cavity preparations, especially those involving calcified and receded pulp chambers, even experienced clinicians may sacrifice cutting speed and efficiency in favor of the increased cutting control of the slow-speed handpiece or an ultrasonic tip.

Burs

Numerous burs have been developed exclusively for access cavity preparation. Providing a detailed, unabridged list of these burs would be difficult, and most clinicians have their own set of preferred access burs. To meet the overall needs in this regard, various companies have developed access kits with a large variety of burs (Dentsply Tulsa Dental Specialities; SS White, Lakewood, New Jersey; SybronEndo, Orange, California; Ultradent Products, Inc., South Jordan, Utah). In reality, creating an access cavity that meets the previously stated guidelines is more important than worrying about which burs are used in the process. This discussion, therefore, covers some of the more common access burs.

Round carbide burs (sizes #2, #4, and #6) (Fig. 5-27) are used extensively in the preparation of access cavities. They are used to excavate caries and to create the initial external outline shape. They also are useful for penetrating through the roof of the pulp chamber and for removing the roof. Some clinicians prefer to use a fissure carbide bur (Fig. 5-28) or a diamond bur with a rounded cutting end (Fig. 5-29) to perform these procedures. The advantage of the fissure carbide and diamond round-end burs is that they also can be used for some of the axial wall extensions of the access cavity preparation. However, when these burs are used for this purpose by inexperienced clinicians, their cutting ends can gouge the pulp floor and axial walls.

Fissure carbide and diamond burs with safety tips (i.e., noncutting ends) (Fig. 5-30) are safer choices for axial wall extensions. They can be used to extend and favorably orient the axial walls of the pulp chamber. Because they have no cutting end, the burs can be allowed to extend to the pulp floor, and the entire axial wall can be moved and oriented all in one plane from the enamel surface to the pulp floor. Such a technique produces axial walls free of gouges as the final access extensions are created. Fissure carbide and diamond burs also can be used to level off cusp tips and incisal edges,

FIG. 5-28 Access bur: #57 fissure carbide bur.

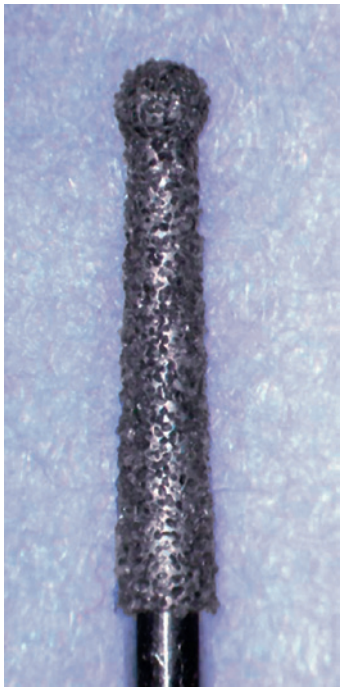

FIG. 5-29 Access bur: Round-end cutting tapered diamond bur.

which are used as reference points for the working length determination.

Round diamond burs (sizes #2 and #4) (Fig. 5-31) are needed when the access must be made through porcelain or metalloceramic restorations.[84] Diamond burs are less traumatic to porcelain than carbide burs and are more likely to penetrate the porcelain without cracking or fracturing it. They should always be used with water spray to control heat buildup in porcelain restorations. After penetrating the porcelain with a diamond bur, a carbide bur, such as a transmetal bur (Dentsply Maillefer) (Fig. 5-32), is used for metal or dentin penetration because of this bur's greater cutting efficiency.[84]

FIG. 5-30 Access burs: Safety-tip tapered diamond bur *(left);* safety-tip tapered carbide bur *(right).*

FIG. 5-31 Access burs: #2 and #4 round diamond burs.

FIG. 5-32 Access bur: Transmetal bur.

A significant trend in restorative dentistry is the increased use of zirconia-based crowns and onlays. Zirconia has different mechanical and thermal characteristics than metal. Carbide burs do not cut zirconia efficiently or safely. Zirconia is a brittle material and when cut can develop cracks that propagate through the framework and lead to eventual failure of the crown or onlay. Diamond bur manufacturers are aware of these issues, and they are currently introducing medium- and

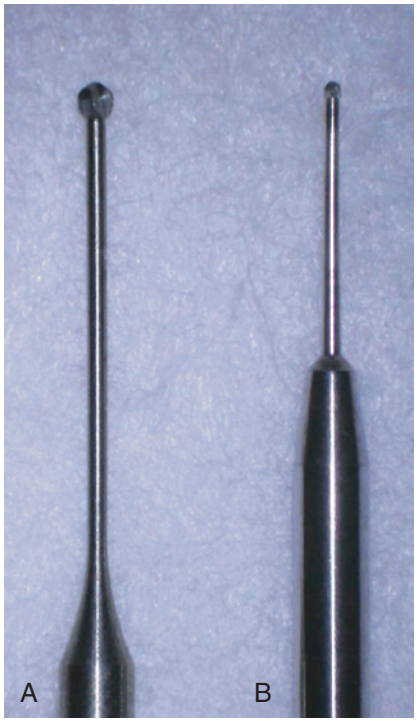

FIG. 5-33 Access burs. **A,** Mueller bur. **B,** LN bur or Extendo Bur.

fine-grit diamond burs that efficiently cut zirconia (Komet USA, Savannah, Georgia; Prima Dental Gloucester, England; SS White). These diamond burs should be used with copious water spray to minimize heat buildup in the zirconia crowns during access preparations.[84] Also, some diamond burs may degrade rapidly when cutting through zirconia and should be thrown away after one use.

Many teeth requiring access cavity preparations have metal restorations that must be penetrated. These restorations may be amalgams, all-metal cast restorations, or metal copings of porcelain fused to metal crowns. As previously mentioned, a transmetal bur is excellent for cutting through metal because of its exceptional cutting efficiency. To penetrate a metallic restoration, a new transmetal bur is recommended for each restoration, together with copious water spray for maximal cutting effect.

When a receded pulp chamber and calcified orifice are identified, or to locate and identify the canal orifice, countersinking or cutting into the root is often indicated. Extended-shank round burs, such as the Mueller bur (Brasseler USA, Savannah, Georgia) (Fig. 5-33, *A*) or Extendo Bur (Dentsply Tulsa Dental Specialties), also known as the LN bur (Dentsply Maillefer) (Fig. 5-33, *B*), can be used. The Munce Discovery bur (CJM Engineering, Santa Barbara, California) is similar to the Mueller bur but has a stiffer shaft and is available in smaller head sizes. The extra-long shank of these burs moves the head of the handpiece away from the tooth, improving the clinician's visibility during this delicate procedure. As an alternative, ultrasonic units offer good visibility with precision cutting.

Once the orifices have been located, they should be flared or enlarged and blended into the axial walls of the access cavity. This process permits the intracanal instruments used during the enlargement and shaping procedures to penetrate the canal effortlessly. Some clinicians might consider this task part of the

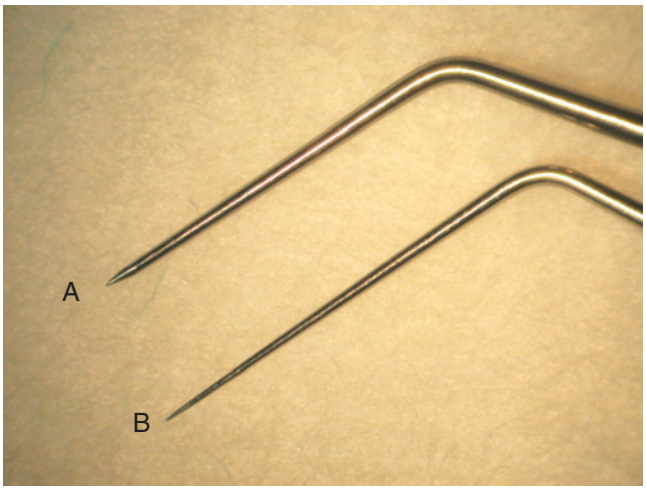

FIG. 5-34 A, Access instruments: DG-16 endodontic explorer. B, JW-17 endodontic explorer.

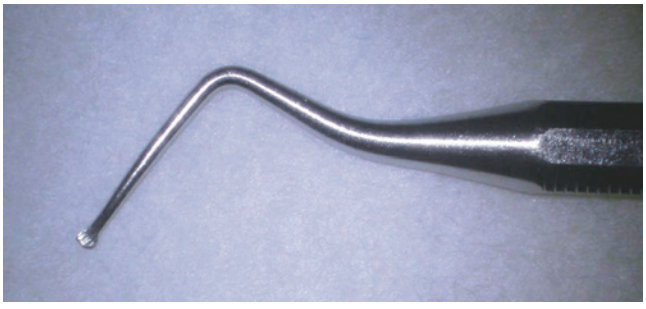

FIG. 5-35 Access instrument: Endodontic spoon.

canal enlargement and shaping phase; however, when done before deep canal penetration, it establishes a well-prepared access opening and may prevent subsequent error.

Endodontic Explorer and Spoon

Various hand instruments are useful for preparing access cavities. The DG-16 endodontic explorer (Fig. 5-34, *A*) is used to identify canal orifices and to determine canal angulation. An alternative, the JW-17 endodontic explorer (Fig. 5-34, *B*) (CK Dental Industries, Orange, California) serves the same purpose, but its thinner, stiffer tip can be useful for identifying the possible location of a calcified canal. A sharp endodontic spoon, which comes in different sizes (Fig. 5-35) can be used to remove coronal pulp and carious dentin. A #17 operative explorer, which can also be found as a doubled-ended instrument with the DG-16, is useful for detecting any remaining overhang from the pulp chamber roof, particularly in the area of a pulp horn of anterior teeth (Fig. 5-36). Failure to remove this overhang, along with the propensity for it to harbor tissue debris, often leads to tooth discoloration, especially in teeth restored only with bonded palatal composite.

Ultrasonic Unit and Tips

Ultrasonic units (Fig. 5-37) and tips specifically designed for activities during access opening preparation are extremely valuable. Ultrasonic tips from various manufacturers can be used to trough and deepen developmental grooves, remove tissue, and explore for canals. Ultrasonic systems provide

FIG. 5-36 Removal of the pulp horn is evaluated with a #17 operative explorer.

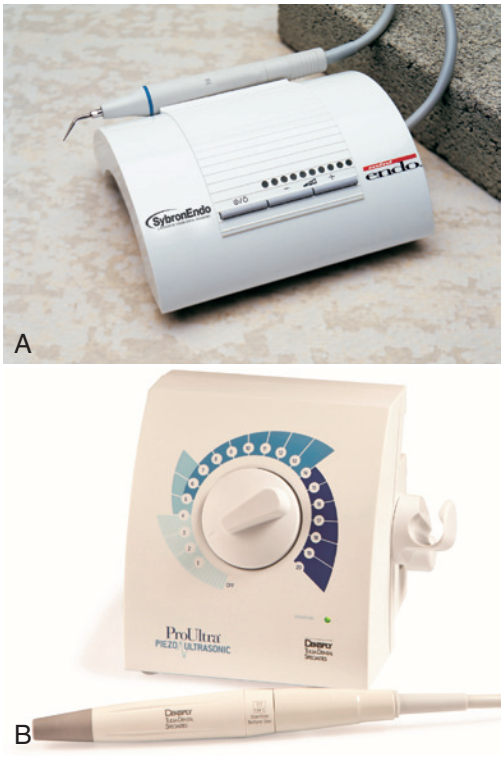

FIG. 5-37 A, Endo ultrasonic unit (MiniEndo II). B, ProUltra Piezo Ultrasonic. (A courtesy SybronEndo, Orange, Calif; B courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, Okla.)

outstanding visibility compared with traditional handpiece heads, which typically obstruct vision. Fine ultrasonic tips, such as Sine Tips, ProUltra Tips, and Smart X BUC Tips (Dentsply Tulsa Dental Specialties; Dentsply Maillefer; and SybronEndo, respectively), are smaller than round burs, and their abrasive coatings or variable surfaces permit careful shaving away of dentin and calcifications during exploration for canal orifices.

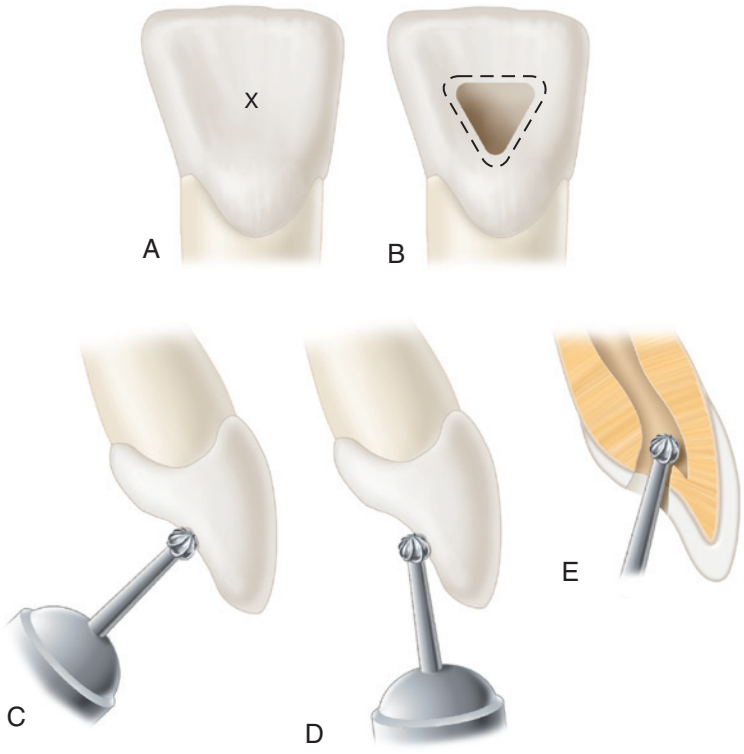

FIG. 5-38 **A,** In anterior teeth the starting location for the access cavity is the center of the anatomic crown on the lingual surface *(X).* **B,** Preliminary outline form for anterior teeth. The shape should mimic the expected final outline form, and the size should be half to three fourths the size of the final outline form. **C,** The angle of penetration for the preliminary outline form is perpendicular to the lingual surface. **D,** The angle of penetration for initial entry into the pulp chamber is nearly parallel to the long axis of the root. **E,** Completion of removal of the pulp chamber roof; a round carbide bur is used to engage the pulp horn, cutting on a lingual withdrawal stroke.

ACCESS CAVITY PREPARATIONS

Anterior Teeth

The following discussion outlines the steps for maxillary and mandibular anterior teeth. Tooth-specific access concerns are illustrated and discussed in the section Morphology of and Access Cavity Preparations for Individual Teeth, later in the chapter.

External Outline Form

Once caries and restorations have been removed as necessary to establish sound tooth margins, an initial external outline opening is cut on the lingual surface of the anterior tooth. This step may also be performed during the removal of caries and restorations. For an intact tooth, cutting commences at the center of the lingual or palatal surface of the anatomic crown (Fig. 5-38, *A*). A #2 or #4 round bur or a tapered fissure bur may be used to penetrate the enamel and slightly into the dentin (approximately 1 mm). An outline form is developed that is similar in geometry to an ideal access shape for the particular anterior tooth (Fig. 5-38, *B*); it is half to three quarters the projected final size of the access cavity. Because most of this step involves removal of enamel, the high-speed handpiece is used for cutting efficiency. The bur is directed perpendicular to the lingual surface as the external outline opening is created (Fig. 5-38, *C*).

Penetration of the Pulp Chamber Roof

Experienced clinicians usually penetrate the pulp chamber roof using a high-speed handpiece; however, less experienced clinicians may find the increased tactile sensation of a slow-speed handpiece a safer option. Continuing with the same round or tapered fissure bur, the angle of the bur is rotated from perpendicular to the lingual/palatal surface to parallel to the long axis of the root (see Fig. 5-38, *D*). Penetration into the tooth continues along the root's long axis until the roof of the pulp chamber is penetrated; frequently, a drop into the chamber effect is felt when this occurs. Measuring the distance from the incisal edge to the roof of the pulp chamber on a dimensionally accurate pretreatment radiograph may serve as guide in limiting penetration and possibly preventing a perforation. If the drop-in effect is not felt at this depth, an endodontic explorer can be used to probe the depth of the access, using magnification or the DOM. Often a small opening into the chamber is present, or the dentin is very thin and the explorer penetrates into the chamber. The depth and angle of penetration should be assessed for any deviation away from the long axis of the root in both the mesiodistal and buccolingual dimensions, and the penetration angle should be realigned if necessary. Angled radiographs can be used to assess progress at any time if any confusion or doubt exists. A little caution and concern at this stage can prevent a mishap.

Removal of the Chamber Roof

Once the pulp chamber has been penetrated, the remaining roof is removed by catching the end of a round bur under the lip of the dentin roof and cutting on the bur's withdrawal stroke (see Fig. 5-38, *E*). Because each tooth has a unique pulp chamber anatomy, working in this manner enables the internal pulp anatomy to dictate the external outline form of the access opening. In teeth with irreversible pulpitis, pulp tissue hemorrhage can impair vision during this process. In such cases, as soon as sufficient roof structure has been removed to allow instrument access, the coronal pulp should be amputated at the orifice level with a spoon or round bur and the chamber irrigated copiously with sodium hypochlorite. If the hemorrhage continues, a tentative canal length can be established by measuring the pretreatment radiograph. A small broach coated

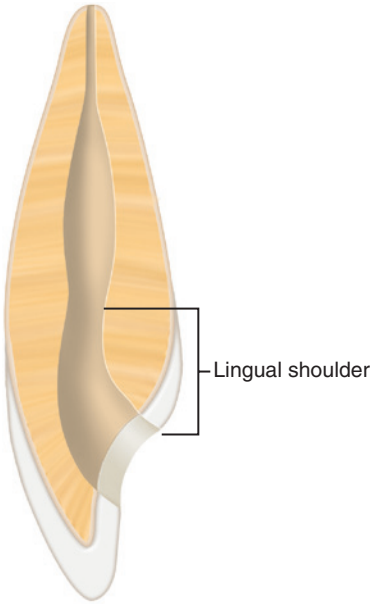

FIG. 5-39 Lingual shoulder of the anterior tooth, extending from the cingulum to 2 mm apical to the orifice.

FIG. 5-40 Placing an incisal bevel on the lingual surface of a maxillary anterior tooth can lead to fracture of the permanent restoration during occlusal function.

with a chelating agent then can be used loosely in the canal to grasp and sever the pulp at a more apical level. Copious irrigation with sodium hypochlorite helps control hemorrhage. Subsequently, the chamber roof, including the pulp horns, is removed and all internal walls are flared to the lingual surface of the tooth. Complete roof removal is confirmed with a #17 operative explorer if no "catches" are identified as the explorer tip is withdrawn from the pulp chamber along the mesial, distal, and facial walls.

Removal of the Lingual Shoulder and Coronal Flaring of the Orifice

Once the orifice or orifices have been identified and confirmed, the lingual shoulder or ledge is removed. This is a shelf of dentin that extends from the cingulum to a point approximately 2 mm apical to the orifice (Fig. 5-39). Its removal improves straight-line access and allows for more intimate contact of files with the canal's walls for effective shaping and cleaning. In addition, its removal from mandibular anterior teeth may often expose an extra orifice and canal.

The contemporary approach to flaring the orifice involves the use of rotary nickel-titanium (NiTi) orifice openers that allow rapid, safe removal of the lingual ledge, following the manufacturer's directions for use (DFUs). When used properly, these openers allow refinement of the orifice shape or help to enhance straight-line access to the canal with minimal removal of dentin. Although there may be subtle differences in these instruments from manufacturer to manufacturer, proper application achieves the same objective. A time-tested and traditional way to achieve the same goal involves the use of a tapered, safety-tip diamond or carbide bur or a Gates-Glidden bur. However, use of these instruments may result in excessive removal of cervical dentin. When a fine, safety-tip diamond bur is used, the tip is placed approximately 2 mm apical to the canal orifice and inclined to the lingual during rotation to slope the lingual shoulder. The bur should be placed so as to avoid putting a bevel on the incisal edge of the access preparation (Fig. 5-40). When Gates-Glidden burs are used, the largest that can be placed passively 2 mm apical to the orifice is used first. During rotation, gentle pressure is applied on the bur as it cuts against the lingual shoulder and then is withdrawn. The size of these burs can be increased sequentially, depending on the size of the canal, with repeated shaping of the lingual wall until the lingual shoulder of dentin has been eliminated in anterior teeth. During this process, the orifice is often concomitantly flared so that it is contiguous with all walls of the access preparation. If this is not achieved, use of the orifice openers is recommended.

Straight-Line Access Determination

After removal of the lingual shoulder and flaring of the orifice, straight-line access must be determined. Ideally, a small intracanal file can reach the apical foramen or the first point of canal curvature with no deflections. Unnecessary deflection of the file can result in numerous consequences related to loss of instrument control. Deflected instruments function under more stress than those with minimal or no deflection pressure and are more susceptible to separation during enlargement and shaping (Fig. 5-41). Deflected instruments also lack access to critical areas of the canal and therefore do not function effectively. Without straight-line access, procedural errors (e.g., ledging, transportation, and zipping) may occur, but this is primarily seen with the use of hand files or larger NiTi instruments (Fig. 5-42).

If the lingual shoulder has been removed properly and a file still binds on the incisal edge, the access cavity should be extended further incisally until the file is not deflected (Fig. 5-43). The final position of the incisal wall of the access cavity is determined by two factors: (1) complete removal of the pulp horns and (2) straight-line access.

Visual Inspection of the Access Cavity

Appropriate magnification and illumination should be used to inspect and evaluate the completed access cavity. Although this

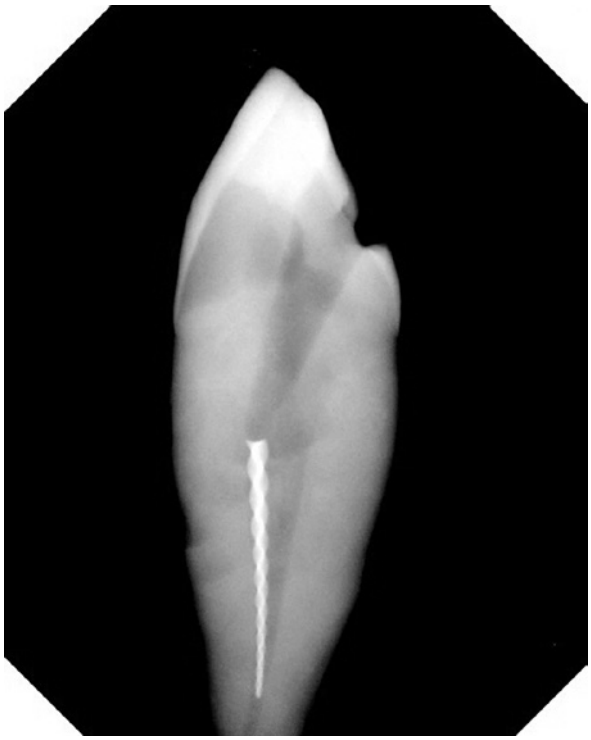

FIG. 5-41 Separation of a rotary endodontic instrument as a result of underextended access preparation rather than canal binding.

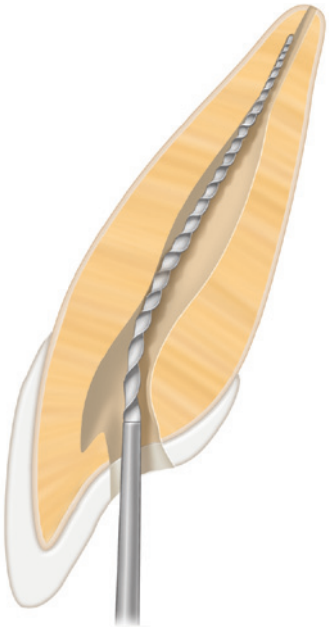

FIG. 5-42 Inadequate access preparation. The lingual shoulder was not removed, and incisal extension is incomplete. The file has begun to deviate from the canal in the apical region, creating a ledge.

can be done during any stage of the preparation, it should always be done at this point. The axial walls at their junction with the orifice must be inspected for grooves that might indicate an additional canal. The orifice and coronal portion of the canal must be evaluated for a bifurcation.

FIG. 5-43 A, The lingual ledge of dentin remains, deflecting the file toward the labial wall. As a result, portions of the lingual canal wall will not be shaped and cleaned. B, Removal of the lingual ledge results in straight-line access.

Refinement and Smoothing of Restorative Margins

The final step in the preparation of an access cavity is to refine and smooth the cavosurface margins. Rough or irregular margins can contribute to coronal leakage through a permanent or temporary restoration. Proper restorative margins are important because anterior teeth may not require a crown as the final restoration. Smooth cavosurface margins allow the placement of a composite resin restoration with the precision necessary to minimize coronal leakage. Such leakage could jeopardize the success of the root canal procedure.

Another factor to consider regarding the access margin of a maxillary anterior tooth is that the final composite resin restoration will be placed on a functional tooth surface. The incisal edges of the mandibular anterior teeth slide over these maxillary lingual surfaces during excursive jaw movements. Therefore, the restorative margins of an access cavity in maxillary anterior teeth should be created to allow a bulk of restorative material at the margin. Butt joint margins are indicated, rather than beveled margins, which produce thin composite edges that can fracture under functional loads and ultimately result in coronal leakage. If the anterior tooth requires a crown as the final restoration, the cavosurface margin becomes a less critical factor, although if it is not restored in a timely fashion, breakdown and leakage may occur.

Individual Anterior Teeth

See the figures in the section Morphology of and Access Cavity Preparations for Individual Teeth, later in this chapter.

Posterior Teeth

Preparing access cavities on posterior teeth is similar to the process for anterior teeth, but significant differences warrant a separate discussion.[84] Posterior teeth requiring root canal procedures typically have been heavily restored or the carious process was extensive. Such conditions, along with the complex pulp anatomy and position of posterior teeth in the oral cavity, can make the access process challenging.

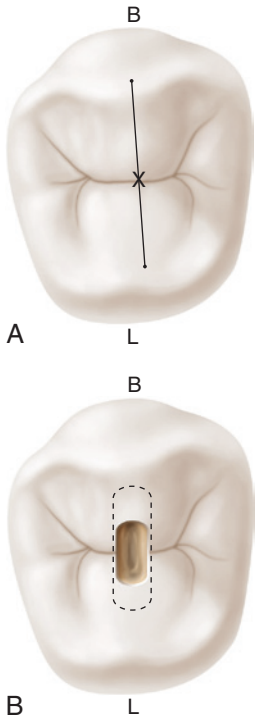

FIG. 5-44 A, Starting location for access to the maxillary premolar *(X)*. B, Initial outline form *(dark area)* and projected final outline form *(dashed line)*. *B,* Buccal; *L,* lingual.

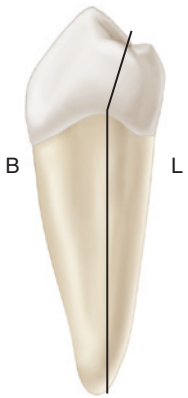

FIG. 5-45 The crown of a mandibular premolar is tilted lingually relative to the root. *B,* Buccal; *L,* lingual.

External Outline Form

Removal of caries and existing restorations from a posterior tooth requiring a root canal procedure often results in the development of an acceptable access outline form. However, if the tooth is intact, the access starting location must be determined for an intact tooth. The pulp chamber of posterior teeth is positioned in the center of the tooth at the level of the CEJ. Therefore, in maxillary premolars, the point of entry that determines the external outline form is on the central groove between the cusp tips (Fig. 5-44). Crowns of mandibular premolars are tilted lingually relative to their roots (Fig. 5-45); therefore, the starting location must be adjusted to compensate for this tilt (Fig. 5-46). In mandibular first premolars, the starting location is halfway up the lingual incline of the buccal cusp on a line connecting the cusp tips. Mandibular second premolars require less of an adjustment because they have less lingual inclination. The starting location for this tooth is one third the way up the lingual incline of the buccal cusp on a line connecting the buccal cusp tip and the lingual groove between the lingual cusps.

To determine the starting location for molar access cavity preparations, the mesial-distal and apical-coronal boundary limitations for this outline must be determined (Fig. 5-47). Evaluation of bite-wing radiographs is an accurate method of assessing the mesiodistal extensions of the pulp chamber (Fig. 5-48). The mesial boundary for both the maxillary and mandibular molars is a line connecting the mesial cusp tips. Pulp chambers are rarely found mesial to this imaginary line. A good initial distal boundary for maxillary molars is the oblique ridge. For mandibular molars, the initial distal boundary is a line connecting the buccal and lingual grooves. For molars the correct starting location is on the central groove halfway between the mesial and distal boundaries.

Penetration through the enamel into the dentin (approximately 1 mm) is achieved with a #2 or #4 round bur for premolars and a #4 or #6 round bur for molars. A tapered fissure bur may be used instead of round burs. The bur is directed perpendicular to the occlusal table, and an initial outline shape is created at about half to three fourths its projected final size. The premolar shape is oval and widest in the buccolingual dimension. The molar shape is also oval initially; it is widest in a buccolingual dimension for maxillary molars and in a mesiodistal direction for mandibular molars. The final outline shape for molars is approximately triangular (for three canals) or rhomboid (for four canals); however, the canal orifices dictate the position of the corners of these geometric shapes. Therefore, until the orifices have been located, the initial outline form should be left as roughly oval.

Penetration of the Pulp Chamber Roof

Once initial penetration into the pulp chamber has been achieved, the angle of penetration changes from perpendicular to the occlusal table to an angle appropriate for penetration through the roof of the pulp chamber. In premolars the angle is parallel to the long axis of the root or roots, both in the mesiodistal and buccolingual directions. Failure to analyze this penetration angle carefully can result in gouging or perforation because premolar roots often are tilted relative to the occlusal plane. In molars the penetration angle should be toward the largest canal because the pulp chamber space usually is largest just occlusal to the orifice of this canal. Therefore, in maxillary molars, the penetration angle is toward the palatal orifice, and in mandibular molars, it is toward the distal orifice (Fig. 5-49).

As with anterior teeth, penetration is limited to the distance measured on a pretreatment radiograph to just penetrate the roof of the pulp chamber. If the drop-in effect is not felt at this depth, a careful evaluation of the angle of penetration is necessary before going deeper into the chamber. In multirooted posterior teeth, lateral and furcation perforations may occur rapidly without attention to 3D detail during this penetration. As with anterior teeth, aggressive probing with an endodontic explorer at any time during the penetration often can help locate the pulp chamber.

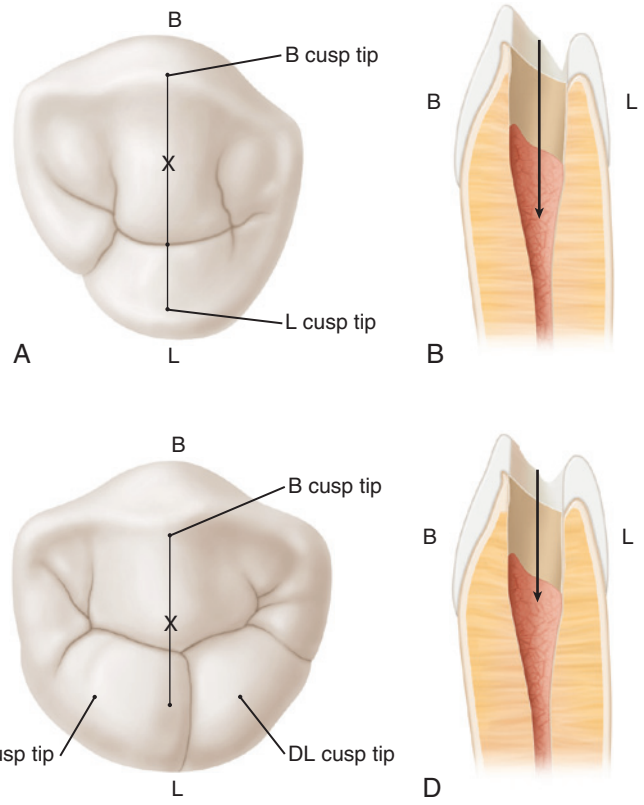

FIG. 5-46 **A,** Mandibular first premolar and access starting location *(X)* (occlusal view). **B,** Mandibular first premolar and starting location (proximal view). **C,** Mandibular second premolar and access starting location *(X)* (occlusal view). **D,** Mandibular second premolar and starting location (proximal view). *B,* Buccal; *DL,* distolingual; *L,* lingual; *ML,* mesiolingual.

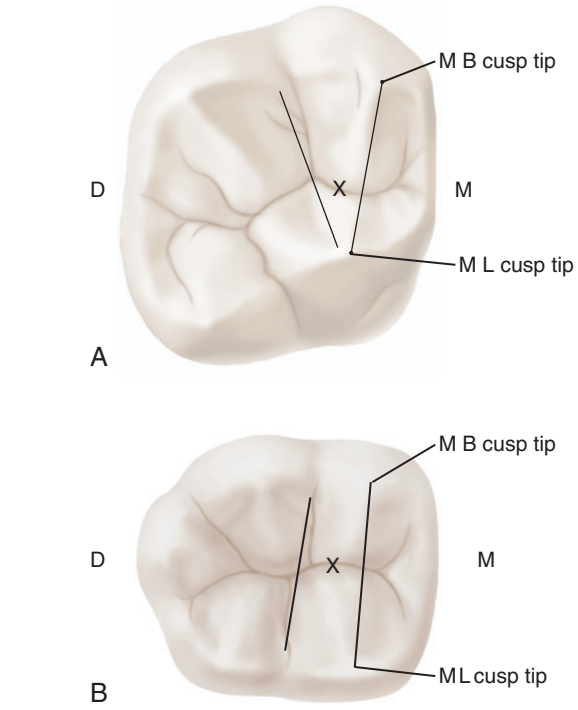

FIG. 5-47 **A,** Mesial and distal boundary of a maxillary molar with the access starting location *(X)*. **B,** Mesial and distal boundary of a mandibular molar showing the access starting location *(X)*. *D,* Distal; *M,* mesial; *MB,* mesiobuccal; *ML,* mesiolingual.

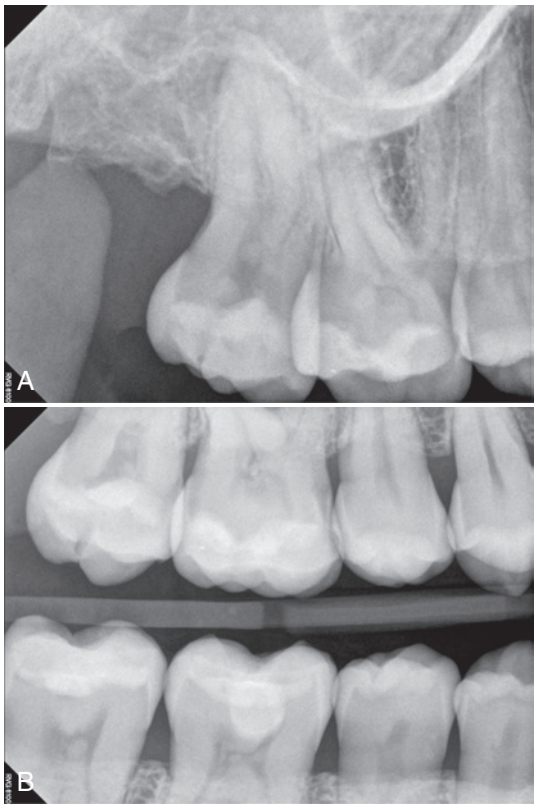

FIG. 5-48 **A,** Periapical radiograph of the teeth in the maxillary right quadrant. **B,** Bite-wing radiograph of the same teeth provides a clearer delineation of the pulpal morphology.

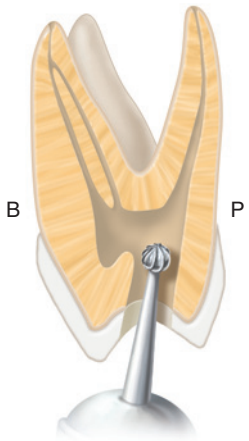

FIG. 5-49 Angle of penetration toward the largest canal (palatal) in a maxillary molar. *B,* Buccal; *P,* palatal.

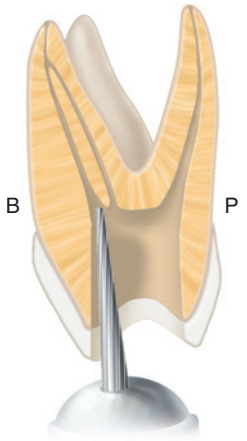

FIG. 5-51 Safety-tip carbide bur is used to shape the axial wall in one plane from the orifice to the cavosurface margin. *B,* Buccal; *P,* palatal.

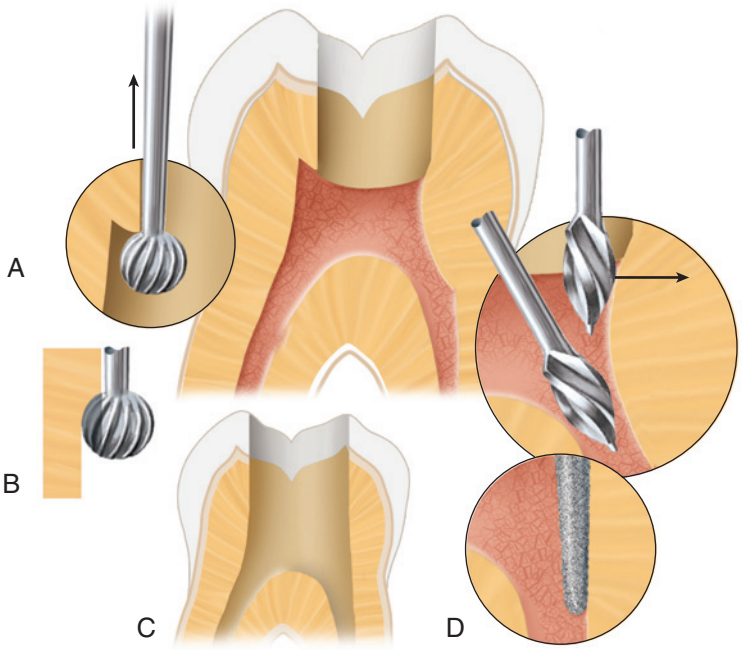

FIG. 5-50 **A,** Pulp roof/pulp horn removal. The round bur hooks under the lip of the pulp horn. **B,** The bur is rotated and withdrawn in an occlusal direction to remove the lip. **C,** Removal of a cervical dentin bulge. A Gates-Glidden bur is placed just apical to the orifice and withdrawn in a distoocclusal direction. **D,** A safety-tip tapered diamond bur is used to blend and funnel the axial wall from the cavosurface margin to the orifice.

Removal of the Chamber Roof

The bur of choice is used to remove the roof of the pulp chamber completely, including all pulp horns (Fig. 5-50, *A* and *B*). Visibility problems caused by pulpal hemorrhage should be handled as described in the previous section for anterior teeth. The goal is to funnel the corners of the access cavity directly into the orifices, and a safety-tip diamond or carbide bur (Multi Bur, Dentsply Tulsa Dental Specialties) performs this task nicely (Fig. 5-50, *C* and *D*); it can be set on the pulp floor and the entire axial wall shaped at one time, with little or no apical pressure (Fig. 5-51). These burs are passed between the orifices along the axial walls to remove the roof, taper the internal walls, and create the desired external outline shape simultaneously.

Identification of All Canal Orifices

In posterior teeth with multiple canals, the canal orifices play an important role in determining the final extensions of the external outline form of the access cavity. Ideally, the orifices are located at the corners of the final preparation to facilitate all of the root canal procedures (Fig. 5-52). Internally, the access cavity should have all orifices positioned entirely on the pulp floor and should not extend into an axial wall. Extension of an orifice into the axial wall creates a "mouse hole" effect (Fig. 5-53), which indicates internal underextension and impedes straight-line access. In such cases the orifice must be repositioned onto the pulp floor without interference from axial walls.

Removal of the Cervical Dentin Bulges and Orifice and Coronal Flaring

In posterior teeth the internal impediments to an ideal access opening are the cervical dentin ledges or bulges and the natural coronal canal constriction.[84] The cervical bulges are shelves of dentin that frequently overhang orifices in posterior teeth, restricting access into root canals and accentuating existing

canal curvatures.[117] They can develop from mesial, distal, buccal, and lingual walls inward. These bulges can be removed safely with burs or ultrasonic instruments. The removal instruments should be placed at the orifice level, and light pressure should be used to cut laterally toward the dentin bulge to remove the overhanging ledge (Fig. 5-54). After removal of the ledge, the orifice and constricted coronal portion of the canal can be flared with NiTi orifice openers, Gates-Glidden burs, or large, tapered rotary instruments (.10/.12), which are used in a sweeping upward motion with minimal lateral pressure away from the furcation. As the orifice is enlarged, it should be tapered and blended into the axial wall so that an explorer can slide down the corner of the external outline form, down the axial wall, and into the orifice without encountering any obstructions (see Fig. 5-54).

FIG. 5-52 An endodontic explorer is used to search for canal orifices.

Straight-Line Access Determination

Straight-line access is paramount to successful shaping, especially given the complexity of the root canal systems in posterior teeth. Files must have unimpeded access to the apical foramen or the first point of canal curvature to perform properly. Therefore, each canal must have straight-line access, and adjustments must be made accordingly (see Fig. 5-54, *O*).

Visual Inspection of the Pulp Chamber Floor

The floor and walls must be inspected, using appropriate magnification and illumination, to ensure that all canal orifices are visible and no roof overhangs are present (Fig. 5-55).

Refinement and Smoothing of the Restorative Margins

In both temporary and interim permanent restorations, the restorative margins should be refined and smoothed to minimize the potential for coronal leakage. The final permanent restoration of choice for posterior teeth that have undergone a root canal procedure is generally a crown or onlay, although this may vary, depending on the opposing dentition and patient function.

Individual Posterior Teeth

See the figures in the section Morphology of and Access Cavity Preparations for Individual Teeth, later in the chapter.

CHALLENGING ACCESS PREPARATIONS

Teeth with Minimal or No Clinical Crown

Forming an access cavity on a tooth with little or no clinical crown might seem to be a simple procedure. For example, in young teeth, traumatic fractures often expose the pulp chamber, making access preparation easy. However, in older teeth with previous caries or large restorations, the pulp chambers typically have receded or calcified (Fig. 5-56). Loss of significant coronal anatomy to guide penetration angles can make access quite difficult. A thorough evaluation of these teeth clinically and their root angulation on pretreatment radiographs is essential. Pulp chambers are located at the center of the crown at the level of the CEJ. Access often is started without a dental dam in place so that root eminences can be visualized and palpated as access preparation progresses (Fig. 5-57). Every effort is made to stay centered within the root for the best

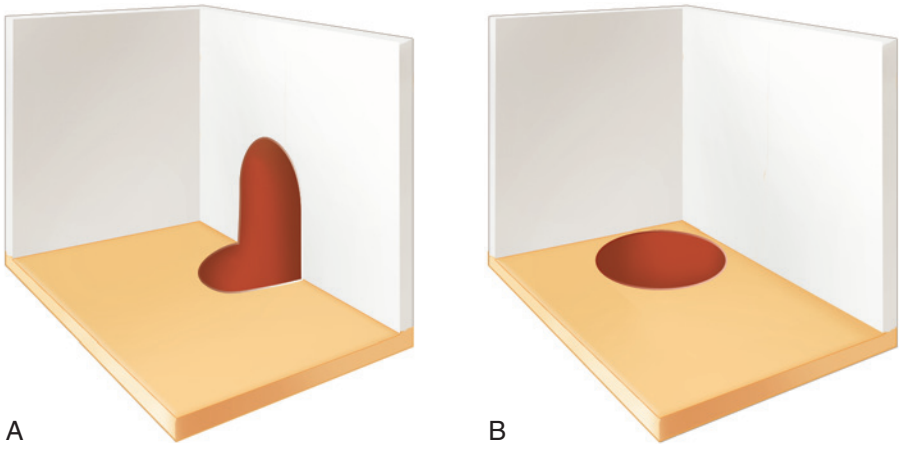

FIG. 5-53 A, Mouse hole effect caused by extension of the orifice into the axial wall. B, Orifice that lies completely on the pulp floor.

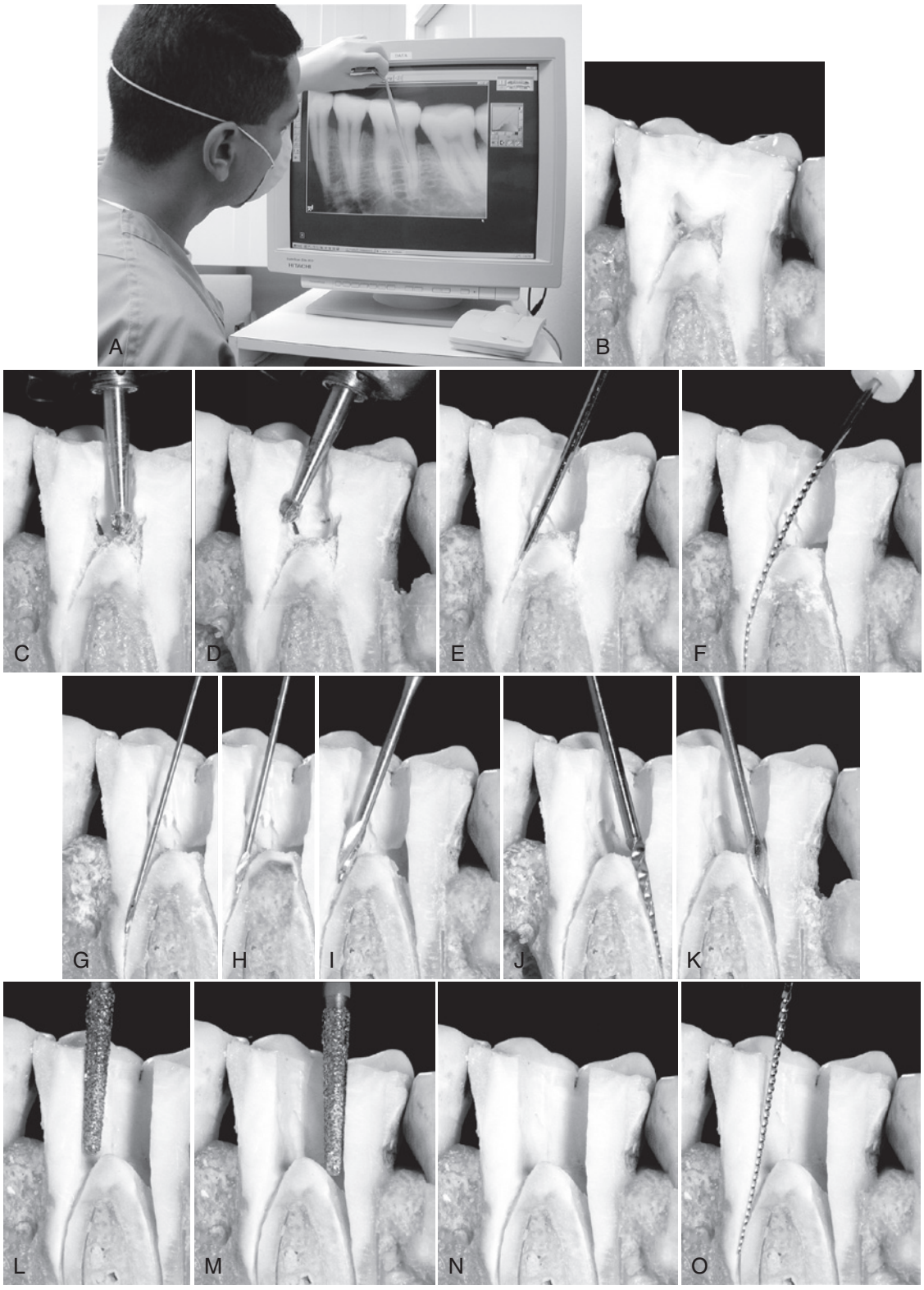

FIG. 5-54 A, Evaluation of the pretreatment radiograph. B, Clinical evaluation of the tooth. C, Penetration of the pulp roof. D, Removal of the pulp roof/pulp horns with a round carbide bur. E, Location of the orifice with a Mueller or LN bur. F, Exploration of the canal with a small K-file. G to I, Flaring of the orifice/coronal third of the mesial canal with Gates-Glidden burs. J, Flaring of the orifice/coronal third of the distal canal with a #.12 taper nickel-titanium rotary file. K, Flaring of the orifice/coronal third of the distal canal with a Gates-Glidden bur. L, Funneling of the mesial axial wall from the cavosurface margin to the mesial orifice. M, Funneling of the distal axial wall from the cavosurface margin to the distal orifice. N, Completed access preparation. O, Verification of straight-line access.

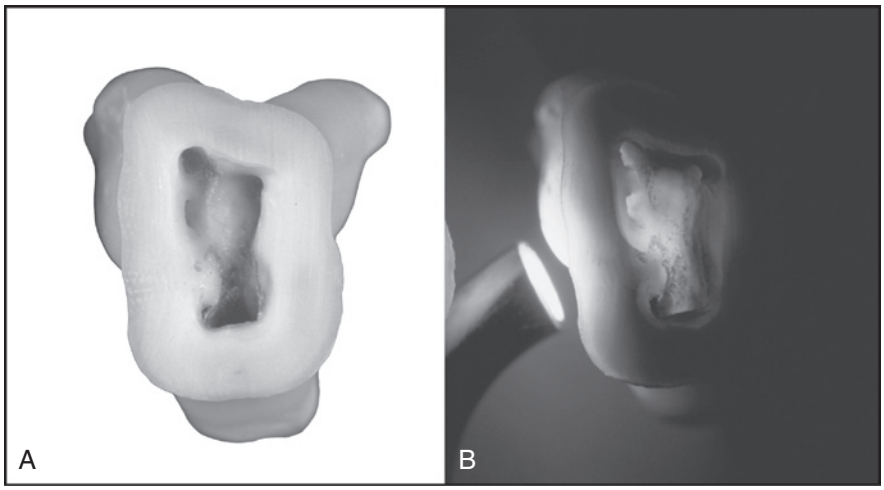

FIG. 5-55 A fiberoptic light can be applied to the cervical aspect of the crown to help obtain maximal visibility with magnification. Transillumination often reveals landmarks otherwise invisible to the unaided eye.

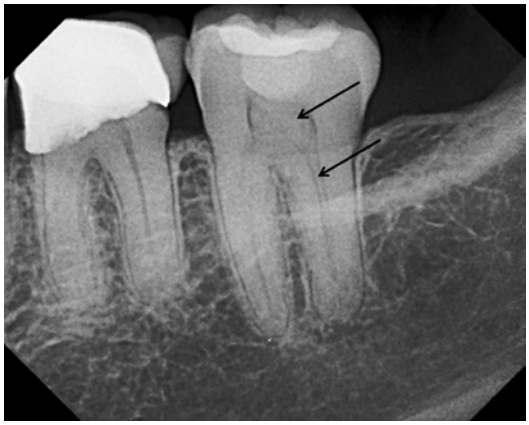

FIG. 5-56 Mandibular molar with significant calcification of the pulp chamber and canal spaces *(arrows).*

chance of locating the pulp canal.[25] The depth of penetration needed to reach the pulp canal is measured on a pretreatment radiograph. If this depth is reached without locating the canal, two radiographs should be taken before the process proceeds. A straight-on radiograph shows whether the preparation is deviating in a mesial or distal direction. Applying the buccal object rule, an angled radiograph shows a buccal or lingual deviation in penetration. After checking these radiographs, the operator can alter the penetration angle, if necessary, while penetrating apically. As soon as the pulp canal is identified, the dental dam must be placed and the access preparation finalized, using the guidelines discussed previously.

Heavily Restored Teeth (Including Those with Full Coronal Coverage)

Restorative materials often alter the external anatomic landmarks on the crown of a tooth, making access preparation difficult. Restorative materials and full crowns rarely reproduce the original tooth anatomy in the exact same position. The crown-to-root angulation often is altered when large restorations or crowns correct occlusal discrepancies (Fig. 5-58). Most restorative materials block the passage of light into the internal aspects of the tooth, resulting in poor visibility during preparation of the access cavity. All these factors, singly or together, complicate the preparation of access cavities on heavily restored teeth (Fig. 5-59).[1] The DOM and transillumination of the cervical area of a heavily restored tooth can greatly improve visibility and reveal landmarks that otherwise would be missed (see Fig. 5-55).

In most cases, complete removal of large restorations is the wisest course, and treatment should be planned accordingly (Fig. 5-60). These restorations often have leaking, defective margins or recurrent caries, or both. Removing the restoration enhances visibility of the internal anatomic structures through direct visualization and increased light penetration. With increased visibility, recurrent caries and fracture lines on the pulp chamber walls or floor can be seen, especially with the DOM. Clinicians are 40% more likely to miss these anomalies when restorations are not removed completely.[1] Better visibility also makes locating receded or calcified canals easier.

Coronal leakage often occurs when parts of large restorations are left in the tooth because the restorations are loosened by the vibration of the access drilling. Furthermore, removal of these restoration remnants prevents pieces of the restorative material from falling into the root canal. Instruments can rub against restoration fragments during the root canal procedures, creating filings that can be carried into the canal system. Thorough removal prevents these problems.

Complete removal of an extensive restoration from the cervical region of the tooth permits more direct access to the root canal or canals. For example, class V restorations often cause calcifications in the coronal portion of the canal, making location of the canal through the occlusal approach quite difficult. Removal of the class V restoration allows more direct access to the canal, which makes location and penetration much easier. Any remaining canals can be managed through the traditional occlusal access cavity (Fig. 5-61).

When an extensive restoration is a full metalloceramic or partial veneer crown, the restoration must be evaluated thoroughly. If any concerns arise about recurrent decay or leaking margins, the crown should be removed before an access cavity is formed. Removal of the crown allows elimination of all recurrent caries and improves the visibility of the pulp spaces.

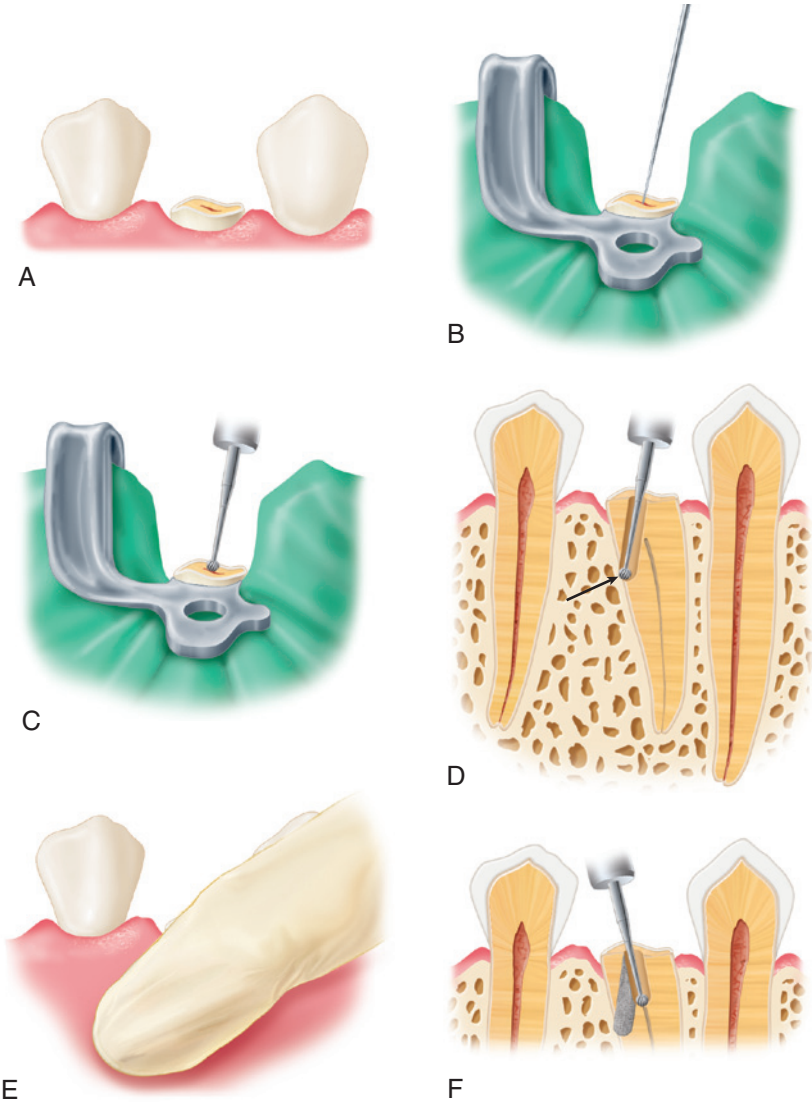

FIG. 5-57 Access cavity preparation when the anatomic crown is missing. **A**, Mandibular first premolar with the crown missing. **B**, An endodontic explorer fails to penetrate the calcified pulp chamber. **C**, A long-shank round bur is directed in the assumed long axis of the root. **D**, Perforation of the root wall *(arrow),* resulting from failure to consider root angulation. **E**, Palpation of the buccal root anatomy without a dental dam in place to determine root angulation. **F**, Correct bur angulation after repair of the perforation with mineral trioxide aggregate (MTA). The dental dam is placed as soon as the canal has been identified.

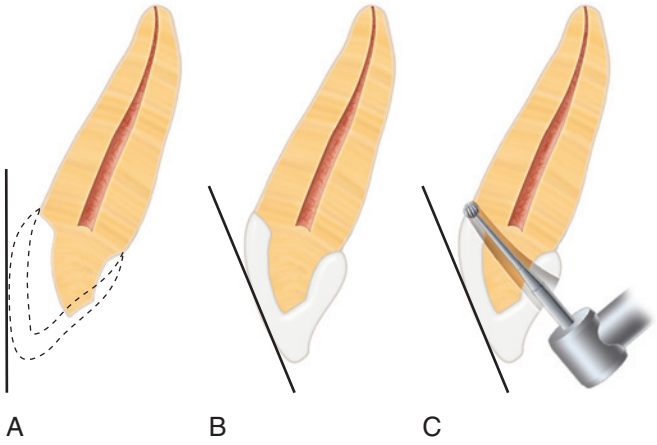

FIG. 5-58 Access cavity error resulting from alteration of the original tooth contours by a full veneer crown. **A**, Original crown contour of the tooth. **B**, A full veneer crown is used to change the original crown contour for esthetic purposes. **C**, Access perforation resulting from reliance on the full veneer crown contour rather than the long axis of the root.

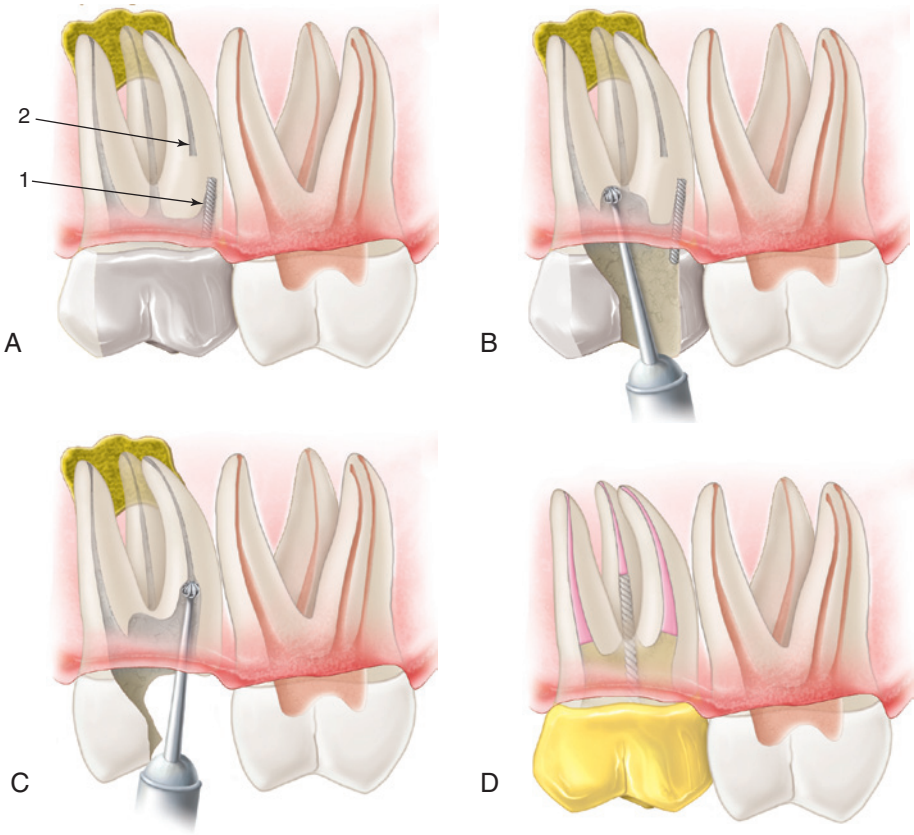

FIG. 5-59 **A,** In a heavily restored maxillary second molar that requires root canal therapy, the clinician may attempt access to the canals. Pretreatment radiographs demonstrate three important factors: (1) a reinforcing pin is in place *(arrow);* (2) at least two thirds of the coronal portion is restorative material; and (3) the mesiobuccal canal appears calcified *(arrow)*. These factors suggest complete excavation. **B,** A patient may ask the clinician to attempt an unexcavated search for the canals; this may result in a furcal perforation, compromising the prognosis. In such cases the patient should be engaged in the decision to continue treatment, which unquestionably involves removal of the existing restoration. **C,** A safer, more conservative approach is to remove the amalgam, the pin, and any old cements. Careful excavation, using enhanced vision, results in access to the pulp chamber. **D,** The clinician now can perform sound root canal therapy, followed by internal reinforcement and full coverage.

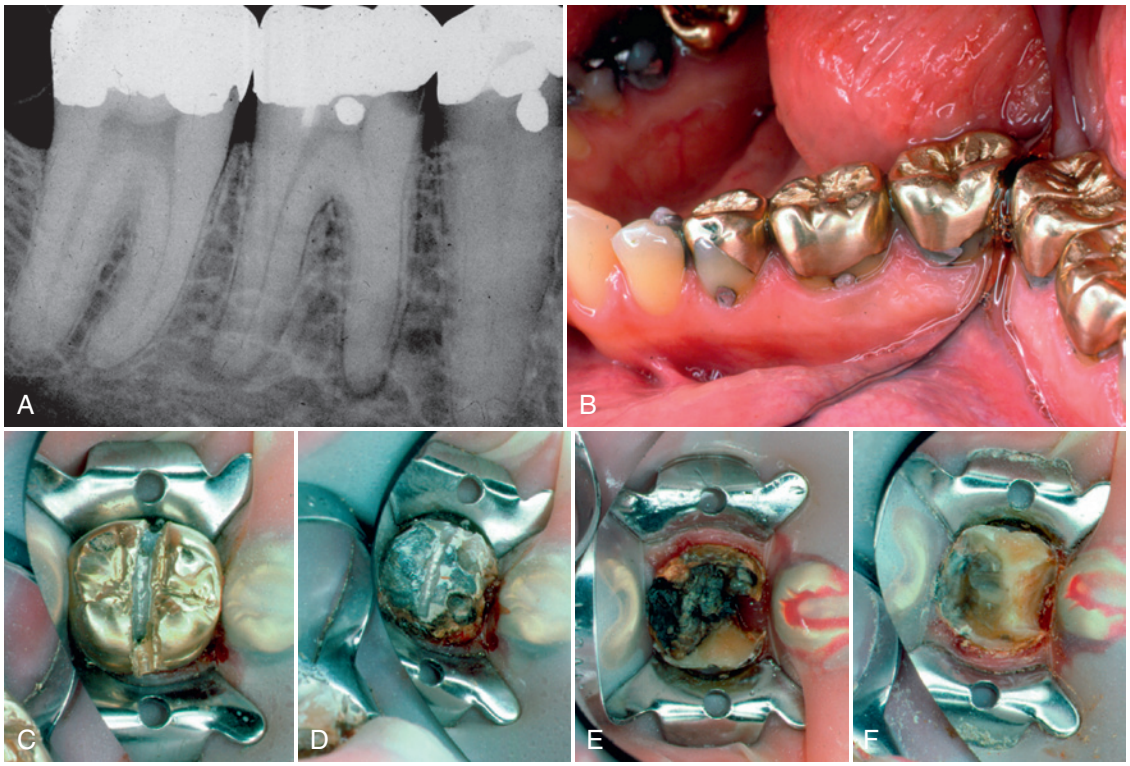

FIG. 5-60 **A**, Radiograph showing apical lesions on both roots and recurrent caries under the mesial margin of the crown. **B,** Clinical photograph of the crown and tissues that appear normal. **C,** Cutting of the crown from the tooth. **D,** Crown has been removed, and decay is evident around the core restoration. **E,** Removal of the old restoration shows significant decay. **F,** Final excavation, which allows for evaluation of the tooth structure and facilitates direct access to the pulp chamber. (From Gutmann JL, Lovdahl PE: *Problem solving in endodontics,* ed 5, St Louis, 2011, Elsevier.)

Development of an access through an intact metalloceramic crown should be done with caution. When such restorations are placed, they often change the crown-to-root angulation to correct preexisting occlusal discrepancies. Metalloceramic crowns often alter previous tooth rotation. Both these situations make the preparation of access cavities challenging. Pretreatment radiographs can be helpful, but the metal in the full veneer crown usually masks the underlying pulp chamber. In these situations the best approach is to stay as centered in the tooth as possible, using all available clinical and radiographic information. The DOM and transillumination of the CEJ are valuable aids in this process.

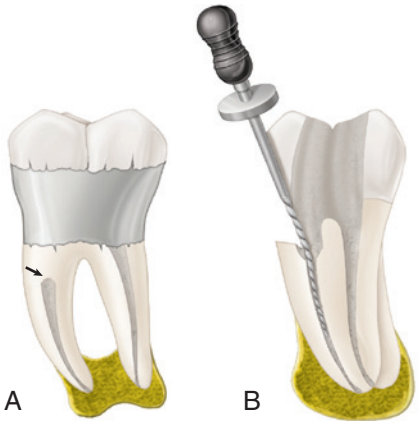

FIG. 5-61 A, Extensive class V restoration necessitated by root caries and periodontal disease that led to canal calcification *(arrow)*. B, Access to the canal is occluded by calcification. Removal of the facial restoration may be required to obtain access from the buccal surface.

Metalloceramic crowns are best penetrated with new, sharp carbide burs. Round burs work well, but tungsten carbide transmetal burs are more efficient. These crosscut fissure burs are specifically designed to cut through metal restorative materials. Porcelain or metalloceramic restorations must be handled delicately to minimize the potential for fracture (Fig. 5-62). Whatever the nature of the crown, efforts should be made to avoid being too conservative. Attempting to save the crown often leads to an underextended preparation. All the guidelines for access cavity preparations discussed earlier must be followed. When the preparation is complete, the margins and internal spaces must be evaluated for caries, leakage, and fractures.

Access in Teeth with Calcified Canals

Often a 2D pretreatment radiograph may be interpreted as showing total or nearly total calcification of the pulp chamber and radicular canal spaces (see Fig. 5-56). Unfortunately, these spaces have adequate room to allow passage of millions of microorganisms. Chronic inflammatory processes (e.g., caries, medications, occlusal trauma, and aging) often cause pulpal degeneration and concomitant narrowing of the root canal system.[84] Although the coronal portion of the canal may appear diminished significantly, canals often become less calcified as they approach the root apex. Despite these perceived 2D anatomic alterations, many canals do exist, and attempts must be made to manage them to the canal terminus.

Chambers and roots that demonstrate significant calcifications may present problems with locating, penetrating, and

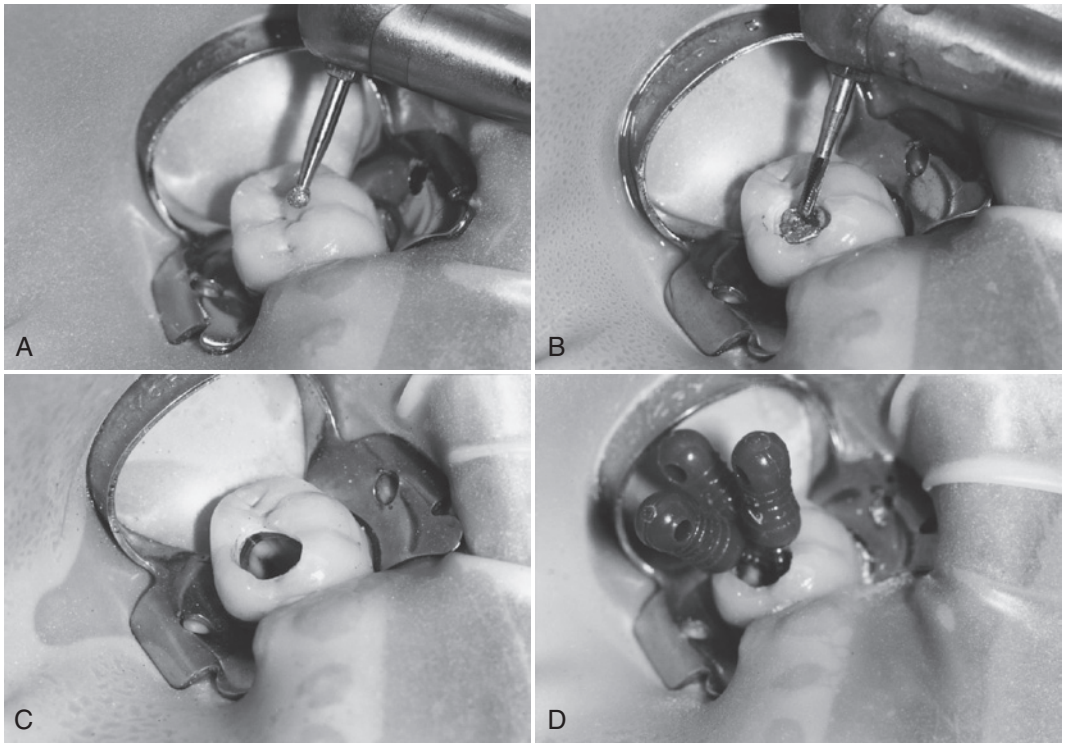

FIG. 5-62 Access cavity preparation through a metalloceramic crown. A, A round diamond bur is used to penetrate the porcelain. B, Following the access outline with the round diamond bur, a transmetal bur is used to cut through the metal. C, Prepared access cavity allowing direct approach to the canals. D, Files are placed on the access cavity walls without impingement.

FIG. 5-63 Mandibular molar with what appears to be almost complete calcification of the pulp chamber and root canals. However, pathosis is present, which indicates the presence of bacteria and some necrotic tissue in the apical portion of the roots.

negotiating the pulpal spaces.[84] The use of magnification and transillumination, as well as careful examination of color changes and pulp chamber shapes, assists in locating the canals (Fig. 5-63). However, the search for the root canal orifices should be made only after the pulp chamber has been completely prepared and its floor has been cleaned and dried (95% denatured ethanol may be useful for drying the floor and enhancing visibility). A fiberoptic light directed through the CEJ can reveal subtle landmarks and color changes that may not otherwise be visible. The chamber floor is darker in color than its walls, and developmental grooves connecting orifices are lighter in color than the chamber floor. Awareness of these color differences when searching for calcified orifices is essential, especially when searching for canal orifices that are located at the angles formed by the floor and walls and at the end points of developmental grooves. Additional methods to help locate calcified root canals include staining the pulp chamber floor with 1% methylene blue dye, performing the sodium hypochlorite "champagne bubble" test (see Fig. 5-2) and searching for canal bleeding points. These approaches are enhanced when the area is viewed through magnification.

In teeth with significant calcifications that obscure and block the root canal, the calcified material must be removed slowly down the root. Long, thin ultrasonic tips should be used under the high magnification of a DOM to avoid removing too much tooth structure. As the clinician proceeds apically, exposure of two radiographs should be considered, one from the straight-on direction and the other from an angled direction. A very small piece of lead foil placed at the apical extent of the penetration can provide a radiographic reference.

Uncovering canals that contain calcified material is a challenge. When the canal is located, a small K-file (#6, #8, or #10 or, preferably, a C or C+ file [Dentsply Tulsa and Maillefer, respectively]) coated with a chelating agent should be introduced into the canal to determine patency. These instruments provide added stiffness to the shaft for better penetration. The file should not be removed until some canal enlargement has occurred. The file should be used in short up-and-down movements and in a selective circumferential filing motion, with most of the lateral pressure directed away from the furcation. This enlarges the coronal aspect of the canal safely and moves it laterally, to avoid thinning of the dentin wall adjacent to the furcation. It also creates a path of insertion for larger files and for preflaring burs. Figs. 5-64 through 5-69 illustrate several methods that can be used to locate calcified spaces. For the most successful results, the sequence should be followed as shown.

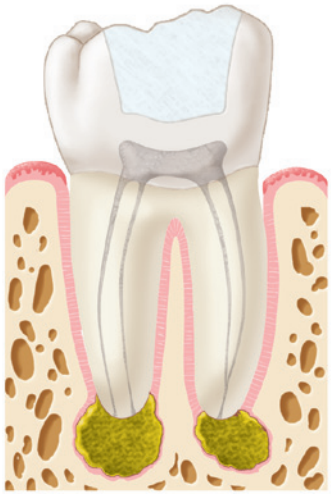

FIG. 5-64 Mandibular first molar with a class I restoration, calcified canals, and periradicular radiolucencies. Presumably a pulp exposure has occurred, resulting in calcification and ultimate necrosis of the pulp tissue.

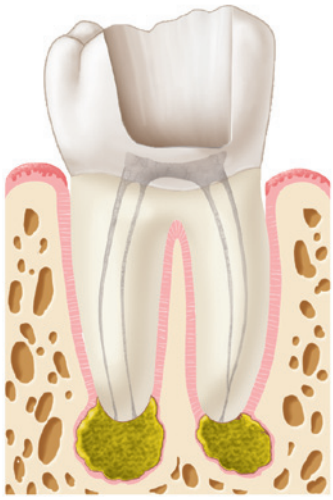

FIG. 5-65 Excavation of a restoration and base material. The cavity preparation is extended toward the assumed location of the pulp chamber, keeping in mind that pulp chambers are located in the center of the tooth at the level of the cementoenamel junction *(CEJ)*.

If the canal orifice cannot be found, it is wise to stop excavating the dentin to avoid weakening the tooth structure or perforating into the periodontal ligament. Management of these problems can be found in Chapter 19. There is no rapid technique or solution for dealing with calcified root canals.[84] Painstaking removal of small amounts of dentin with the aid of the DOM and radiographic confirmation has proved to be the safest approach.

Crowded or Rotated Teeth

Traditional access preparations may not be possible in patients with crowded teeth. The decision about an alternative approach

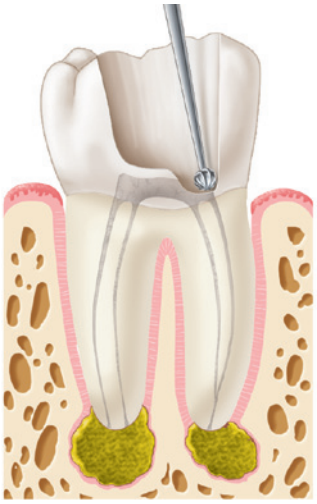

FIG. 5-66 Use a long-shank #2 or #4 round bur to remove dentin and attempt to locate calcified canals.

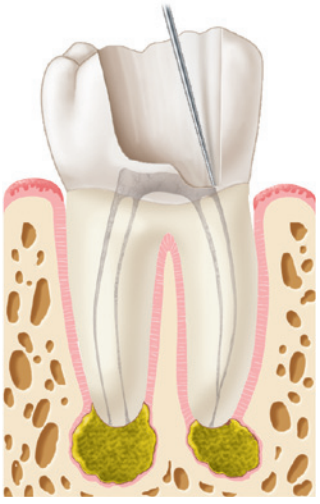

FIG. 5-67 An endodontic explorer is used to probe the pulp floor. A straight ultrasonic tip may be used to remove dentin. Angled radiographs must be taken to monitor progress.

must be based on straight-line access principles and conservation of tooth structure. In certain circumstances a buccal access preparation may be the treatment of choice (Fig. 5-70).[84] Modern restorative materials have made repair of this access esthetically acceptable.

Rotated teeth can present problems during access cavity preparation because of the altered crown-to-root relationships. According to a study by Moreinis,[144] diagnostic periapical radiographs, although only 2D, are indispensable for "determining the anatomic relationship of the crown to the root and the angle of the root in the arch." When these factors are identified, reasonable variations in the access opening must be visualized before the tooth is entered. Perforations in rotated teeth during access preparation usually occur because of faulty angulation of the bur with respect to the long axis of the root.

Other problems can occur when tooth angulations are not considered during preparation of an access cavity. Such problems include the following:

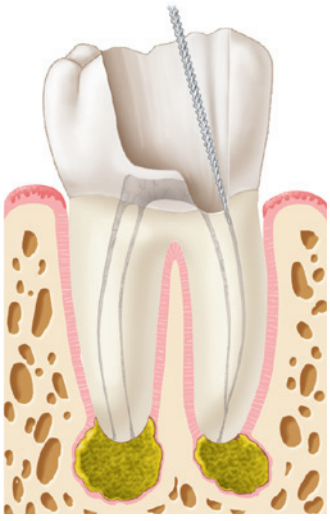

FIG. 5-68 At the first indication of a canal space, the smallest instrument (i.e., a #.06 or #.08 C or C+ file or micro openers) should be introduced into the canal. Gentle passive movement, both apical and rotational, often produces some penetration. A slight pull, signaling resistance, usually is an indication that the canal has been located. This should be confirmed by radiographs.

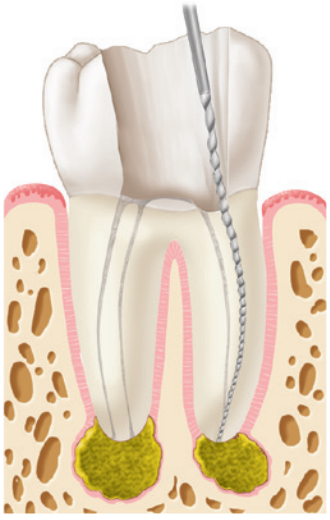

FIG. 5-69 A small K-file negotiates the canal to its terminus. An apex locator or radiograph is used to confirm the file's position.

- Mistaken identification of an already located canal, resulting in a search in the wrong direction for additional canals. Whenever a difficult canal is located, a file should be placed in the canal and an angled radiograph taken. This determines which canal has been located. A search for another canal orifice can then begin in the correct direction.
- Failure to locate a canal or extra canals.
- Excessive gouging of coronal or radicular tooth structure.
- Instrument separation during attempts to locate an orifice.
- Failure to débride all pulp tissue from the chamber.

The best way to handle any these problems is to prevent them from occurring. A thorough radiographic examination is

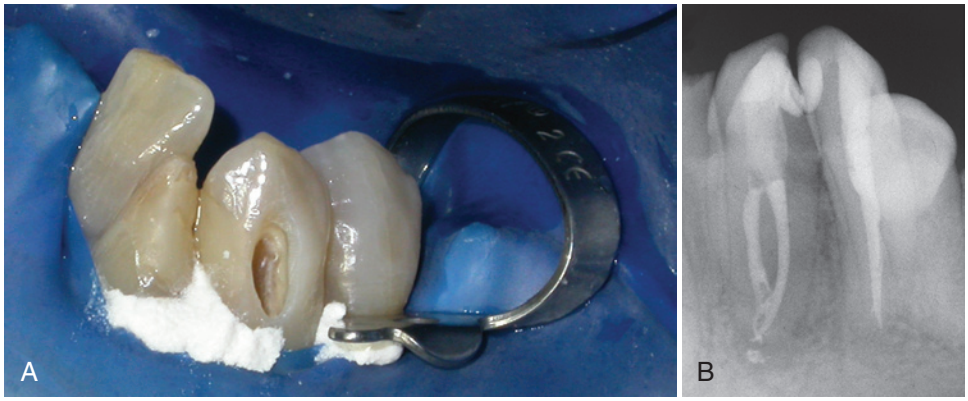

FIG. 5-70 A, Access cavity on crowded mandibular anterior teeth. The access preparation is cut through the buccal surface on the canine. The lateral incisor has also been accessed through the buccal surface; root canal procedures were performed, and the access cavity was permanently restored with composite. B, Obturation.

crucial. The initial outline form occasionally can be created without the dental dam, which facilitates positioning of the bur with the long axis of the tooth. Bur penetration for both depth and angulation should be confirmed frequently with radiographs.

ERRORS IN ACCESS CAVITY PREPARATION

Unfortunately, errors can occur in the preparation of an access cavity. Most are the result of failure to follow the access guidelines; others reflect a lack of understanding of the internal and external tooth morphology. Common errors are discussed and illustrated in Figs. 5-71 through 5-73 and detailed extensively elsewhere in this text and other publications.[84]

MORPHOLOGY OF AND ACCESS CAVITY PREPARATIONS FOR INDIVIDUAL TEETH

The anatomy shown in the following figures was obtained from human teeth through the use of recently developed 3D imaging techniques. The teeth were scanned in a high-resolution, microcomputer-assisted tomographic scanner. The data were then manipulated with proprietary computer programs to produce the 3D reconstructions and visualization. The following individuals and sources are recognized to have contributed greatly to this endeavor.

The 3D reconstructed images in this chapter were obtained from the tooth and canal morphology database at the School of Stomatology, Wuhan University, China. The database was established by Dr. Bing Fan's group and supported by the National Natural Science Foundation of China (grant no. 30572042, 30872881, 81070821) and the Key Technologies R&D Programme of Hubei Province of China (grant no. 2007AA302B06). The microcomputed tomography machine used for scanning was the μCT-50, Scanco Medical, Bassersdorf, Switzerland. The multiple softwares used for 3D reconstruction included 3D-Doctor (Able Software Corp., Lexington, Massachusetts) and VGStudio MAX (Volume Graphics GmbH, Heidelberg, Germany).

Radiographs: Courtesy Dr. L. Stephen Buchanan, Santa Barbara, California; Dr. John Khademi, Durango, Colorado; Dr. Raed S. Kasem, Clearwater, Florida; Dr. Gary Manasse, Jacksonville, Florida; Dr. Michael DeGrood, DeBary, Florida; and Dr. Kevin Melker, Clearwater, Florida.

Access cavity illustrations: Designed and formatted by Dr. Richard Burns, San Mateo, California; and Dr. Eric Herbranson, San Leandro, California.

All tables in this section that provide literature documentation for the studies on root canal anatomy are available online at the Expert Consult site and can be accessed accordingly.

Maxillary Central Incisor

The root canal system outline of the maxillary central incisor reflects the external surface outline (Fig. 5-74). A newly erupted central incisor has three pulp horns, and the pulp chamber is wider mesiodistally than buccolingually. A lingual shoulder usually is present, and it must be removed to gain access to the lingual wall of the root canal. The lingual shoulder prevents direct access to the root canal and deflects files labially, often resulting in a ledge or perforation. In cross section, the root canal at the CEJ is triangular in young teeth and oval in older teeth. It gradually becomes round as it approaches the apical foramen (see Table 5-8 online at the Expert Consult site). The μCT scans of this tooth are seen in Fig. 5-75. (See Video 5-1 online at the Expert Consult site for rotational views of these teeth.)

The external access outline form for the maxillary central incisor is a rounded triangle with its base toward the incisal aspect (Figs. 5-76 through 5-79). The width of the triangular base is determined by the distance between the mesial and distal pulp horns. The mesial and distal external walls should converge toward the cingulum. All internal walls should funnel to the root canal orifice. If the lingual shoulder has been removed properly, the entire orifice should be seen through the access opening. The incisal internal wall should approach the lingual surface of the tooth in a near butt joint to allow for a bulk of restorative material on this functional surface.

Variation

The outline form of the access cavity changes to a more oval shape as the tooth matures and the pulp horns recede because the mesial and distal pulp horns are less prominent.

Maxillary Lateral Incisor

The pulp chamber outline of the maxillary lateral incisor is similar to that of the maxillary central incisor; however, it is

TABLE 5-8

Studies of Apical Canal Configurations for the Maxillary Central Incisor

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)	Four Canals (%)
Vertucci[223]	Clearing	United States	100	—	—
Pineda and Kuttler[170]	Radiographic	Mexico	100	—	—
Calişkan et al.[33]	Clearing	Turkey	100	—	—
Kasahara et al.[101]	Clearing	Japan	—	—	—
Mangani and Ruddle[129]	Clinical	United States	—	—	Case report
Todd[218]	Clinical	United States	—	Case report	—
Genovese and Marsico[74]	Clinical	Italy	—	Case report	—
Sinai and Lustbader[194]	Clinical	United States	—	Case report	—
Von der Vyver and Traub[228]	Clinical	South Africa	—	Case report	—
Cabo-Valle and Gonzalez-Gonzalez[31]	Clinical	Spain	—	Case report	—
Mader and Konzelman[127]	Clinical	United States	—	Case report	—
Lambruschini and Camps[111]	Clinical	France	—	Case report	—
Gonzalez-Plata and Gonzalez-Plata[78]	Clinical	Mexico	—	Case report	—
Patterson[160]	Clinical	United States	—	Case report	—
Heling[88]	Clinical	Israel	—	Case report	—
Al-Nazhan[7]	Clinical	Saudi Arabia	—	Case report	—
Lin et al.[119]	Clinical	China	—	Case report	—
Sponchiado et al.[204]	Clinical	Brazil	—	Case report	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	98 (98) 100 (98)	2 (2) 2 (2)	— —

*Number in parentheses is the percentage of cases with two canals joining into one canal.

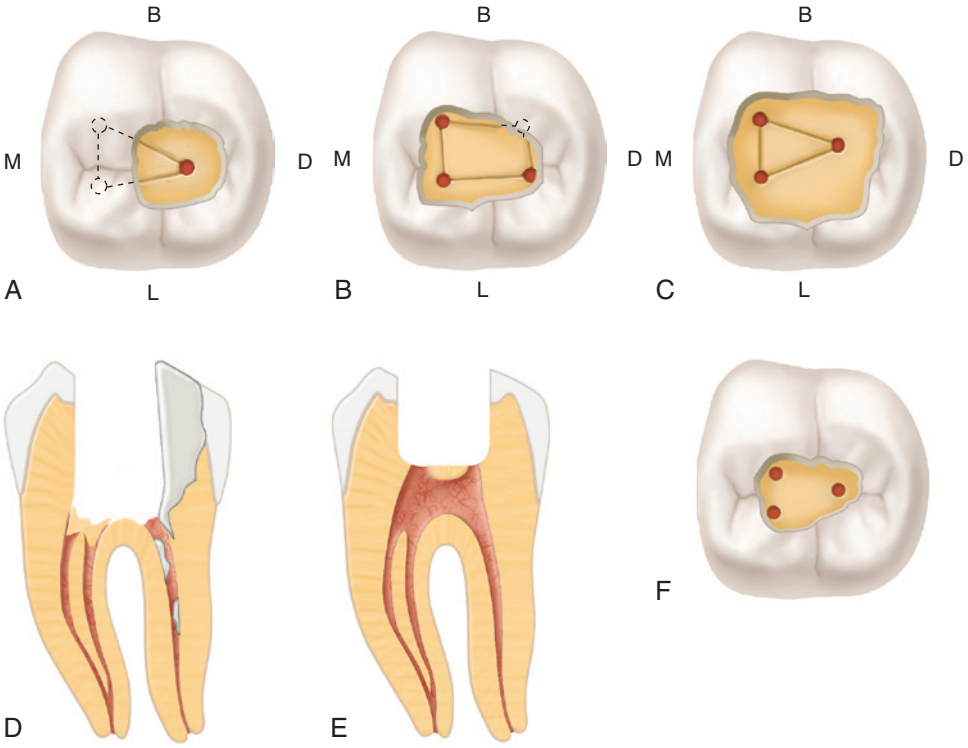

FIG. 5-71 **A,** Poor access placement and inadequate mesial extension leave both mesial orifices uncovered. Information about the position and location of pulp chambers can be obtained through evaluation of pretreatment radiographs, especially bite-wing radiographs, and assessment of the tooth anatomy at the cementoenamel junction (CEJ). **B,** Inadequate extension of the distal access cavity leaves the distobuccal canal orifice unexposed. All developmental grooves must be traced to their termination and must not be allowed to disappear into an axial wall. **C,** Gross overextension of the access cavity weakens the coronal tooth structure and compromises the final restoration. This mistake results from failure to determine correctly the position of the pulp chamber and the angulation of the bur. **D,** Allowing debris to fall into canal orifices results in an iatrogenic mishap. Amalgam fillings and dentin debris block canal orifices, preventing proper shaping and cleaning. Complete removal of the restoration and copious irrigation help prevent this problem. **E,** Failure to remove the roof of the pulp chamber is a serious underextension error; the pulp horns have been exposed. Bite-wing radiographs are excellent aids in determining vertical depth. **F,** Access preparation in which the roof of the pulp chamber remains and the pulp horns have been mistaken for canal orifices. The whitish color of the roof, the depth of the access cavity, and the lack of developmental grooves are clues to this underextension. Root canal orifices generally are positioned at or slightly apical to the CEJ. *B,* Buccal; *D,* distal; *L,* lingual; *M,* mesial.

smaller, and two or no pulp horns may be present (Fig. 5-80). This tooth is wider mesiodistally than buccolingually. A cross section at the CEJ shows a pulp chamber centered in the root, and its shape may be triangular, oval, or round. From the CEJ the pulp canal becomes round in cross section in the midroot and apical areas. The lingual shoulder of dentin must be removed before instruments can be used to explore the canal (see Table 5-9 online at the Expert Consult site). The μCT scans of this tooth are shown in Fig. 5-81. (See Video 5-2 online at the Expert Consult site for rotational views of these teeth.) Normally only one root canal is present, but other variations have been reported.

The external access outline form for the maxillary lateral incisor may be a rounded triangle or an oval, depending on the prominence of the mesial and distal pulp horns (Figs. 5-82 through 5-84). When the horns are prominent, the rounded triangular shape is compressed mesiodistally relative to a central incisor, producing a more slender triangle. The outline form usually is oval if the mesial and distal pulp horns are not prominent. All other aspects of the access preparation are the same as those for the central incisor.

The maxillary lateral incisor often has anomalies. One such variation in form is the presence of a palatal radicular or developmental groove (Fig. 5-85).[121,153-155,161-164] Although this groove may be present on the roots of all anterior teeth, it is more common in the maxillary lateral incisor. There is generally direct communication between the groove and the pulp cavity, and this occurs primarily through dentinal tubules.

Dens invaginatus, another anomaly, has been classified into three types based on severity, ranging from simple to more complex.[194] Type 1 is an invagination that is confined to the crown. Type 2 is an invagination that extends past the CEJ but does not involve the periradicular tissues. Type 3 is an invagination that extends beyond the CEJ and can have a second apical foramen. Often both surgical and nonsurgical root canal procedures are necessary to properly manage this condition.*

*References 155, 167, 170, 178, 179, 181, 182, 188, 195, and 208.

TABLE 5-9

Studies of Apical Canal Configurations for the Maxillary Lateral Incisor

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)
Vertucci[223]	Clearing	United States	100	—	—
Pineda and Kuttler[170]	Radiographic	Mexico	100	—	—
Calişkan et al.[33]	Clearing	Turkey	95.1	4.9	—
Pecora et al.[165]	Clinical	Brazil	—	Case report	—
Thompson et al.[216]	Clinical	United States	—	Case report (1)	—
Fabra-Campos[60]	Clinical	Spain	—	Case report	—
Christie et al.[38]	Clinical	Canada	—	Case report	—
Walvekar and Behbehani[233]	Clinical	Kuwait	—	—	Case report
Collins[42]	Clinical	Australia	—	Case report	—
Sert and Bayirli[190]	Clearing (men)	Turkey	97 (6)	(3)	—
	Clearing (women)	Turkey	100 (10)	—	—

*Numbers in parentheses are the percentage of cases with two canals joining into one canal.
†Number in parentheses is the percentage of cases with one canal dividing into two canals.

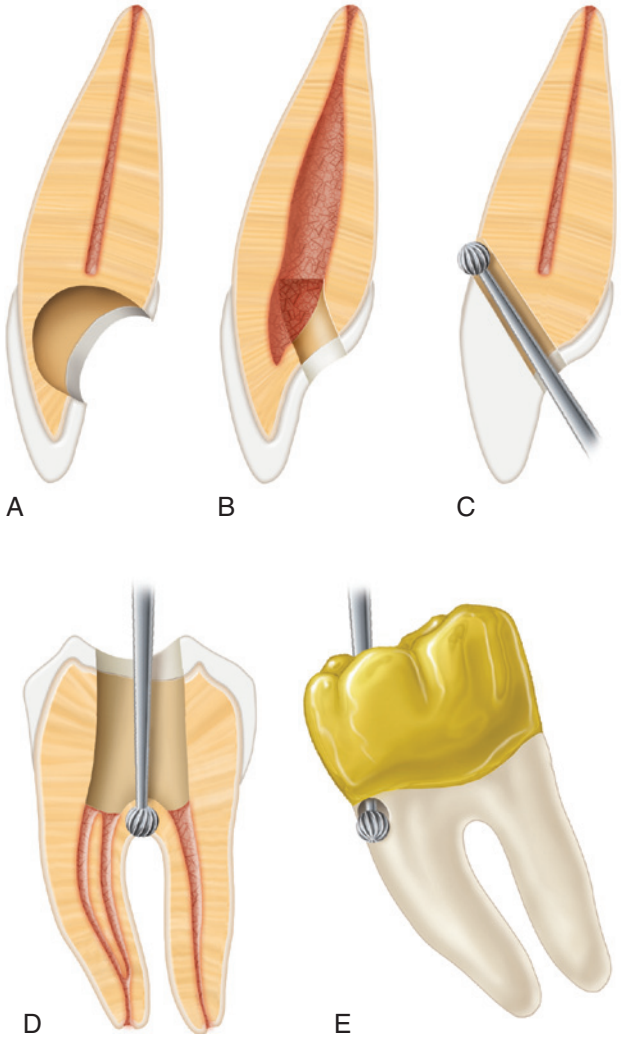

FIG. 5-72 A, Overzealous tooth removal caused by improper bur angulation and failure to recognize the lingual inclination of the tooth. This results in weakening and mutilation of the coronal tooth structure, which often leads to coronal fractures. B, Inadequate opening; the access cavity is positioned too far to the gingival, with no incisal extension. This can lead to bur and file breakage, coronal discoloration because the pulp horns remain, inadequate instrumentation and obturation, root perforation, canal ledging, and apical transportation. C, Labial perforation caused by failure to extend the preparation to the incisal before the bur shaft entered the access cavity. D, Furcation perforation caused by failure to measure the distance between the occlusal surface and the furcation. The bur bypasses the pulp chamber and creates an opening into the periodontal tissues. Perforations weaken the tooth and cause periodontal destruction. To ensure a satisfactory result, they must be repaired as soon as they are made. E, Perforation of the mesial tooth surface caused by failure to recognize that the tooth was tipped and failure to align the bur with the long axis of the tooth. This is a common error in teeth with full crowns. Even when these perforations are repaired correctly, they usually cause a permanent periodontal problem because they occur in a difficult maintenance area.

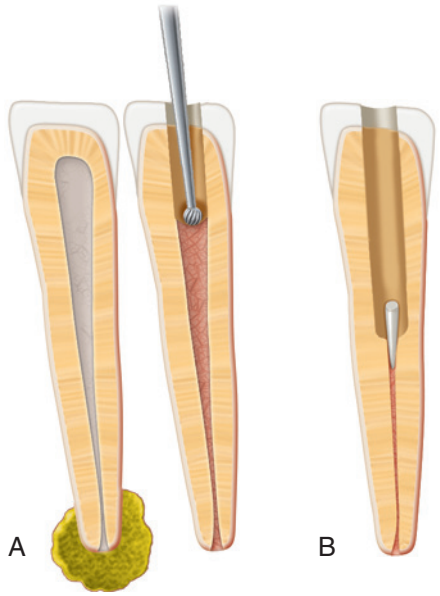

FIG. 5-73 A, The most embarrassing error, with the greatest potential for medical and legal damage, is entering the wrong tooth because of incorrect dental dam placement. When the crowns of teeth appear identical, the clinician should mark the tooth with a felt-tip marker before placing the dental dam. B, Burs and files can be broken if used with an improper motion, excessive pressure, or before the access cavity has been properly prepared. A broken instrument may lock into the canal walls, requiring excessive removal of tooth structure to retrieve it. On occasion, fragments may not be retrievable.

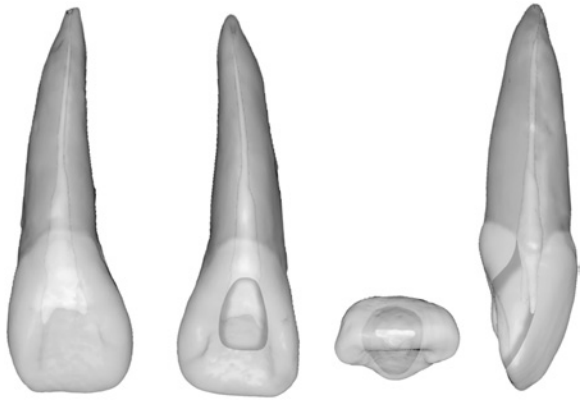

FIG. 5-74 Maxillary central incisor. Average time of eruption—7 to 8 years; average age of calcification—10 years; average length—22.5 mm. Root curvature (most common to least common): straight, labial, distal.

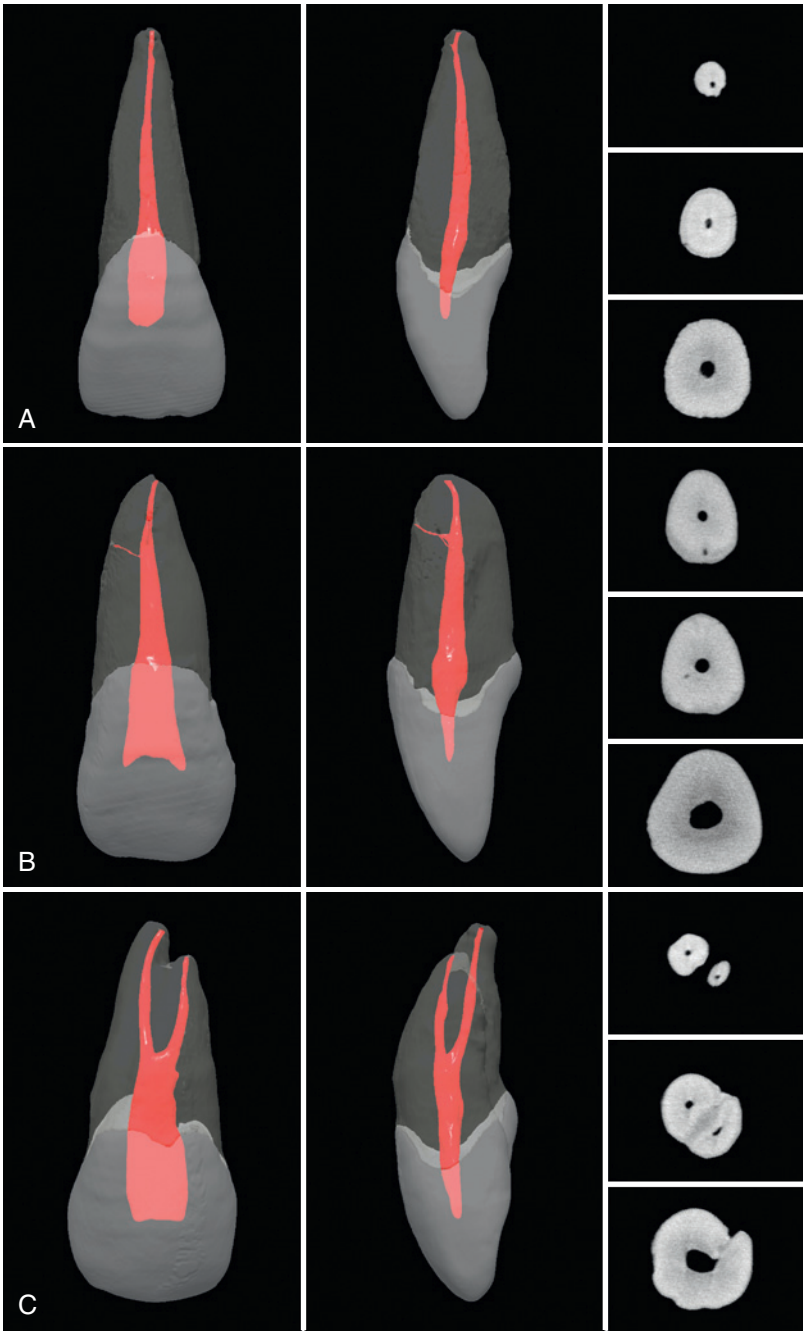

FIG. 5-75 μCT scans of maxillary central incisors. **A**, Common anatomic presentation. **B**, Central incisor with a lateral canal, which is common. **C**, Rare multiple-canal variation. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-1 online at the Expert Consult site for rotational views of these teeth.)

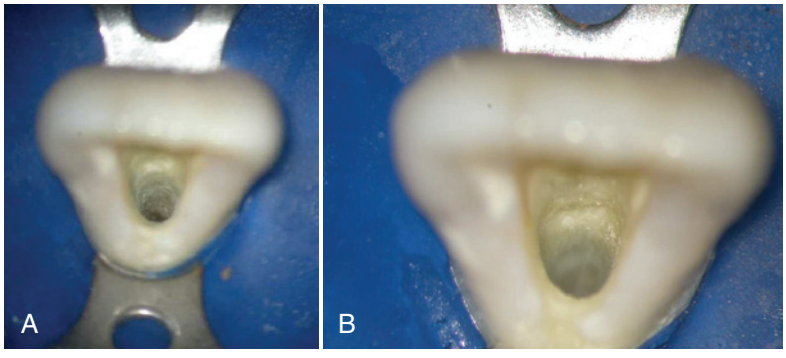

FIG. 5-76 Access cavity for a maxillary central incisor as viewed through the dental operating microscope. **A**, ×3.4 magnification. **B**, ×8.4 magnification.

FIG. 5-77 Curved accessory canal with intersecting straight lateral canal.

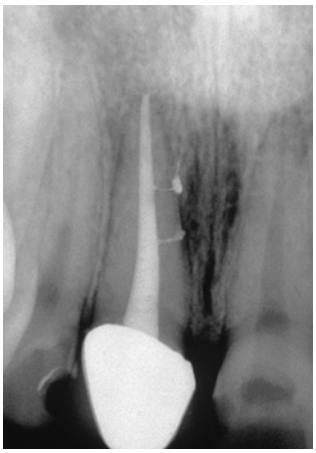

FIG. 5-79 Double lateral canals.

FIG. 5-78 Parallel accessory canal to main canal with a simple lateral canal.

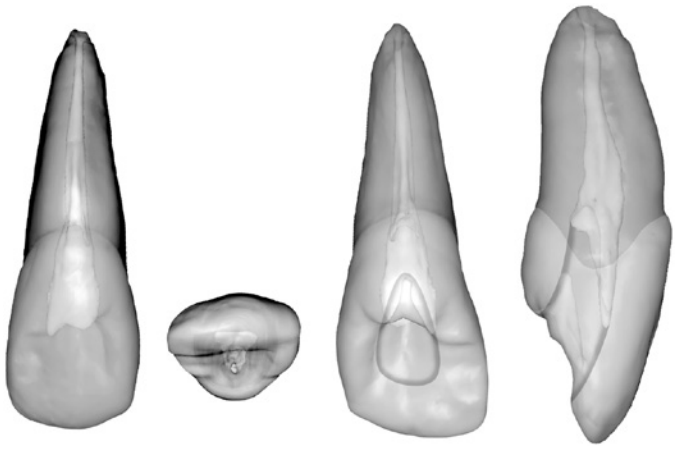

FIG. 5-80 Maxillary lateral incisor. Average time of eruption—8 to 9 years; average age of calcification—11 years; average length—22 mm. Root curvature (most common to least common): distal, straight.

Maxillary Canine

The root canal system of the maxillary canine is similar in many ways to that of the maxillary incisors (Fig. 5-86). A major difference is that it is wider labiolingually than mesiodistally. Another difference is that it has no pulp horns. Its smallest pointed incisal edge corresponds to the single cusp. The pulp chamber outline at the CEJ is oval. A lingual shoulder is present, which may prevent shaping and cleaning of the root canal in its lingual dimension. From this point, the root canal remains oval until it approaches the apical third of the root, where it becomes constricted. Because of this oval shape, the clinician must take care to circumferentially file labially and palatally to shape and clean the canal properly.[124] Usually one root canal is present, although two canals have been reported (see Table 5-10 online at the Expert Consult site). The μCT scans for the maxillary canine can be seen in Fig. 5-87. (See Video 5-3 online at the Expert Consult site) for rotational views of these teeth. The thin buccal bone over the canine eminence often disintegrates, and fenestration is an occasional anatomic finding.

The external access outline form is oval or slot shaped because no mesial or distal pulp horns are present (Figs. 5-88 through 5-90). The mesiodistal width of the slot is determined by the mesiodistal width of the pulp chamber. The incisogingival dimension is determined by straight-line access factors and removal of the lingual shoulder. The incisal extension often approaches to within 2 to 3 mm of the incisal edge to allow for straight-line access. The incisal wall meets the lingual surface of the canine in a butt joint to provide adequate thickness for restorative material because this tooth is heavily involved in excursive occlusal guidance and function. All internal walls funnel to the orifice.

Maxillary First Premolar

Most maxillary first premolars have two root canals, regardless of the number of roots (Fig. 5-91). Ethnicity plays a factor in that Asian people have a higher incidence of one canal than do other ethnic groups.[120,156]

A furcation groove or developmental depression on the palatal aspect of the buccal root is another anatomic feature. Its prevalence has been reported as 62% to 100%.[97,112,211] This groove may pose a risk to root canal and restorative procedures in this tooth.[118] At the deepest part of the invagination, the average dentin thickness was found to be 0.81 mm.

The pulp chamber of the maxillary first premolar is considerably wider buccolingually than mesiodistally. In the buccolingual dimension, the chamber outline shows a buccal and a

TABLE 5-10

Studies of Apical Canal Configurations for the Maxillary Canine

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)
Vertucci[223]	Clearing	United States	100	—
Pineda and Kuttler[170]	Radiographic	Mexico	100	—
Calişkan et al.[33]	Clearing	Turkey	97.8	2.2
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	98 (7) 96	2 4
Weisman[243]	Clinical	Australia	—	Case report
Alapati et al.[4]	Clinical	Kuwait	—	Case report

*Number in parentheses is the percentage of cases with two canals joining into one canal.

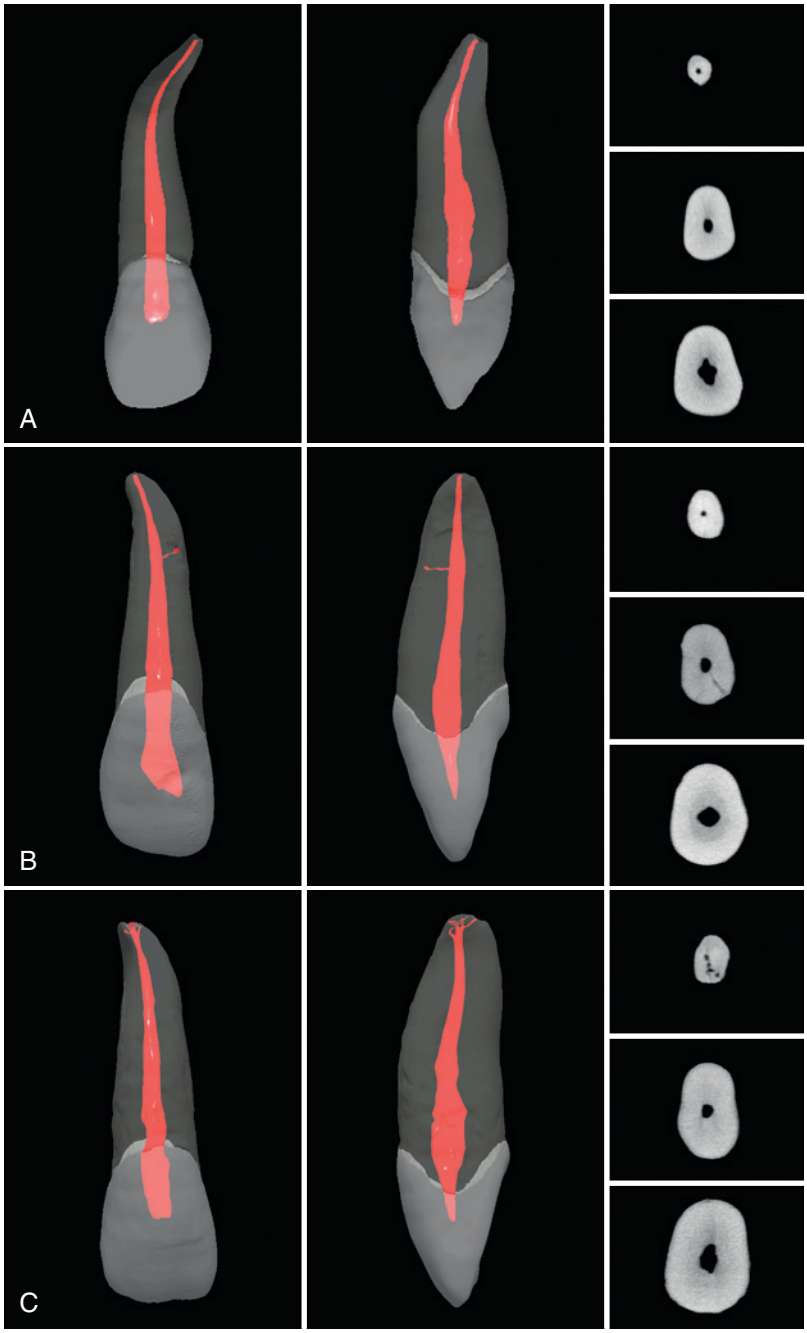

FIG. 5-81 μCT scans of maxillary lateral incisors. **A,** Common anatomic presentation. **B,** Lateral incisor with a large lateral canal, which is common. **C,** Lateral incisor with an apical delta. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-2 online at the Expert Consult site for rotational views of these teeth.)

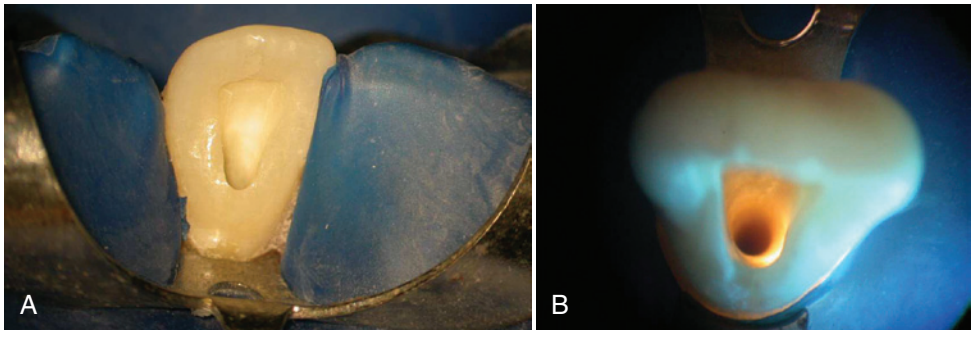

FIG. 5-82 Access cavity for a maxillary lateral incisor as viewed through the dental operating microscope. **A,** ×3.4 magnification. **B,** ×5.1 magnification with cervical fiberoptic transillumination.

FIG. 5-83 Lateral incisor with a canal loop and multiple lateral canals with associated lesions.

FIG. 5-85 Cadaver specimen showing a lingual/palatal groove.

FIG. 5-84 Multiple accessory foramina.

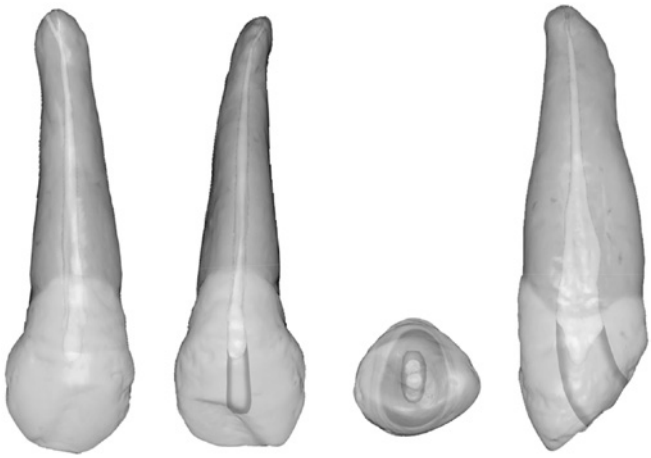

FIG. 5-86 Maxillary canine. Average time of eruption—10 to 12 years; average age of calcification—13 to 15 years; average length—26.5 mm. Root curvature (most common to least common): distal, straight, labial.

palatal pulp horn. The buccal pulp horn usually is larger. From the occlusal level, the chamber maintains a width similar that of the floor, which is located just apical to the cervical line. The palatal orifice is slightly larger than the buccal orifice. In cross section at the CEJ, the palatal orifice is wider buccolingually and kidney shaped because of its mesial concavity. From the floor, two root canals take on a round shape at midroot and rapidly taper to their apices, usually ending in extremely narrow, curved root canals. The palatal canal usually is slightly larger than the buccal canal. The maxillary first premolar may have one, two, or three roots and canals; it most often has two (see Table 5-11 online at the Expert Consult site). The μCT scans for the maxillary first premolar can be seen in Fig. 5-92. (See Video 5-4 available online at the Expert Consult site for rotational views of these teeth.)

If two canals are present, they are labeled buccal and palatal; three root canals are designated mesiobuccal, distobuccal, and palatal. Directional positioning of small files can help identify the anatomy. The roots are considerably shorter and thinner than in the canines. In double-rooted teeth, the roots most often are the same length. The buccal root is often found to be fenestrated through the cortical plate of bone; this can pose both nonsurgical and surgical treatment challenges.

The access preparation for the maxillary first premolar is oval or slot shaped (Figs. 5-93 through 5-96). It also is wide buccolingually, narrow mesiodistally, and centered mesiodistally between the cusp tips. In fact, the mesiodistal width should correspond to the mesiodistal width of the pulp chamber. The buccal extension typically is two thirds to three fourths up the buccal cusp incline. The palatal extension is approximately halfway up the palatal cusp incline. The buccal and palatal walls funnel directly into the orifices. Because of the mesial concavity of the root, the clinician must take care

TABLE 5-11

Studies of Apical Canal Configurations for the Maxillary First Premolar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)‡
Vertucci[223]	Clearing	United States	26	69	5
Pineda and Kuttler[170]	Radiographic	Mexico	50.1	49.4	0.5
Calişkan et al.[33]	Clearing	Turkey	9.8	90.2	—
Carns and Skidmore[34]	Plastic cast resin	United States	22	72	6
Walker[229]	Radiographic	Southern China	36	64	—
Kartal et al.[99]	Clearing	Turkey	9.7	88.6	1.7
Pecora et al.[164]	Clearing	Brazil	17.1	80.4	2.5
Soares and Leonardo[201]	Clinical	Brazil	—	—	Case report
Kerekes and Tronstad[104]	Ground sections	Norway	10	80	2
Green[81]	Ground sections	United States	3 (26)	70 (4)	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	31 (19) 29 (17)	68 (5) 67 (4)	4 (2) 4
Zaatar et al.[260]	Clearing	Kuwait	21.5 (10.1)	74.7	3.8

*Numbers in parentheses are the percentage of cases with two canals joining into one canal.
†Numbers in parentheses are the percentage of cases with one canal dividing into two canals.
‡Number in parentheses is the percentage of cases with one canal dividing into three canals.

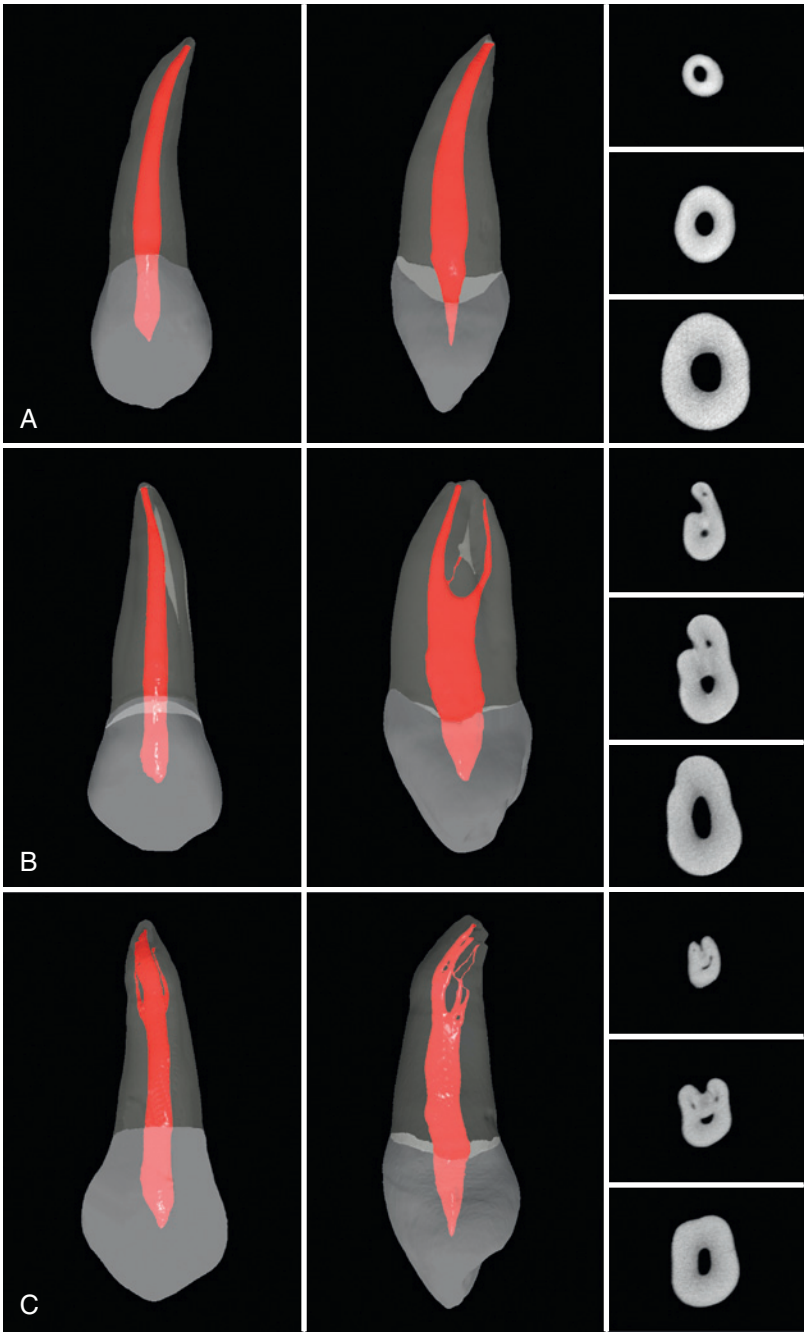

FIG. 5-87 μCT scans for the maxillary canine. **A**, Common anatomic presentation. **B**, Canine with two roots. **C**, Canine with significant deviations of the canal system in the apical thirdAll teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-3 online at the Expert Consult site for rotational views of these teeth.)

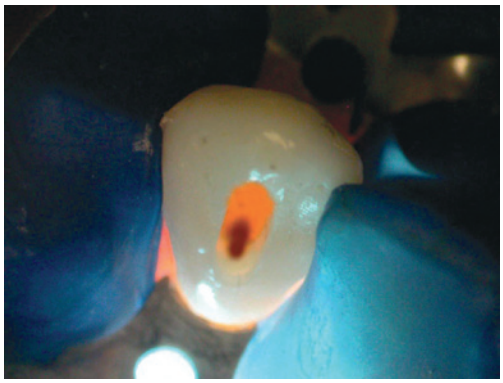

FIG. 5-88 Access cavity for a maxillary canine as viewed through the dental operating microscope. (×5.1 magnification with cervical fiberoptic transillumination.)

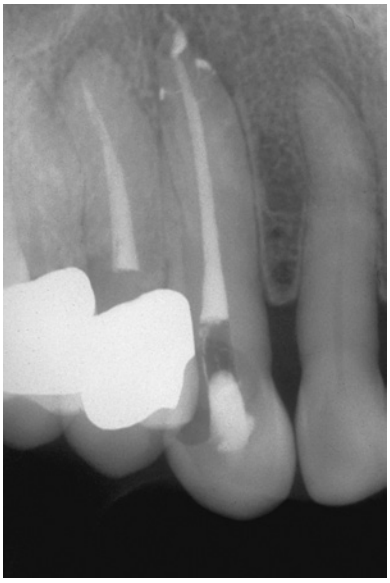

FIG. 5-89 Canine with multiple accessory foramina.

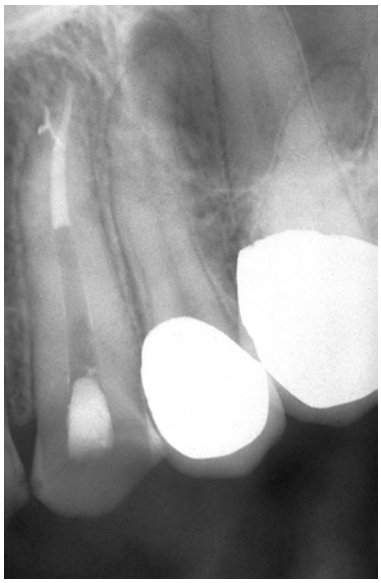

FIG. 5-90 Canine with lateral canal dividing into two canals.

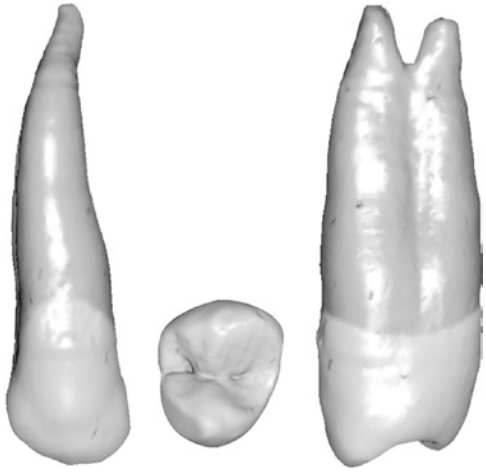

FIG. 5-91 Maxillary first premolar. Development and anatomic data: average time of eruption—10 to 11 years; average age of calcification—12 to 13 years; average length—20.6 mm. Root curvature (most common to least common): buccal root—lingual, straight, buccal; palatal root—straight, buccal, distal; single root—straight, distal, buccal.

not to overextend the preparation in that direction because this could result in perforation.

Variation

When three canals are present, the external outline form becomes triangular, with the base on the buccal aspect. The mesiobuccal and distobuccal corners of the triangle should be positioned directly over the corresponding canal orifices.

Maxillary Second Premolar

The root canal system of the maxillary second premolar is wider buccolingually than mesiodistally (Fig. 5-97). This tooth may have one, two, or three roots and canals (see Table 5-12 online at the Expert Consult site). Two or three canals can occur in a single root.[68] The scans for the maxillary second premolar can be seen in Fig. 5-98. (See Video 5-5 online at the Expert Consult site for rotational views of these teeth.) Directional positioning of small files can help identify the anatomy. The mesiodistal and buccolingual aspects of the pulp chamber are similar to those of the first premolar. A buccal and a palatal pulp horn are present; the buccal pulp horn is larger. A single root is oval and wider buccolingually than mesiodistally. The canal or canals remain oval from the pulp chamber floor and taper rapidly to the apex. The roots of the maxillary second premolar are approximately as long as those of the first premolar, and apical curvature is common, particularly with large maxillary sinus cavities.

When two canals are present in this tooth, the maxillary second premolar access preparation is nearly identical to that of the first premolar. Because this tooth usually has one root, if two canals are present, they are nearly parallel to each other, and the external outline form must have a greater buccolingual extension, to permit straight-line access to these canals, than with the first premolar with two roots and diverging canals. If only one canal is present, the buccolingual extension is less

TABLE 5-12

Studies of Apical Canal Configurations for the Maxillary Second Premolar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)‡
Vertucci[223]	Clearing	United States	75	24	1
Pineda and Kuttler[170]	Radiographic	Mexico	81.8	18.2	—
Calişkan et al.[33]	Clearing	Turkey	72	28	—
Kartal et al.[99]	Clearing	Turkey	55	44.3	0.7
Pecora et al.[163]	Clearing	Brazil	67.3	32.4	0.3
Soares and Leonardo[201]	Clinical	Brazil	—	—	Case report
Ferreira et al.[68]	Clinical	Brazil	—	—	Case report
Barkhordar and Sapone[17]	Clinical	United States	—	—	Case report
Low[121]	Clinical	Hong Kong	—	—	Case report
Green[81]	Ground sections	United States	96 (24)	4 (4)	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	49 (23) 75 (37)	46 (12) 21 (4)	5 (2) 4 (1)
Zaatar et al.[260]	Clearing	Kuwait	69.9 (15.5)	29.1	1
Awawdeh et al.[11]	Clearing	Jordan	13.8 (10.5)	84.1 (4.3)	1.5

*Numbers in parentheses are the percentage of cases with two canals joining into one canal.
†Numbers in parentheses are the percentage of cases with one canal dividing into two canals.
‡Numbers in parentheses are the percentage of cases with one canal dividing into three canals.

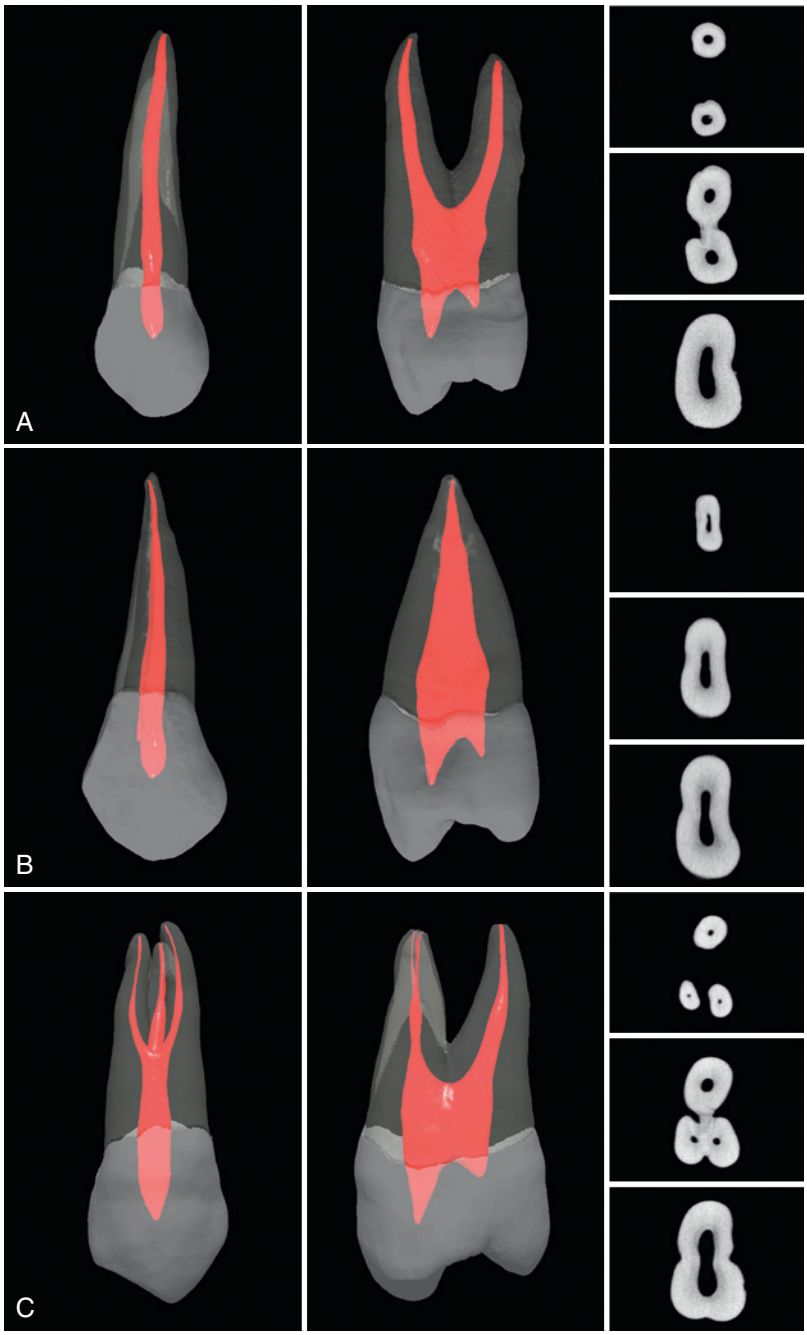

FIG. 5-92 μCT scans of maxillary first premolars. **A**, Common anatomic presentation of this tooth showing two roots. **B**, Premolar with only one canal. **C**, Premolar with three roots. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-4 online at the Expert Consult site for rotational views of these teeth.)

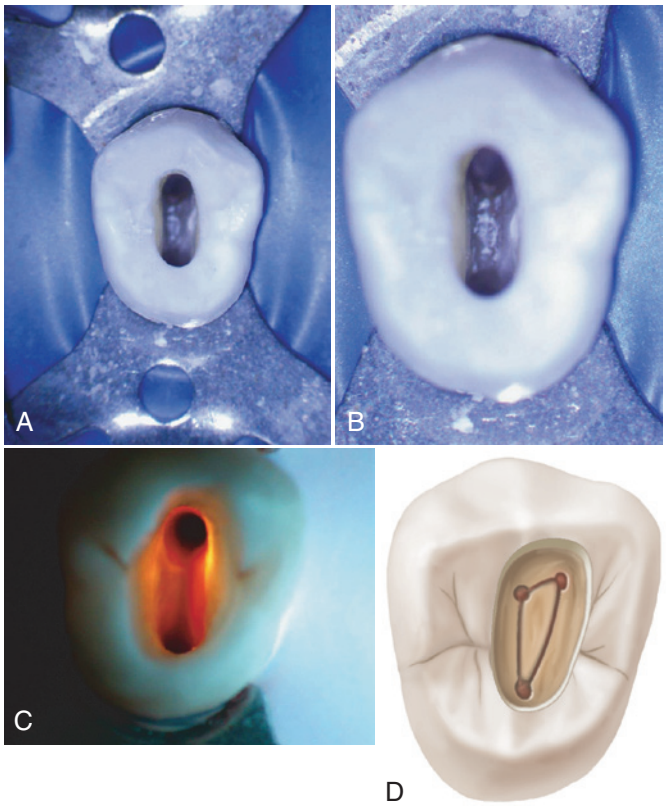

FIG. 5-93 Access cavity for a maxillary first premolar as viewed through the dental operating microscope. **A**, ×3.4 magnification. **B**, ×5.1 magnification. **C**, ×8.4 magnification with cervical fiberoptic transillumination. **D**, Schematic representation of a three-canal access preparation.

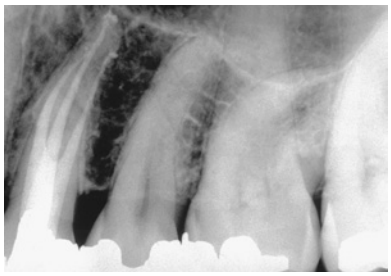

FIG. 5-96 Three canals.

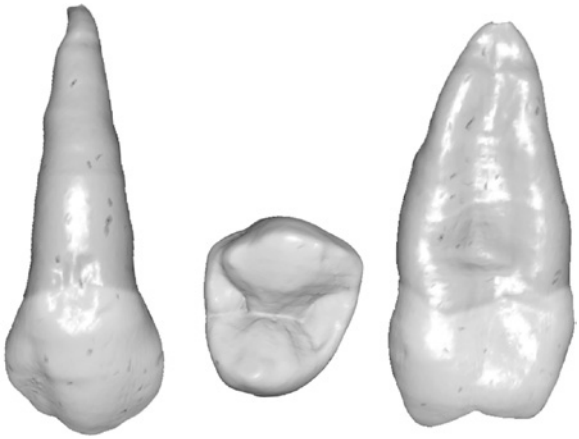

FIG. 5-97 Maxillary second premolar. Average time of eruption—10 to 12 years; average age of calcification—12 to 14 years; average length—21.5 mm. Root curvature (most common to least common): distal, bayonet, buccal, straight.

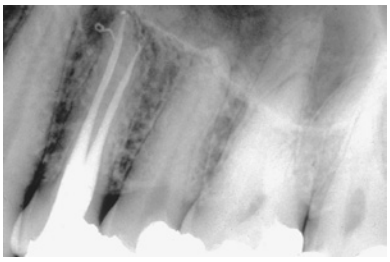

FIG. 5-94 Lateral bony lesion associated with a filled lateral canal.

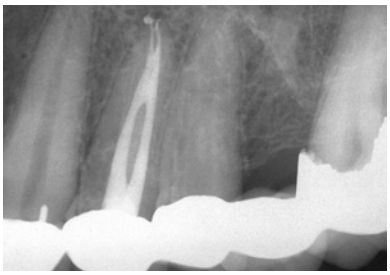

FIG. 5-95 Two canals that have fused and then redivided.

and corresponds to the width between the buccal and palatal pulp horns (Figs. 5-99 through 5-102). If three canals are present, the external access outline form is the same triangular shape illustrated for the maxillary first premolar.

Maxillary First Molar

The maxillary first molar is the largest tooth in volume and one of the most complex in root and canal anatomy (Fig. 5-103).[50] The pulp chamber is widest in the buccolingual dimension, and four pulp horns are present (mesiobuccal, mesiopalatal, distobuccal, and distopalatal). The pulp chamber's cervical outline form has a rhomboid shape, sometimes with rounded corners. The mesiobuccal angle is an acute angle; the distobuccal angle is an obtuse angle; and the palatal angles are basically right angles. The palatal canal orifice is centered palatally; the distobuccal orifice is near the obtuse angle of the pulp chamber floor; and the main mesiobuccal canal orifice is buccal and mesial to the distobuccal orifice and is positioned within the acute angle of the pulp chamber. The mesiopalatal canal orifice (also referred to as the *MB-2*) is located palatal and mesial to the mesiobuccal orifice. A line drawn to connect the three main canal orifices—the mesiobuccal orifice, distobuccal orifice, and palatal orifice—forms a triangle, known as the *molar triangle*.

The three individual roots of the maxillary first molar (i.e., mesiobuccal root, distobuccal root, and palatal root) form a tripod. The palatal root is the longest, has the largest diameter, and generally offers the easiest access. It can contain one, two,

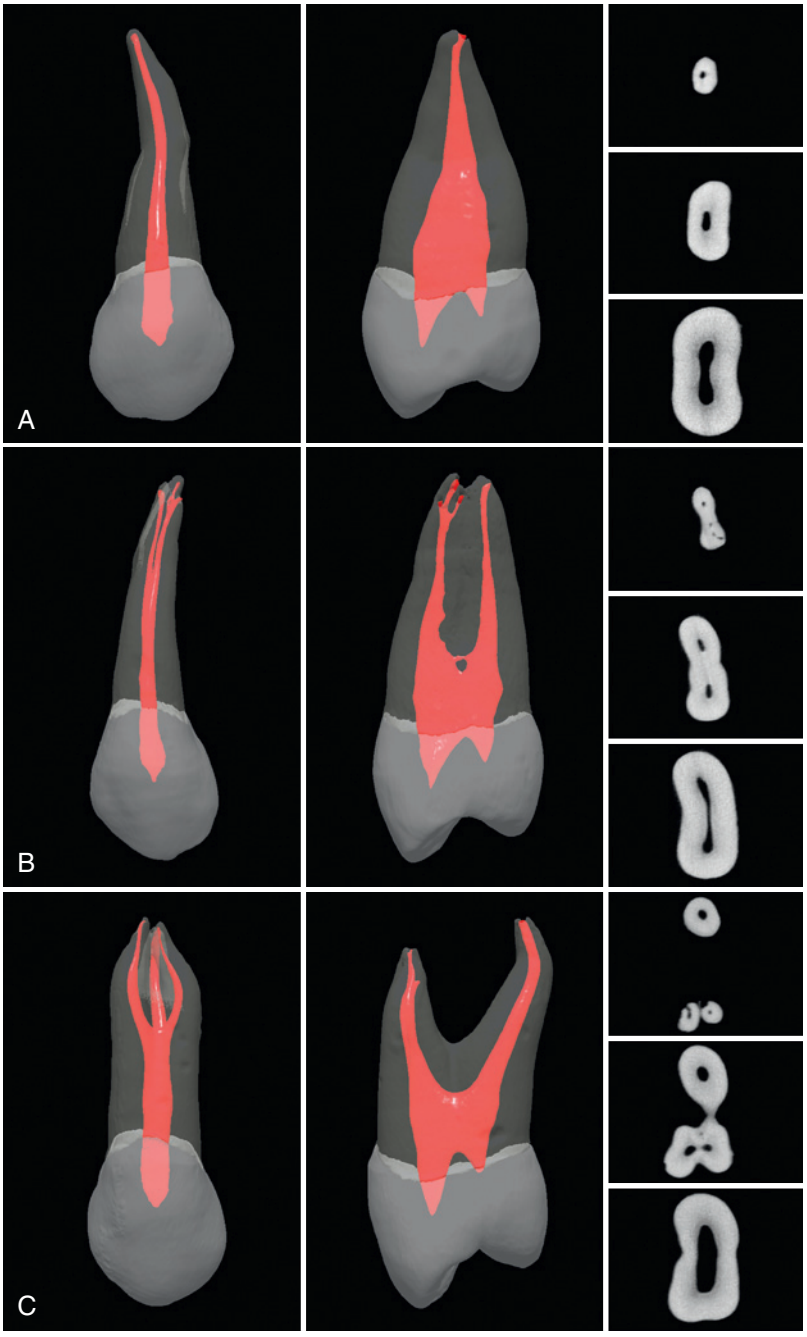

FIG. 5-98 μCT scans of maxillary second premolars. A, Common anatomic presentation showing one canal. B, Second premolar with two canals and an apical delta. C, Second premolar with three roots/canals that divide at the junction of the middle and apical third of the main root. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-5 online at the Expert Consult site for rotational views of these teeth.)

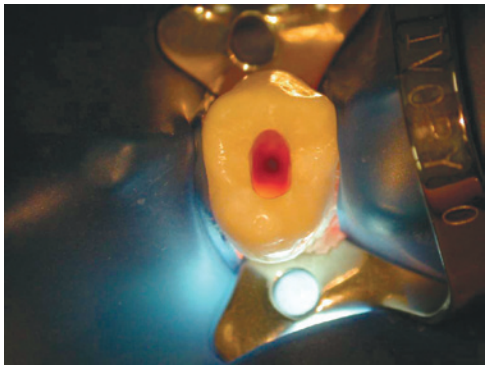

FIG. 5-99 Access cavity for a maxillary second premolar as viewed through the dental operating microscope. (×5.1 magnification with cervical fiberoptic transillumination.)

FIG. 5-100 Second premolar with three canals and a large lateral canal.

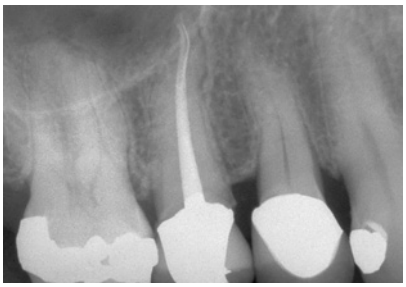

FIG. 5-101 Single canal that has divided into two canals.

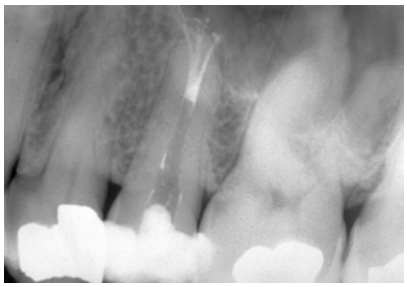

FIG. 5-102 Single canal that has split into three canals.

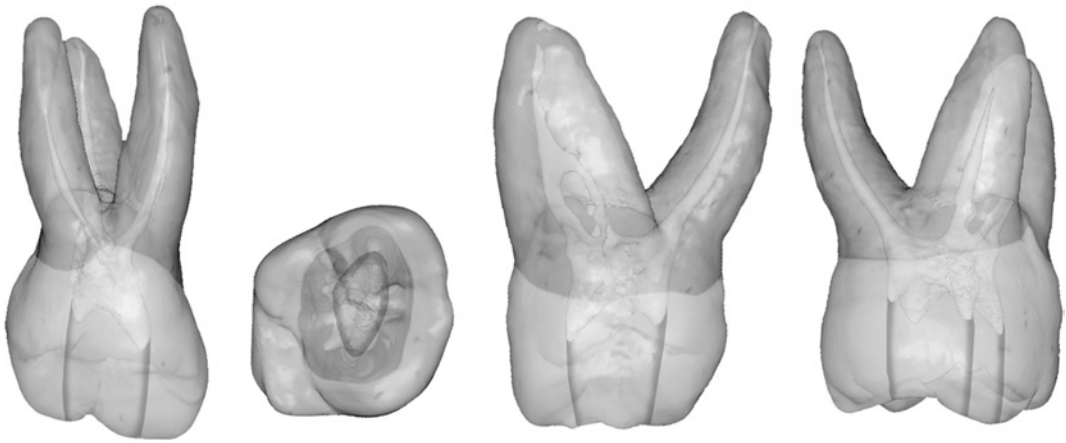

FIG. 5-103 Maxillary first molar. Average time of eruption—6 to 7 years; average age of calcification—9 to 10 years; average length—20.8 mm. Root curvature (most common to least common): mesiobuccal root—distal, straight; distobuccal root—straight, mesial, distal; palatal root—buccal, straight.

or three root canals (see Table 5-13 online at the Expert Consult site). The palatal root often curves buccally at the apical one third, which may not be obvious on a standard periapical radiograph. From its orifice the palatal canal is flat, ribbonlike, and wider in a mesiodistal direction. The distobuccal root is conical and may have one or two canals (see Table 5-14 online at the Expert Consult site). From its orifice, the canal (or canals) first is oval and then becomes round as it approaches the apical third of the root. The mesiobuccal root has generated more research and clinical investigation than any other root in the mouth.[202] It may have one, two, or three root canals (see Table 5-15 online at the Expert Consult site). A single mesiobuccal canal is oval and wider buccolingually; two or three canals are more circular. In general, a concavity exists on the distal aspect of the mesiobuccal root, which makes this wall thin. The μCT scans for the maxillary first molar can be seen in Fig. 5-104. (See Video 5-6 online at the Expert Consult site for rotational views of these teeth.) The DOM has been used to study the location and pathway of the mesiopalatal canal in maxillary first and second molars.[80] The clinician must always keep in mind that the location of this canal varies greatly; it generally is located mesial to or directly on a line between the mesiobuccal and palatal orifices, within 3.5 mm palatally and 2 mm mesially of the mesiobuccal orifice (Figs. 5-105 and 5-106). These authors[80] found that not all mesiopalatal orifices lead to a true canal. A true orifice for this canal was present in only 84% of molars in which a second orifice was identified (Fig. 5-107).[205]

Negotiation of the mesiopalatal canal often is difficult; a ledge of dentin covers its orifice, the orifice has a mesiobuccal inclination on the pulp floor, and the canal's pathway often takes one or two abrupt curves in the coronal part of the root. Most of these obstructions can be eliminated by troughing or countersinking with ultrasonic tips mesially and apically along the mesiobuccal pulpal groove (Figs. 5-108 through 5-111). This procedure may shift the canal mesially, meaning that the access wall also must be moved farther mesially. Troughing can easily be 0.5 to 3 mm deep, and care must be taken to avoid furcal wall perforation in this root. Apical to the troughing level. the canal may be straight or may curve sharply to the distobuccal, buccal, or palatal.

Because the maxillary first molar almost always has four canals, the access cavity has a rhomboid shape, with the corners corresponding to the four orifices (see Fig. 5-108). One study demonstrated that the access cavity should not extend into the mesial marginal ridge.[246] Distally, the preparation can invade the mesial portion of the oblique ridge, but it should not penetrate through the ridge. The buccal wall should be parallel to a line connecting the mesiobuccal and distobuccal orifices and not to the buccal surface of the tooth.

Maxillary Second Molar

Coronally, the maxillary second molar closely resembles the maxillary first molar (Fig. 5-112). The root and canal anatomy are similar to those of the first molar, although there are differences. The distinguishing morphologic feature of the

TABLE 5-13

Studies of Apical Canal Configurations for the Palatal Root of the Maxillary First Molar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)‡
Vertucci[223]	Clearing	United States	100	—	—
Pineda and Kuttler[170]	Radiographic	Mexico	100	—	—
Calişkan et al.[33]	Clearing	Turkey	96.7	33.3	—
Acosta et al.[2]	Ground section and magnification	Chili	100	—	—
Thomas et al.[215]	Radiographic	United States	99.1 (0.9)†	0.9	—
Pecora et al.[163]	Clearing	Brazil	100	—	—
Wasti et al.[236]	Clearing	Pakistan	66.7	33.3	—
al Shalabi et al.[3]	Clearing	Ireland	98.8	1.2	—
Martinez-Berná and Ruiz-Badanelli[132]	Clinical	Spain	Three case reports	—	—
Bond et al.[27]	Clinical	Pakistan	Case report (100)	—	—
Beatty[20]	Clinical	United States	Case report	—	—
Maggiore et al.[128]	Clinical	United States	—	—	Case report
Cecic et al.[35]	Clinical	United States	—	Case report	—
Baratto-Filho et al.[15]	Clinical	Brazil	—	Case report	—
Wong[248]	Clinical	United States	—	—	Case report
Thews et al.[214]	Clinical	United States	—	Two case reports	—
Christie et al.[39]	Clinical	Canada	—	Two case reports	—
Alavi et al.[5]	Clearing	Thailand	100	—	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	97 (3) 95	3 2 (2)	— 3 (3)
Ng et al.[151]	Clearing	Burma	100	—	—
Zaatar et al.[260]	Clearing	Kuwait	100	—	—
Barbizam et al.[16]	Clinical	Brazil	—	Case report	—
Gopikrishna et al.[79]	Clinical (spiral computed tomography)	India	—	Case report	—

*Numbers in parentheses are the percentage of cases with two canals joining into one canal.
†Numbers in parentheses are the percentage of cases with one canal dividing into two canals.
‡Numbers in parentheses are the percentage of cases with one canal dividing into three canals.

TABLE 5-14

Studies of Apical Canal Configurations for the Distobuccal Root of the Maxillary First Molar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†
Vertucci[223]	Clearing	United States	100	—
Pineda and Kuttler[170]	Radiographic	Mexico	96.4	3.6
Calişkan et al.[33]	Clearing	Turkey	98.4	1.6
Acosta et al.[2]	Ground section and magnification	Chili	100	—
Thomas et al.[215]	Radiographic	United States	96.7 (0.5)	3.3
Pecora et al.[163]	Clearing	Brazil	100	—
Wasti et al.[236]	Clearing	Pakistan	83.3	16.7
al Shalabi et al.[3]	Clearing	Ireland	97.5	2.5
Martinez-Berná and Ruiz-Badanelli[132]	Clinical	Spain	—	Case reports (3)
Hulsmann[93]	Clinical	Germany	—	Case report
Bond et al.[27]	Clinical	Pakistan	—	Case report
Beatty[20]	Clinical	United States	Case report	—
Maggiore et al.[128]	Clinical	United States	Case report	—
Cecic et al.[35]	Clinical	United States	Case report	—
Baratto-Filho et al.[15]	Clinical	Brazil	Case report	—
Wu et al.[249]	Clinical	United States	Case report	—
Alavi et al.[5]	Clearing	Thailand	100	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	97 (6) 96 (7)	2 (1) 4
Ng et al.[151]	Clearing	Burma	97.8	1.1
Zaatar et al.[260]	Clearing	Kuwait	100	—

*Numbers in parentheses are the percentage of cases with two canals joining into one canal.
†Numbers in parentheses are the percentage of cases with one canal dividing into two canals.

TABLE 5-15

Studies of Apical Canal Configurations for the Mesiobuccal Root of the Maxillary First Molar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)
Vertucci[223]	Clearing	United States	82 (17)	18	—
Pineda and Kuttler[170]	Radiographic	Mexico	51.5 (12.2)	48.5 (12.8)	—
Calişkan et al.[33]	Clearing	Turkey	75.4 (41)	24.6 (1.6)	—
Acousta Vigouraux and Trugeda Bosaans[2]	Ground section and magnification	Chile	28.4	69.3	2.3
Seidberg et al.[188]	Sections	United States	75 (37)	25	—
Weine et al.[241]	Sectioning	United States	86 (37.5)	14	—
Stropko[207]	Clinical (microscope)	United States	45.1 (38.1)	54.9	—
Fogel et al.[70]	Clinical (microscope)	Canada	68.3 (39.4)	31.7	—
Pomeranz and Fishelberg[173]	Clearing	United States	52 (21)	48	—
Kulild and Peters[108]	In vitro (microscope)	United States	41 (38.6)	59 (2.4)	—
Thomas et al.[215]	Radiographic	United States	73.6 (27.3)	26.4 (12)	—
Neaverth et al.[149]	Clinical	United States	37.7 (16.7)	61.9 (1.8)	0.4
Weine et al.[240]	Radiographs with files	Japan	66.2 (24.2)	33.8 (3.4)	—
Gilles and Reader[76]	SEM and microscope	United States	61.9 (52.4)	38.1 (4.8)	—
Pecora et al.[165]	Clearing	Brazil	92.5 (17.5)	7.5	—
Hartwell and Bellizzi[86]	Clinical (mesiobuccal root of maxillary first molar)	United States	81.4	18.6	—
Weller and Hartwell[245]	Radiograph of root canal teeth	United States	61	39	—
Wasti et al.[236]	Clearing	Pakistan	56.6 (23.3)	43.4 (13.3)	—
al Shalabi et al.[3]	Clearing	Ireland	36.4 (15.9)	62.6 (6.1)	1
Bond et al.[27]	Clinical	Pakistan	—	Case report	—
Beatty[20]	Clinical	United States	—	—	Case report
Maggiore et al.[128]	Clinical	United States	—	Case report	—
Cecic et al.[35]	Clinical	United States	—	Case report	—
Baratto-Filho et al.[15]	Clinical	Brazil	Case report	—	—
Wong[248]	Clinical	United States	Case report	—	—
Alavi et al.[5]	Clearing	Thailand	53.8	46.2	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	65 (62) 57 (7)	34 (6) 43 (16)	— —
Ng et al.[151]	Clearing	Burma	57.8	41.1	—
Zaatar et al.[260]	Radiographs of root canal teeth	Kuwait	85	15	—
Sykaras and Economou[210]	Radiographs and microscope	Greece	91 (23)	9	—
Imura et al.[94]	Clearing	Japan	11.8	88.2	—
Nosonowitz and Brenner[153]	Clinical	United States	84.9	15.1	—
Pattanshetti et al.[159]	Clinical	Kuwait	100 (41.8)	—	—
Smadi and Khraisat[198]	Clearing	Jordan	53 (30)	47 (11)	—
Rwenyonyi et al.[183]	Clearing (mesiobuccal root) Clearing (distal buccal root)	Uganda Uganda	80.1 (5) 98.2 (0.5)	19.4 (9.1) (1.8)	0.5 —

SEM, Scanning electron microscopy.
*Numbers in parentheses represent the percentage of cases in which two canals joined to form one.
†Numbers in parentheses represent the percentage of cases in which one canal divided to form two.

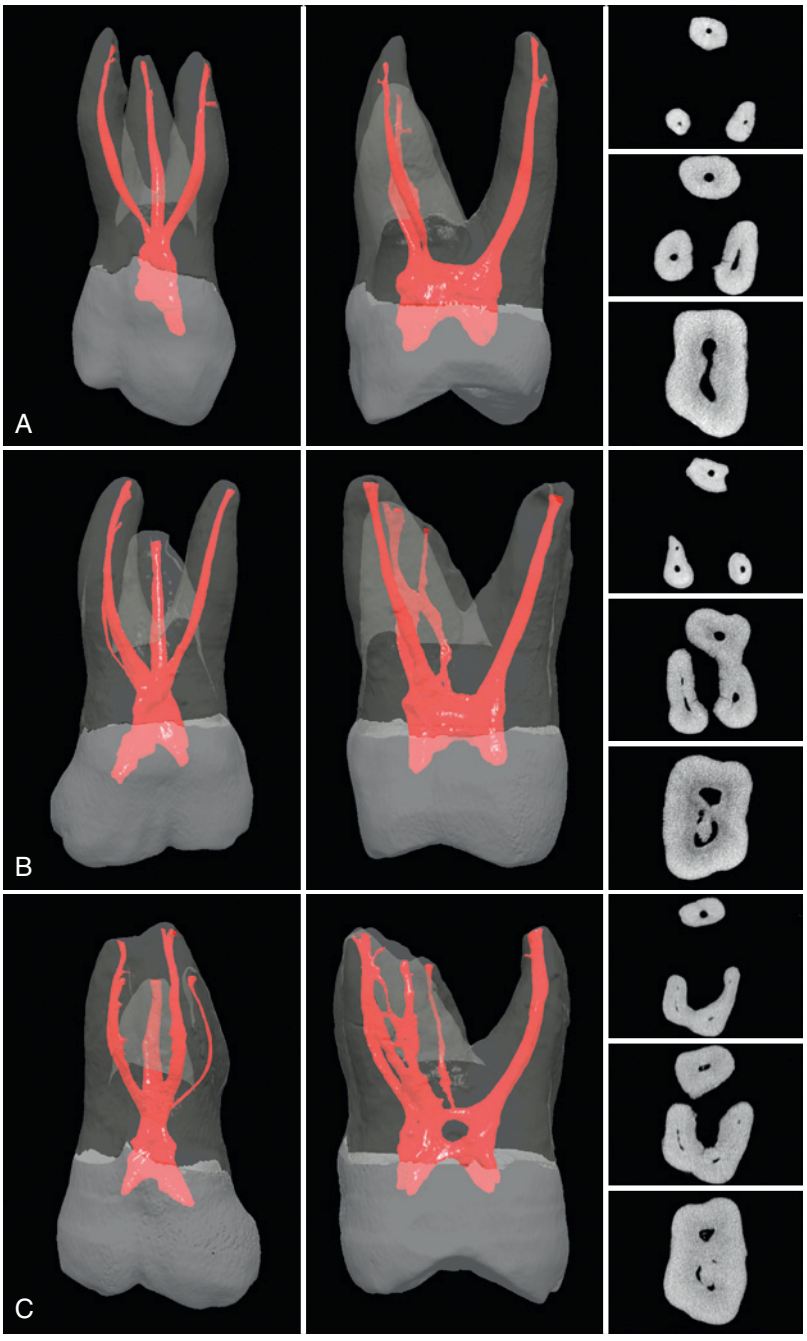

FIG. 5-104 μCT scans of maxillary first molars. **A,** Common anatomic presentation showing accessory/lateral canals. **B,** First molar with four canals, with mesiobuccal and mesiopalatal sharing an anastomosis in the midroot. **C,** Maxillary molar with four pulp horns, five canals, and significant anastomoses between the canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-6 online at the Expert Consult site for rotational views of these teeth.)

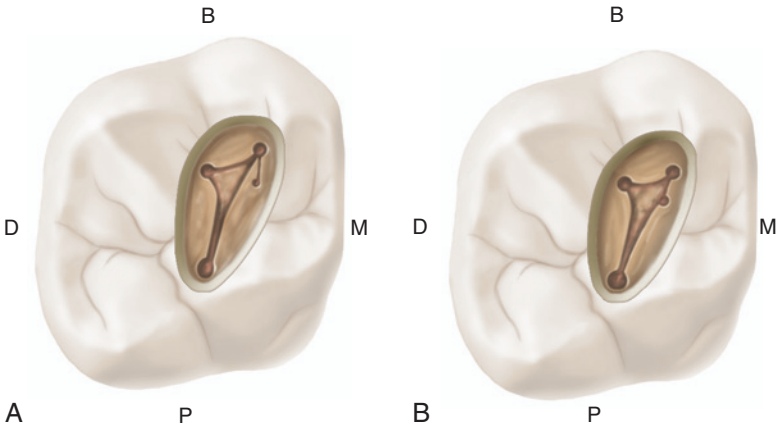

FIG. 5-105 The two locations of the second mesiobuccal *(MB-2)* canal orifices in a maxillary first molar. *B,* Buccal; *D,* distal; *M,* mesial; *P,* palatal.

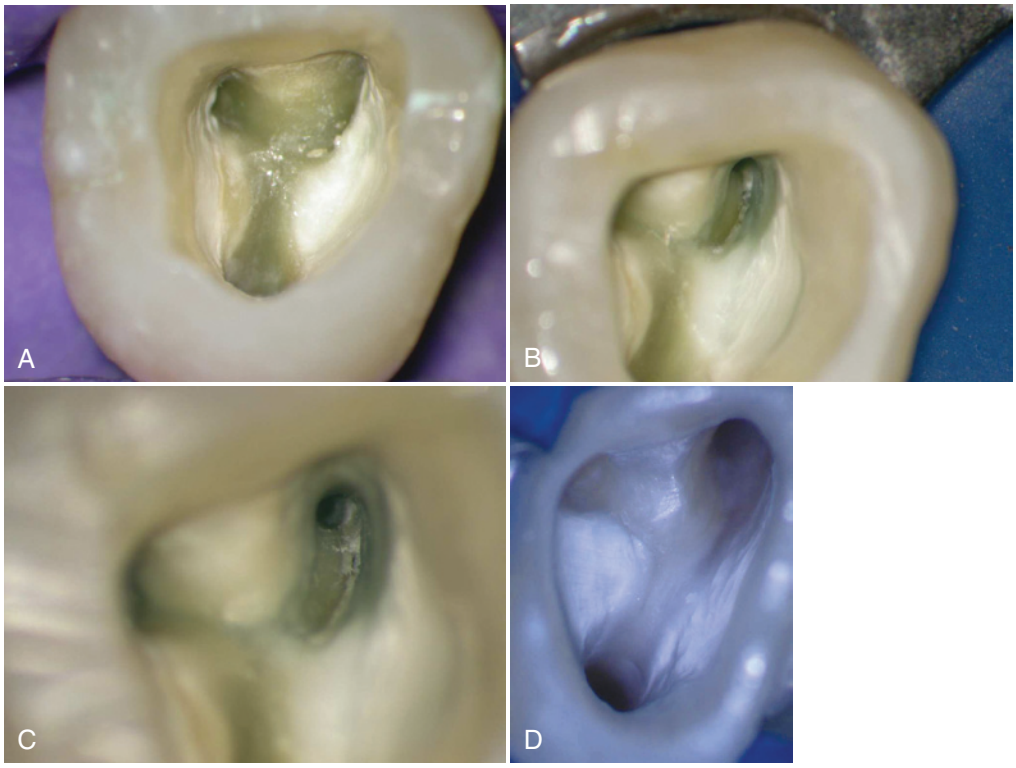

FIG. 5-106 Access cavity for a maxillary first molar as viewed through the dental operating microscope. **A,** Four apparent orifices, with a projection of dentin covering the mesial groove. (×3.4.) **B,** Removal of the mesial projection and troughing of the mesial groove to locate the second mesiobuccal *(MB-2)* canal. (×5.1.) **C,** Despite deepening of the mesial groove, the MB-2 canal cannot be located. (×8.5.) **D,** The MB-2 canal cannot be found even after removal of the mesial groove. (×13.6.)

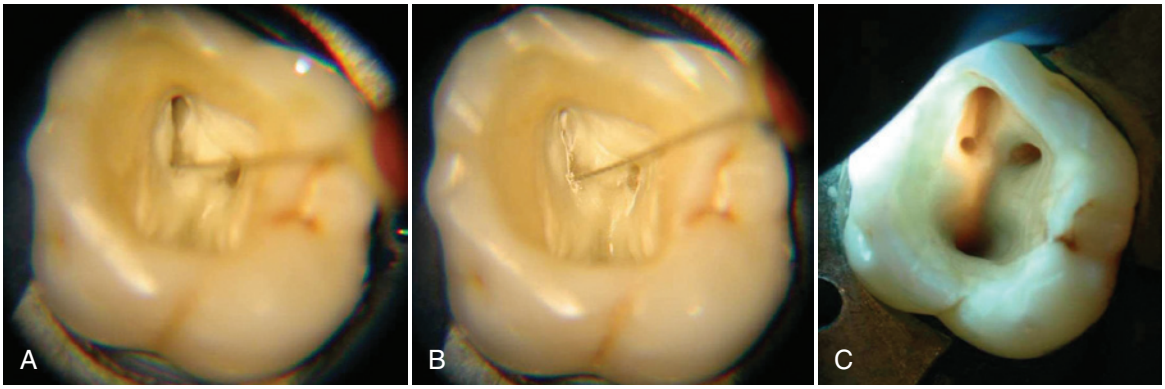

FIG. 5-107 Access preparation for a maxillary first molar as viewed through the dental operating microscope. **A,** Four apparent orifices located under ×3.4 magnification. **B,** Second mesiobuccal canal is located by deepening the mesial groove. (×5.1.) **C,** Four distinct canal orifices can be seen. (×5.1 magnification with cervical fiberoptic transillumination.)

FIG. 5-108 Four canals with loops and accessory canals.

FIG. 5-109 Two canals in both buccal roots with a common foramen in each root.

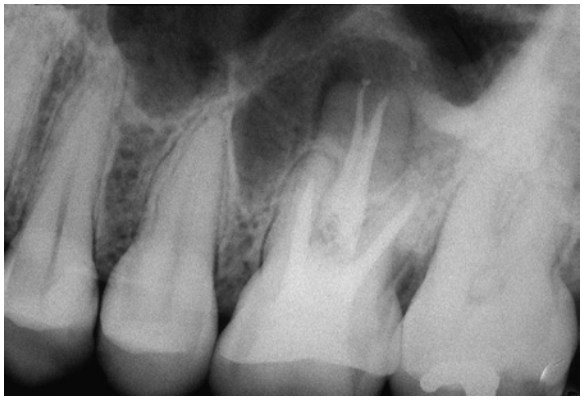

FIG. 5-110 Two separate canals in palatal root. (Courtesy Dr. Paulo Nogueira, São Paulo, Brazil.)

maxillary second molar is that its three roots are grouped closer together and are sometimes fused. Also, they generally are shorter than the roots of the first molar and not as curved. The second molar usually has one canal in each root; however, it may have two or three mesiobuccal canals, one or two distobuccal canals, or two palatal canals (see Tables 5-16 to 5-18 online at the Expert Consult site). Four canals are less likely to be present in the second molar than in the first molar. The μCT scans for the maxillary second molar can be seen in Fig. 5-113. (See Video 5-7 online at the Expert Consult site for rotational views of these teeth.) The three main orifices usually form a flat triangle and sometimes almost a straight line (Figs. 5-114 through 5-118). The mesiobuccal canal orifice is located more to the buccal and mesial than in the first molar; the distobuccal orifice approaches the midpoint between the mesiobuccal and palatal orifices[250]; and the palatal orifice usually is

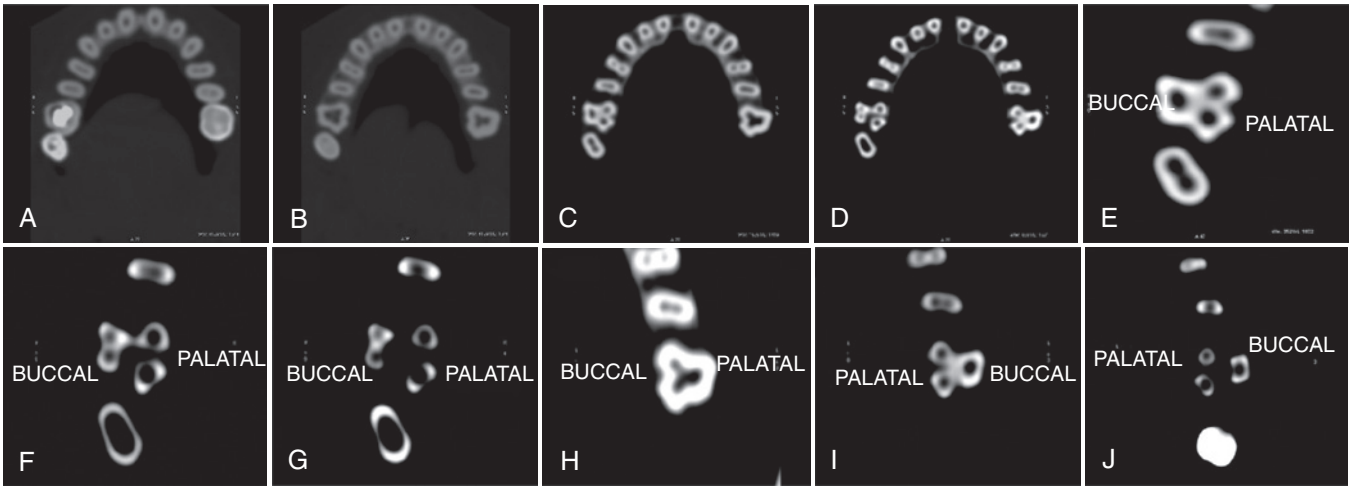

FIG. 5-111 A to D, Axial section spiral computed tomography (SCT) image of the maxillary arch. E to G, Enlarged axial section SCT image of tooth #3 showing two palatal roots and two canals in the fused buccal root. H to J, Enlarged axial section SCT image of tooth #14 showing the two palatal roots and a single canal in the fused buccal root. (From Gopikrishna V, Reuben J, Kandaswamy D: Endodontic management of a maxillary first molar with two palatal roots and a fused buccal root diagnosed with spiral computed tomography: a case report, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 105:e74, 2008.)

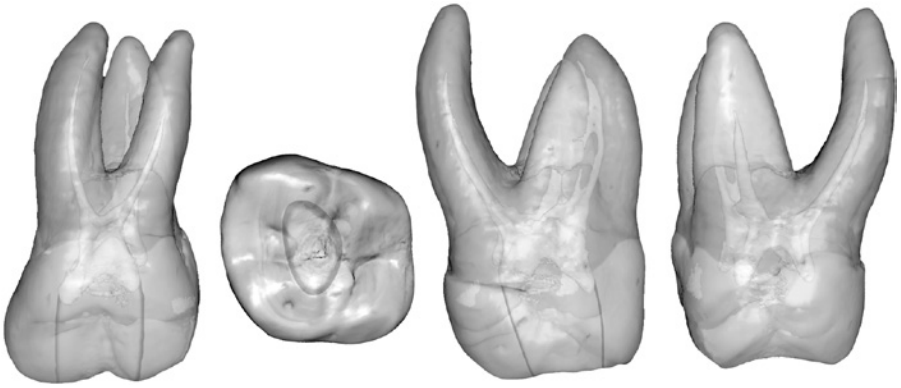

FIG. 5-112 Maxillary second molar. Average time of eruption—11 to 13 years; average age of calcification—14 to 16 years; average length—20 mm. Root curvature (most common to least common): mesiobuccal root—distal, straight; distobuccal root—straight, mesial, distal; palatal root—straight, buccal.

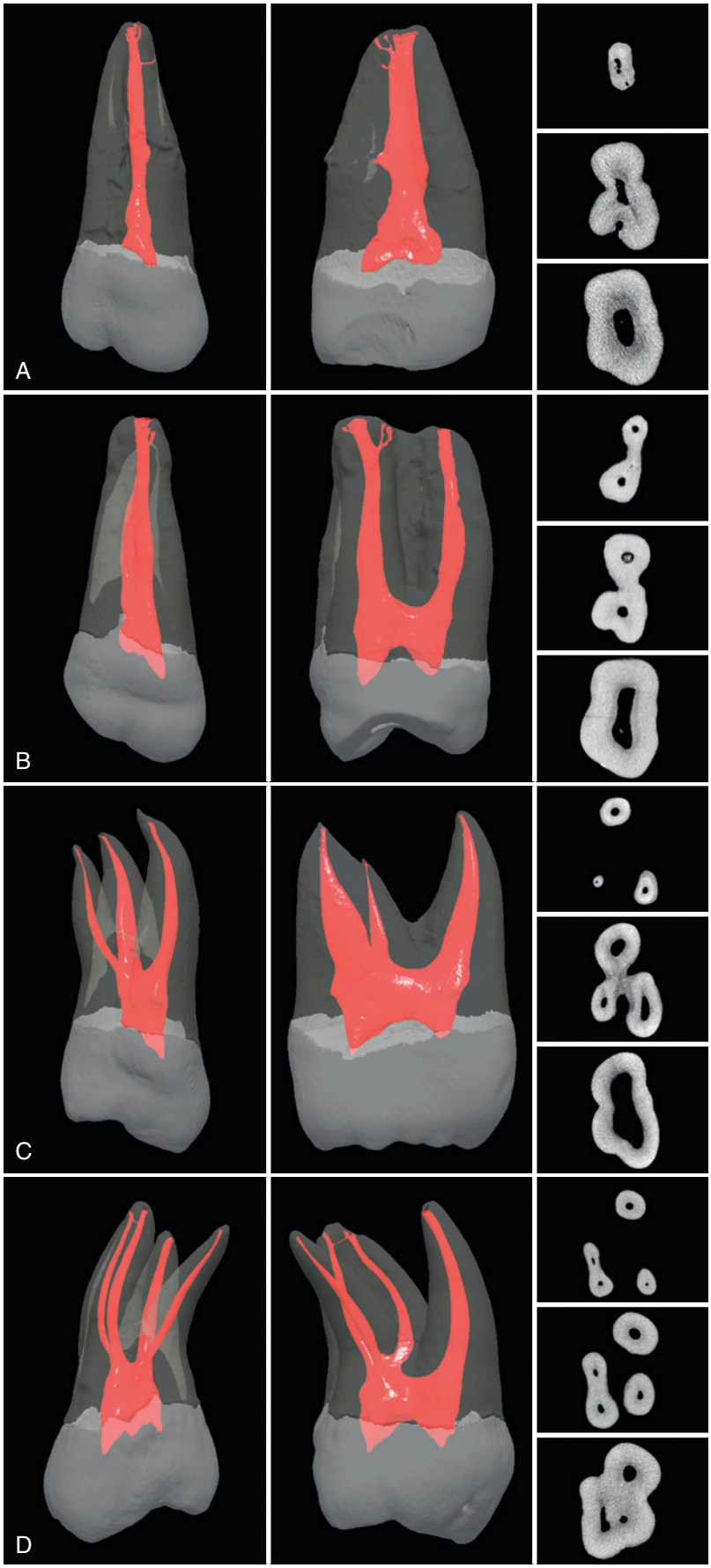

FIG. 5-113 μCT scans of maxillary second molars; four possible variations. **A,** Uncommon anatomic presentation of this tooth with one canal. **B,** Second molar with two canals. **C,** Second molar with three canals. **D,** Second molar with four distinct canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-7 online at the Expert Consult site for rotational views of these teeth.)

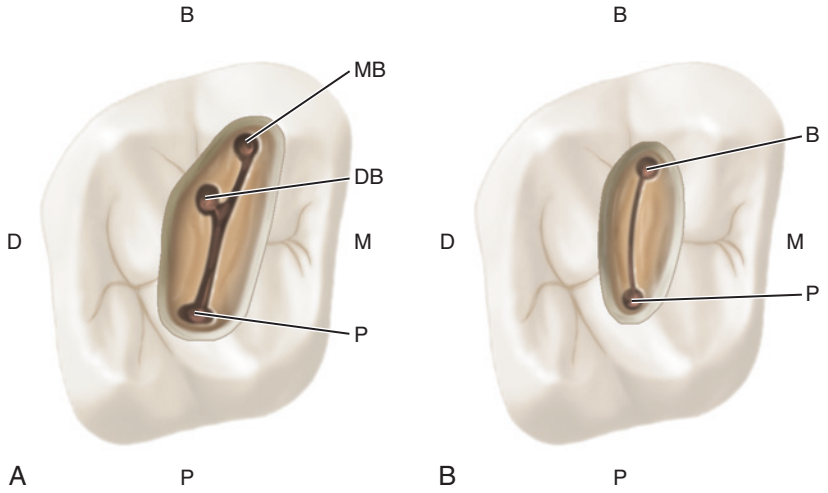

FIG. 5-114 A, Three canal orifices in a maxillary second molar. B, Two canal orifices in a maxillary second molar. *B,* Buccal; *P,* palatal; *D,* distal, *DB,* distobuccal; *M,* medial; *MB,* mesiobuccal.

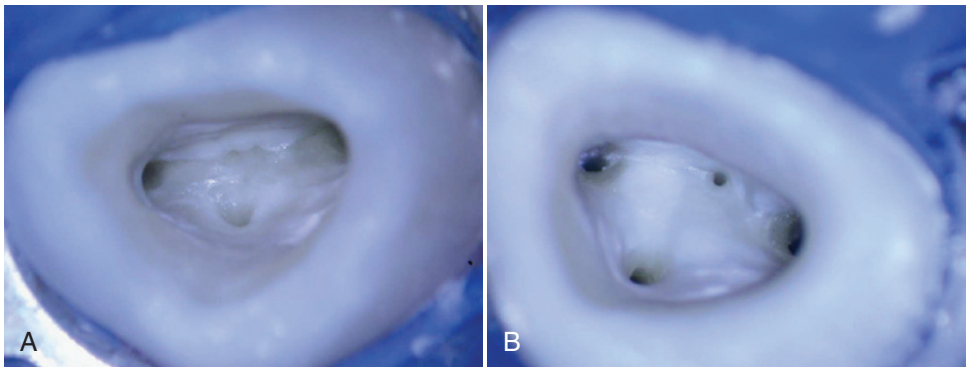

FIG. 5-115 Access cavity for a maxillary second molar as viewed through the dental operating microscope. A, Dentin projection covering the mesial aspect of the floor of the pulp chamber. (×8.4.) B, Fourth canal orifice (second mesiobuccal, *MB-2*), which was identified after removal of the dentin projection and troughing of the groove connecting the MB-1 orifice and the palatal *(P)* canal orifice. (×8.4.)

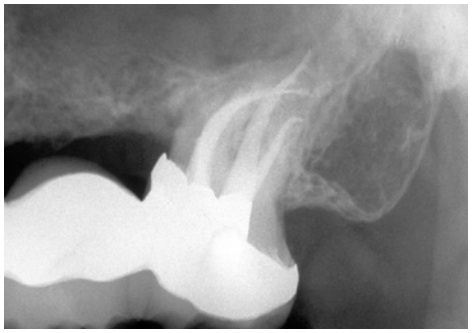

FIG. 5-116 Severely curved mesiobuccal root with a right-angle curve in the distobuccal *(DB)* root.

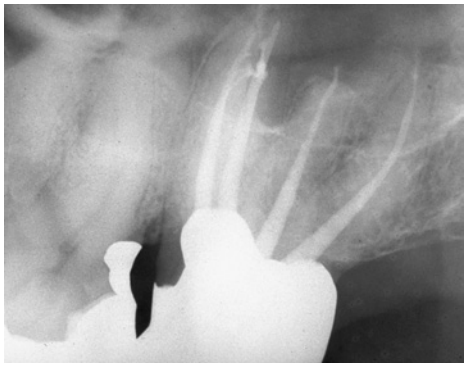

FIG. 5-117 Four canals (*MB-1, MB-2, DB,* and *P*) can be seen.

located at the most palatal aspect of the root. In general, the canal orifices in the maxillary second molar are closer mesially to each other than they are in the maxillary first molar.

The floor of the pulp chamber is markedly convex, which gives the canal orifices a slight funnel shape. On occasion the canals curve into the chamber at a more horizontal angle, requiring removal of a lip of dentin so that a canal can be entered more in a direct line with the axis. Teeth with two canals usually have a buccal and a palatal canal of equal length and diameter (see Fig. 5-114, *B*). These parallel root canals are frequently superimposed radiographically, but they can be imaged by exposing the radiograph from a distal angle. To enhance radiographic visibility, especially when interference arises from the malar process, a more perpendicular and

TABLE 5-16

Studies of Apical Canal Configurations for the Mesiobuccal Root of the Maxillary Second Molar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)	Four Canals (%)
Vertucci[223]	Clearing	United States	88 (17)	12	—	—
Pineda and Kuttler[170]	Radiographic	Mexico	72.8 (8.2)	27.2 (14.4)	—	—
Calişkan et al.[33]	Clearing	Turkey	68.7 (23.6)	27.1 (4.2)	4.2	—
Pecora et al.[165]	Clearing	Brazil	80 (22)	20	—	—
Gilles and Reader[76]	SEM and light microscopy	United States	62 (32.4)	38 (2.7)	—	—
Stropko[207]	Clinical (microscope)	United States	78 (45.6)	22	—	—
Eskoz and Weine[58]	Radiographic		80.6 (20.9)	19.4 (3)	—	—
Pomeranz and Fishelberg[173]	Clinical	United States	75.9 (13.8)	24.1	—	—
Nosonowitz and Brenner[153]	Clinical	United States	94.4 (25.5)	5.6	—	—
al Shalabi et al.[3]	Clearing	Ireland	50 (5.6)	50 (16.7)	—	—
Benenati[23]	Clinical	United States	Case report	—	—	—
Fahid and Taintor[61]	Clinical	United States	Case report	—	—	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	71 (45) 81 (25)	28 18	1 —	— 1
Kulild and Peters[108]	In vitro (microscope)	United States	54.2	45.8	—	—
Alavi et al.[5]	Clearing	Thailand	53.8	44.6	—	1
Zaatar et al.[259]	Radiographs of root canal teeth	Kuwait	93.5 (17.4)	6.5	—	—
Imura et al.[94]	Clearing	Japan	30	70	—	—
Ng et al.[151]	Clearing	Burma	74	26	—	—
Rwenyonyi et al.[183]	Clinical (mesial root) Clinical (distal buccal root) Clinical (palatal root)	Uganda Uganda Uganda	88.7 (1.8) 99.5 99.5 (0.5)	11.7 (5) (0.5) (0.5)	— — —	— — —

*Numbers in parentheses are the percentage of cases in which two canals joined into one.
†Numbers in parentheses are the percentage of cases in which one canal divided to form two.

TABLE 5-17

Studies of Apical Canal Configurations for the Distobuccal Root of the Maxillary Second Molar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)
Vertucci[223]	Clearing	United States	100	—
Pineda and Kuttler[170]	Radiographic	Mexico	100	—
Calişkan et al.[33]	Clearing	Turkey	100	—
Pecora et al.[165]	Clearing	Brazil	100	—
al Shalabi et al.[3]	Clearing	Ireland	100	—
Benenati[23]	Clinical	United States	Case report	—
Fahid and Taintor[61]	Clinical	United States	—	Case report
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	100 (2) 100 (2)	— —
Zaatar et al.[260]	Radiographs of root canal teeth	Kuwait	100	—

*Numbers in parentheses are the percentage of cases with two canals joining into one canal.

TABLE 5-18

Studies of Apical Canal Configurations for the Palatal Root of the Maxillary Second Molar

Authors	Type of Study	Country	One Canal (%)	Two Canals (%)	Three Canals (%)
Vertucci[223]	Clearing	United States	100	—	—
Pineda and Kuttler[170]	Radiographic	Mexico	100	—	—
Calişkan et al.[33]	Clearing	Turkey	97.9	2.1*	—
Pecora et al.[165]	Clearing	Brazil	100	—	—
al Shalabi et al.[3]	Clearing	Ireland	100	—	—
Benenati[23]	Clinical	United States	—	Case report	—
Sert and Bayirli[190]	Clearing (men)	Turkey	100	—	—
	Clearing (women)	Turkey	100	—	—
Zaatar et al.[260]	Radiographs of root canal teeth	Kuwait	100	—	—
Ulusoy and Görgul[221]	Clinical	Turkey	—	Case report	—
Shin et al.[191]	Clinical (microscope)	South Korea	—	Case report	—

*Number is the percentage of cases in which one canal separates into two.

distoangular radiograph may be exposed. When two roots are present, each root may have one canal, or the buccal root may have two canals that join before reaching a single foramen. One study found that two palatal roots and two palatal canals occur in 1.47% of these teeth.[166]

When four canals are present, the access cavity preparation of the maxillary second molar has a rhomboid shape and is a smaller version of the access cavity for the maxillary first molar (see Fig. 5-115). If only three canals are present, the access cavity is a rounded triangle with the base to the buccal. As with the maxillary first molar, the mesial marginal ridge need not be invaded. Because the tendency in maxillary second molars is for the distobuccal orifice to move closer to a line connecting the mesiobuccal and palatal orifices, the triangle becomes more obtuse and the oblique ridge usually is not invaded.

If only two canals are present, the access outline form is oval and widest in the buccolingual dimension. Its width corresponds to the mesiodistal width of the pulp chamber, and the oval usually is centered between the mesial pit and the mesial edge of the oblique ridge.

Maxillary Third Molar

Loss of the maxillary first and second molars often is the reason the third molar must be considered a strategic abutment (Fig. 5-119). Careful examination of the root morphology is important before treatment is determined. The radicular anatomy of the third molar is completely unpredictable, and it may be advisable to explore the root canal morphology to evaluate the likelihood and degree of success. Even so, many third molars have adequate root formation; given reasonable accessibility, they can serve well as functioning dentition after root canal therapy.

The root anatomy of the maxillary third molar varies greatly. This tooth can have one to four roots and one to six canals, and C-shaped canals also can occur. The third molar usually has three roots and three root canals (see Table 5-19 online at the Expert Consult site). The tooth may be tipped significantly to the distal, the buccal, or both, which creates an even greater access problem than with the second molar. The μCT scans for the maxillary molar can be seen in Fig. 5-120. (See Video 5-8 online at the Expert Consult site for rotational views of these teeth.)

The access cavity form for the third molar can vary greatly. Because the tooth typically has one to three canals, the access preparation can be anything from an oval that is widest in the buccolingual dimension to a rounded triangle similar to that used for the maxillary second molar. All the canal orifices often lie nearly in a straight line as the distobuccal orifice moves even closer to the line connecting the mesiobuccal and palatal orifices. The resultant access cavity is an oval or highly obtuse triangle (Figs. 5-121 and 5-122).

FIG. 5-118 Four canals with two distinct palatal roots and canals.

Mandibular Central and Lateral Incisors

The root canal systems and access cavities for the two mandibular incisors are so similar they are discussed together (Fig. 5-123). As with the maxillary incisors, a lingual shoulder must be eliminated to allow direct-line access. The shoulder conceals the orifice to a second canal that, if present, is found immediately beneath it. Unlike the maxillary incisors, the pulp outline of the mandibular incisors is wider labiolingually. At the CEJ the pulp outline is oval and wider labiolingually than mesiodistally. At midroot the canal outline is still oval, but the canal is more constricted and narrower labiolingually. Most mandibular incisors have a single root with what radiographically appears to be a long, narrow canal. However, it is a broad canal labiolingually. Often a dentinal bridge is present in the pulp chamber that divides the root into two canals. The two canals usually join and exit through a single apical foramen, but they may persist as two separate canals. On occasion one canal branches into two canals, which subsequently rejoin into a single canal before reaching the apex (see Table 5-20 online

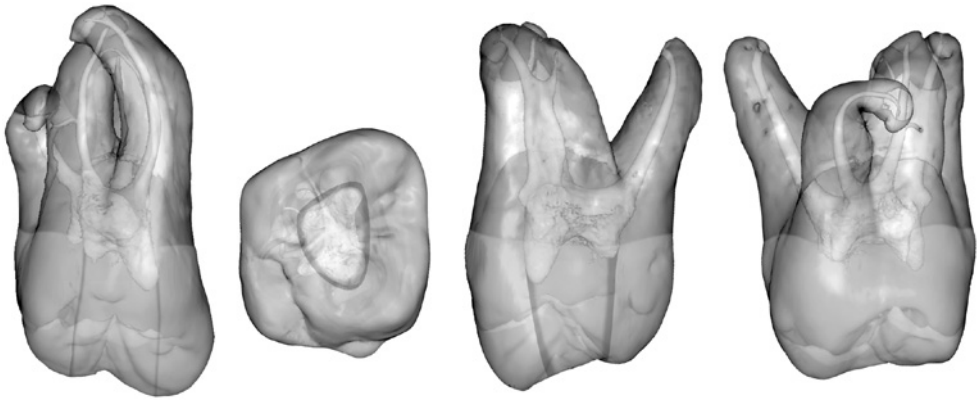

FIG. 5-119 Maxillary third molar. Average time of eruption—17 to 22 years; average age of calcification—18 to 25 years; average length—17 mm.

TABLE 5-19

Studies of Apical Canal Configurations for the Maxillary Third Molar

Authors	Type of Study	Country	Number of Roots	Number of Canals (%)					
				1	2	3	4	5	6
Sidow et al.[192]	Clearing	United States	1	2.7	2.7	6	2	1.3	0.7
	Clearing	United States	2 (4.7%*)	—	—	22	4.7	0.7	—
	Clearing	United States	3	—	0.7	29.3	14.7	1.7	—
	Clearing	United States	4	—	—	—	6	2.6	—
Pineda and Kuttler[170]	Radiographic	Mexico	Mesiobuccal	42 (20.6*)	31.1	21	5.9	—	—
Pecora et al.[165]	Clearing	Brazil	Mesiobuccal	86 (18*)	14	—	—	—	—

*Percentage of cases in which two canals joined to form one.

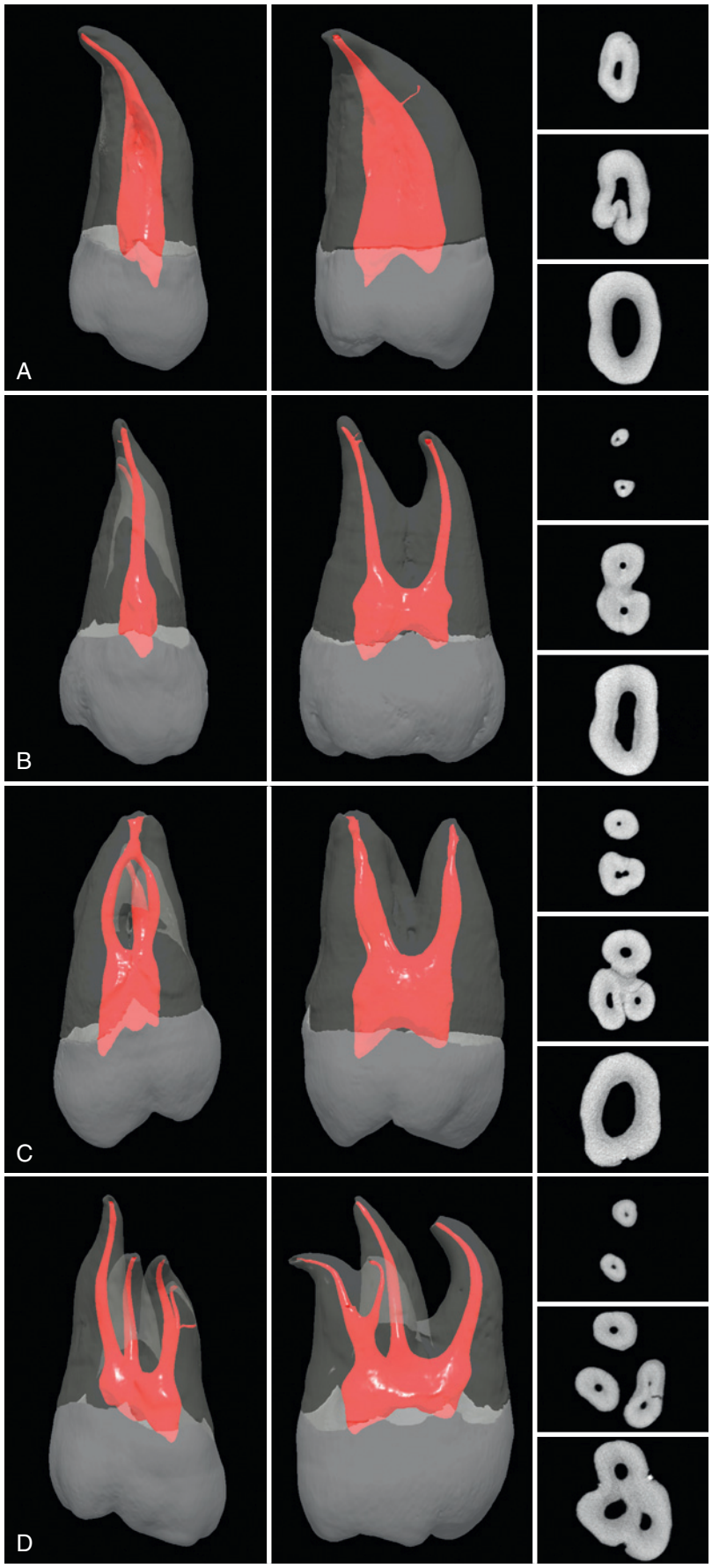

FIG. 5-120 μCT scans of maxillary third molars show a range of anatomic variations; **A**, Single-canal tooth. **B**, Two-rooted third molar. **C**, Two-rooted, three-canal third molar with significant root curvatures. **D**, Three-rooted, four-canal third molar. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-8 online at the Expert Consult site for rotational views of these teeth.)

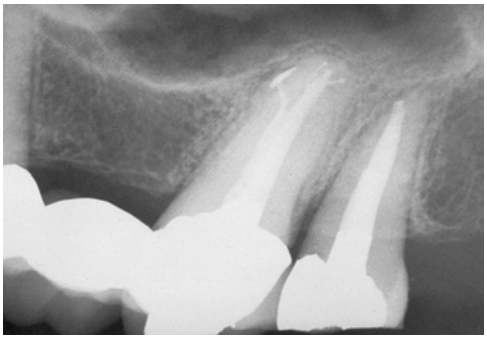

FIG. 5-121 Canals that have fused into a single canal. Multiple accessory canals can be seen in the second molar.

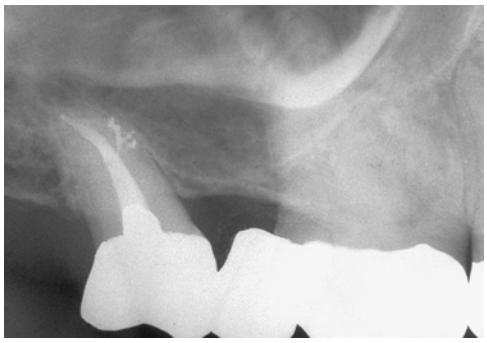

FIG. 5-122 Distal bridge abutment with major accessory canal.

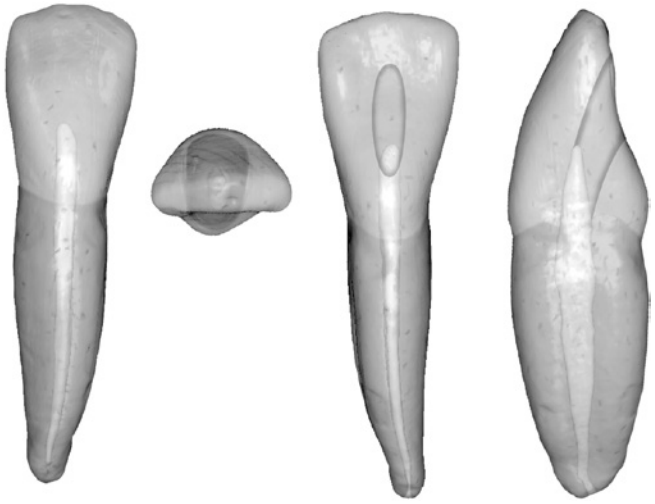

FIG. 5-123 Mandibular central/lateral incisors. Average time of eruption—6 to 8 years; average age of calcification—9 to 10 years; average length—20.7 mm. Root curvature (most common to least common): straight, distal, labial.

at the Expert Consult site). The scans of the mandibular central incisor can be seen in Fig. 5-124. The scans for the mandibular lateral incisors can be seen in Fig. 5-125. (See Videos 5-9 and 5-10 online at the Expert Consult site for rotational views of these teeth.)

One study determined that a relationship existed between crown size and the incidence of bifid root canals in these teeth.[234,235] Double root canals occur more often in teeth with a smaller index.

The mandibular incisors, because of their small size and internal anatomy, may be the most difficult access cavities to prepare. The external outline form may be triangular or oval, depending on the prominence of the mesial and distal pulp horns (Figs. 5-126 through 5-128). When the form is triangular, the incisal base is short and the mesial and distal legs are long incisogingivally, creating a long, compressed triangle. Without prominent mesial and distal pulp horns, the oval external outline form also is narrow mesiodistally and long incisogingivally. One study[152] determined that by age 40 years, the mandibular incisor pulp chamber has decreased in size sufficiently to justify an oval access cavity routinely. Complete removal of the lingual shoulder is critical because this tooth often has two canals that are buccolingually oriented, and the lingual canal most often is missed. To avoid missing this canal, the access preparation is extended well into the cingulum gingivally. Because the lingual surface of this tooth is not involved with occlusal function, restoration of the access opening with butt joint junctions between the internal walls and the lingual surface is not required.

Mandibular Canine

The root canal system of the mandibular canine is very similar to that of the maxillary canine, except that the dimensions are smaller, the root and root canal outlines are narrower in the mesiodistal dimension, and the mandibular canine occasionally has two roots and two root canals located labially and lingually (Fig. 5-129) (see Table 5-21 online at the Expert Consult site). The μCT scans of the mandibular canine can be seen in Fig. 5-130. (See Video 5-11 online at the Expert Consult site for rotational views of these teeth.) A fourth type is seen in subsequent Figs. 5-131 to 5-135.[222]

The root canal of the mandibular cuspid is narrow mesiodistally but usually very broad buccolingually. A lingual shoulder must be removed to gain access to the lingual wall of the root canal or to the entrance of a second canal. The lingual wall is almost slitlike compared with the larger buccal wall, which makes the canal a challenge to shape and clean.

The access cavity for the mandibular canine is oval or slot shaped (see Fig. 5-131). The mesiodistal width corresponds to the mesiodistal width of the pulp chamber. The incisal extension can approach the incisal edge of the tooth for straight-line access, and the gingival extension must penetrate the cingulum to allow a search for a possible lingual canal.

Mandibular First Premolar

As a group, the mandibular premolars present anatomic challenges because of the extreme variations in their root canal morphology (Fig. 5-136).[123,157,252] The root canal system of the mandibular first premolar is wider buccolingually than mesiodistally.[253] Two pulp horns are present: a large, pointed buccal horn and a small, rounded lingual horn. At the cervical line the root and canal are oval; this shape tends to become round as the canal approaches the middle of the root. If two canals are present, they tend to be round from the pulp chamber to their foramen. In another anatomic variation, a single, broad root canal may bifurcate into two separate root canals. Direct access to the buccal canal usually is possible, whereas the lingual canal may be quite difficult to find. The lingual canal tends to diverge from the main canal at a sharp angle. In addition, the lingual inclination of the crown tends to direct files

Text continued on p. 190

TABLE 5-20

Studies of Apical Canal Configurations for the Mandibular Incisors

Authors	Type of Study	Country	Tooth	One Canal (%)*	Two Canals (%)†	Three Canals (%)‡
Vertucci[223]	Clearing	United States	Central	97 (5)	3	—
Pineda and Kuttler[170]	Radiographic	Mexico	Central	97.9 (2)	2.1	—
Calişkan et al.[33]	Clearing	Turkey	Central	96 (13.7)	2†	2
Madeira and Hetem[126]	Clearing	Brazil	Central	99.7 (11)	0.3	—
Funato et al.[72]	Clinical	Japan	Central	—	Case report	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	Central Central	89 (54) 85 (55)	10 (2) 13 (3)	1 (1) 2
Walker[232]	Radiographic	Southern China	Central	99	1	—
Vertucci[223]	Clearing	United States	Lateral	98 (5)	2	—
Pineda and Kuttler[170]	Radiographic	Mexico	Lateral	98.7 (3.2)	1.3	—
Calişkan et al.[33]	Clearing	Turkey	Lateral	98 (13.7)	2†	—
Madeira and Hetem[126]	Clearing	Brazil	Lateral	99.2 (11.1)	0.8	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	Lateral Lateral	90 (52) 90 (54)	10 10 (2)	— —
Walker[232]	Radiographic	Southern China	Lateral	99	1	—
Karagöz-Kucukay[98]	Clearing	Turkey	Incisors	85	15†	—
Kartal and Yanikoglu[100]	Clearing	Turkey	Incisors	92 (17)	9 (3†)	—
Benjamin and Dowson[24]	Radiographic with files	United States	Incisors	98.7 (40.1)	1.3	—
Miyashita et al.[138]	Clearing	Japan	Incisors	96.9 (9.3)	3.1 (1.7†)	—
Rankine-Wilson and Henry[176]	Radiographic	Australia	Incisors	94.6	5.4	—
Kartal and Yanikoglu[100]	Clearing	Turkey	Incisors	92	7	1
Green[81]	Ground sections and microscope	United States	Incisors (both)	95 (17)	5 (1)	—
Al-Qudah and Awawdeh[9]	Clearing	Jordan	Incisors (both)	91.3 (17.6)	8.7 (3.6)	—

*Numbers in parentheses are the percentage of cases with two canals joining into one canal.
†Numbers in parentheses are the percentage of cases with one canal dividing into two canals.
‡Number in parentheses is the percentage of cases with one canal dividing into three canals.

TABLE 5-21

Studies of Apical Canal Configurations for the Mandibular Canine

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)	Three Canals (%)
Vertucci[223]	Clearing	United States	94 (14)	6	—
Pineda and Kuttler[170]	Radiographic	Mexico	95 (13.5)	5	—
Calişkan et al.[33]	Clearing	Turkey	98 (3.9)	2†	—
Pecora et al.[164]	Clearing	Brazil	97.1 (4.9)	2.9	—
D'Arcangelo et al.[49]	Clinical	Italy	—	Case report	—
Orguneser and Kartal[155]	Clinical	Turkey	—	Case report (3 → 2)‡	—
Heling et al.[89]	Clinical	Israel	—	—	Case report
Green[81]	Ground sections and microscope	United States	97 (10)	3 (2)	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	100 (10) 97 (35)	— 3	— —

*Numbers in parentheses are the percentage of cases in which two canals joined to form one.
†Percentage of cases in which one canal divided to form two.
‡Three canals reconfigured to form two.

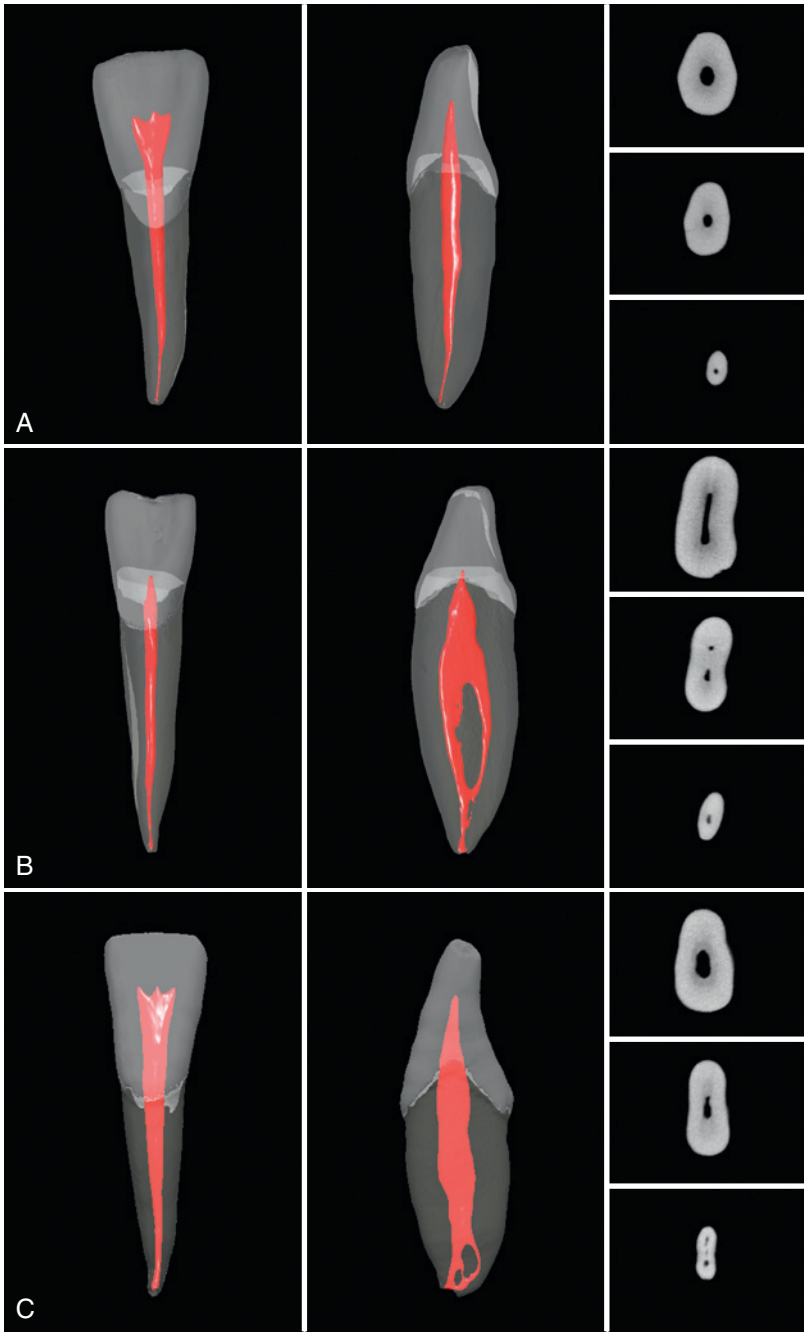

FIG. 5-124 μCT scans of mandibular central incisors. **A**, Common anatomic presentation. **B**, Central incisor with two canals. **C**, Central incisor with an apical delta. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-9 online at the Expert Consult site for rotational views of these teeth.)

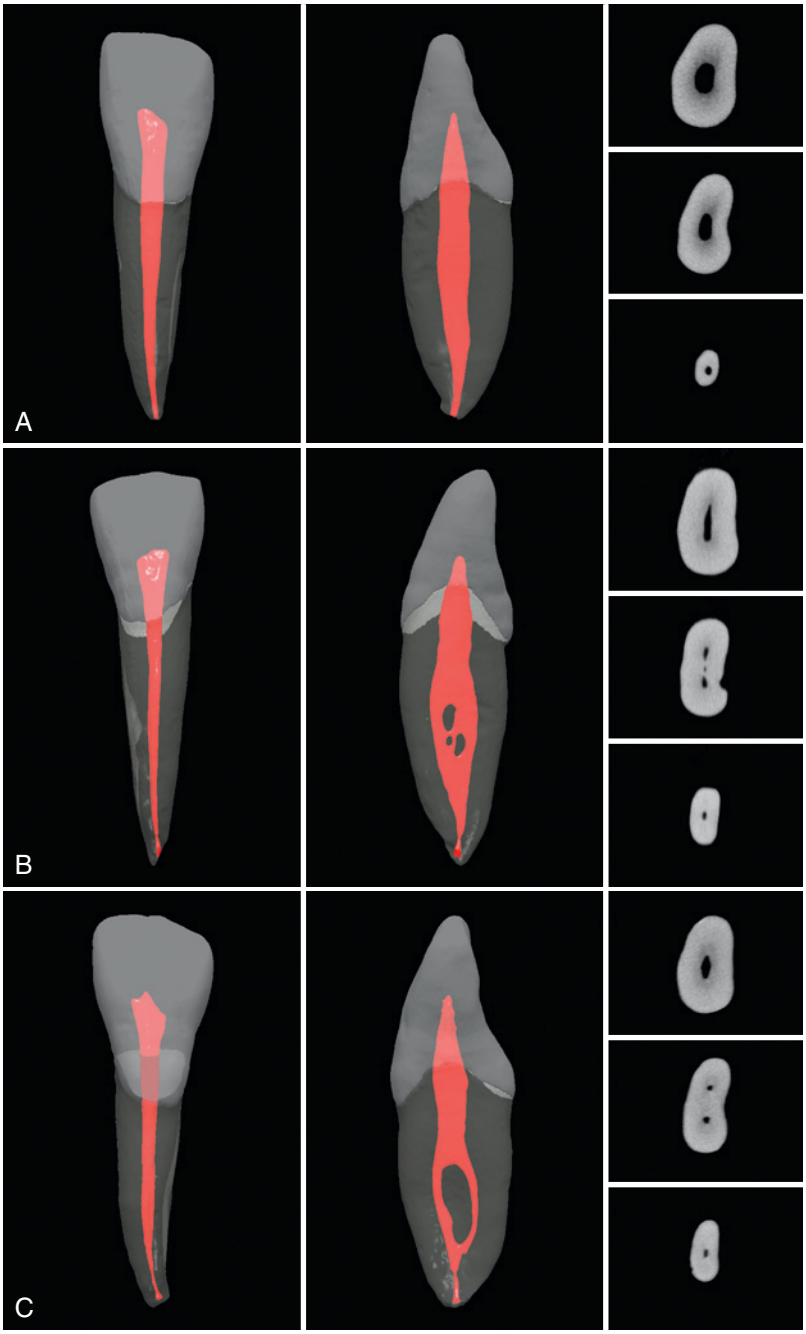

FIG. 5-125 μCT scans of mandibular lateral incisors. **A,** Common anatomic presentation. **B,** Lateral incisor with broad, thin buccolingual anatomy. **C,** Lateral incisor in which the canal splits into two but returns to form one canal apically. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-10 online at the Expert Consult site for rotational views of these teeth.)

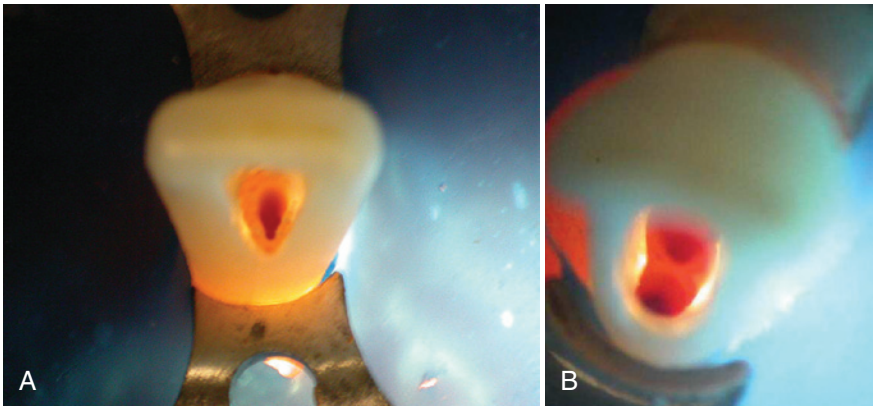

FIG. 5-126 Access cavity for mandibular incisors as viewed through the dental operating microscope. **A**, One canal orifice. (×8.5 magnification with cervical fiberoptic transillumination.) **B**, Two canal orifices. (×8.5 magnification with cervical fiberoptic transillumination.)

FIG. 5-127 Double-rooted mandibular lateral incisor.

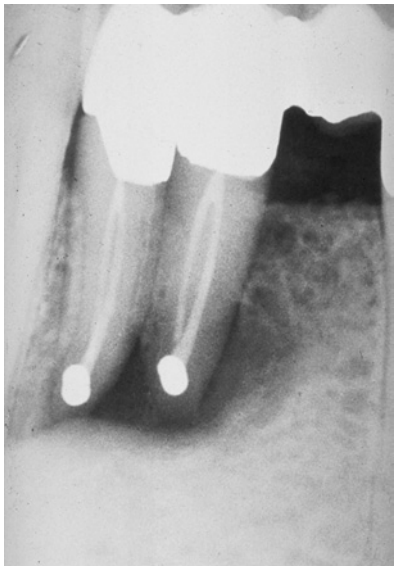

FIG. 5-128 Two canals in the mandibular lateral and central incisors.

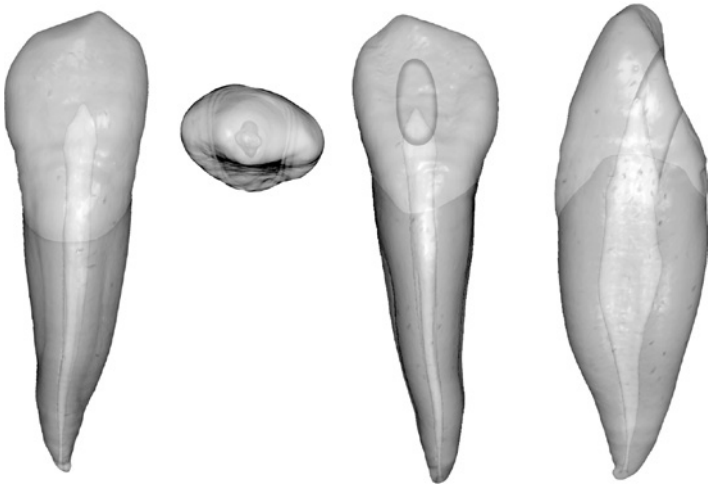

FIG. 5-129 Mandibular canine. Average time of eruption—9 to 10 years; average age of calcification—13 years; average length—25.6 mm. Root curvature (most common to least common): straight, distal, labial.

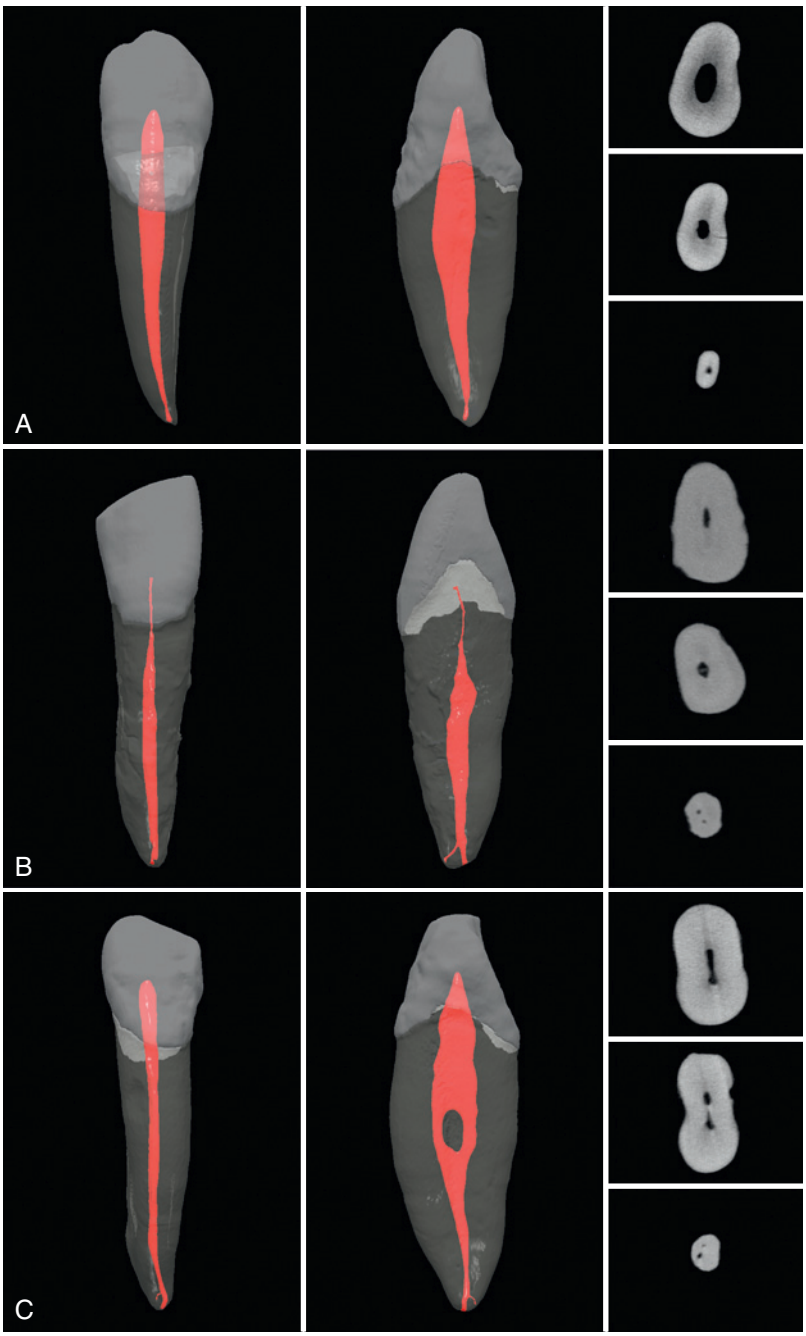

FIG. 5-130 μCT scans of mandibular canines. **A**, Common anatomic presentation. **B**, Canine with an extra apical canal. **C**, Canine that splits into two but returns to one canal apically. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. A fourth type is seen in Figs. 5-133 through 5-135. (See Video 5-11 online at the Expert Consult site for rotational views of these teeth.)

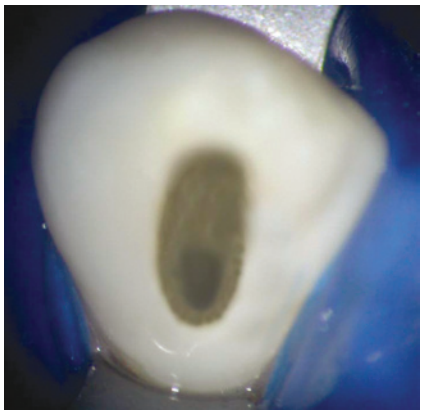

FIG. 5-131 Access preparation for a mandibular canine as viewed through the dental operating microscope. (×5.1.)

FIG. 5-134 Two separate root canals.

FIG. 5-132 One canal with a sharp mesial curvature at the apex.

FIG. 5-135 Mandibular canine and lateral incisor with two canals.

FIG. 5-133 Two canals with the lateral canal above the crest of bone; the lateral canal is probably responsible for the pocket depth.

buccally, making location of a lingual canal orifice more difficult. To counter this situation, the clinician may need to extend the lingual wall of the access cavity farther lingually; this makes the lingual canal easier to locate. The mandibular first premolar sometimes may have three roots and three canals (see Table 5-22 online at the Expert Consult site). Multiple studies have reported C-shaped canal anatomy in this tooth.[13,65,67] The μCT scans for the mandibular first premolar can be seen in Fig. 5-137. (See Video 5-12 online at the Expert Consult site for rotational views of these teeth.)

The oval external outline form of the mandibular first premolar typically is wider mesiodistally than its maxillary counterpart, making it more oval and less narrow (Figs. 5-138 through 5-141). Because of the lingual inclination of the crown, buccal extension can nearly approach the tip of the buccal cusp to achieve straight-line access. Lingual extension

TABLE 5-22

Studies of Apical Canal Configurations for the Mandibular First Premolar

Authors	Type of Study	Country	One Canal (%)	Two Canals (%)*	Three Canals (%)	Four Canals (%)
Vertucci[223]	Clearing	United States	74	25.5 (24)	0.5	—
Pineda and Kuttler[170]	Radiographic	Mexico	74.2	24.9 (23.4)	0.9	—
Calişkan et al.[33]	Clearing	Turkey	75.5 (7.6†)	18.8 (9.4)	5.7	—
Zillich and Dowson[262]	Radiographic	United States	80.7	18.9	0.4	—
Baisden et al.[13]	Serial sections	United States	76	24 (24)	—	—
Yoshioka et al.[258]	Clearing	Japan	80.6	15.1‡	4.3	—
Trope et al.[219]	Radiographic	United States				
Black patients			67.3	—	32.8	—
White patients			86.3	—	13.7	—
Green[81]	Ground sections and microscope	United States	94 (4)	6 (2)	—	—
Sert and Bayirli[190]	Clearing (men)	Turkey	85 (20)	15 (5)	0	—
	Clearing (women)	Turkey	94 (38)	4	2	—
Zaatar et al.[260]	Radiographs of root canal teeth	Kuwait	60	40	—	—
Walker[231]	Radiographic	South China	65	35	—	—
Nallapati[146]	Clinical	United States	—	—	Case report	—
Moayedi and Lata[142]	Clinical	—	—	—	Case report	—
Yang[256]	Clinical	Australia	—	—	Case report	—
Chan et al.[37]	Clinical	Taiwan, China	—	—	Case report	—
England et al.[57]	Clinical	United States	—	Case report	—	—
Hülsmann[92]	Clinical	Germany	—	—	Case report	—
Lu et al.[122]	Radiography and sectioning	China	60	40	—	—
Al-Qudah and Awawdeh[9]	Clearing	Jordan	64.8 (6.6)	33 (18.6)	2.2	—

*Numbers in parentheses are the percentage of cases in which one canal divided to form two.
†Percentage of cases in which two canals joined to form one.
‡Percentage of cases in which one canal divided into two, then rejoined to form one (Vertucci type VI).

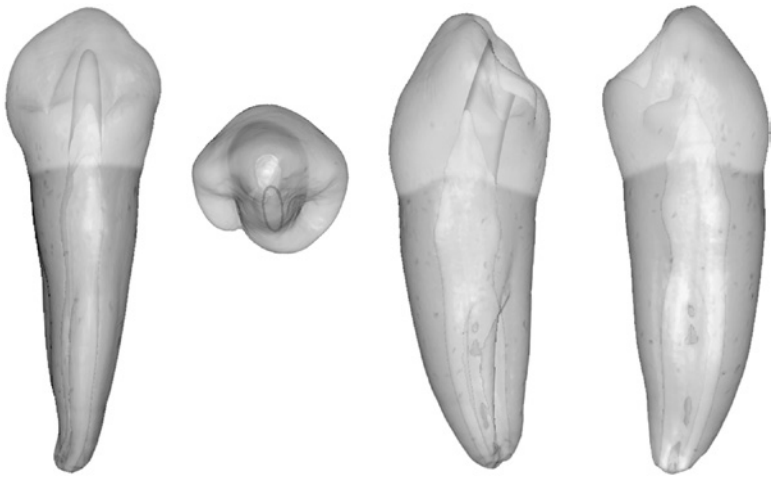

FIG. 5-136 Mandibular first premolar. Average time of eruption—10 to 12 years; average age of calcification—12 to 13 years; average length—21.6 mm. Root curvature (most common to least common): straight, distal, buccal.

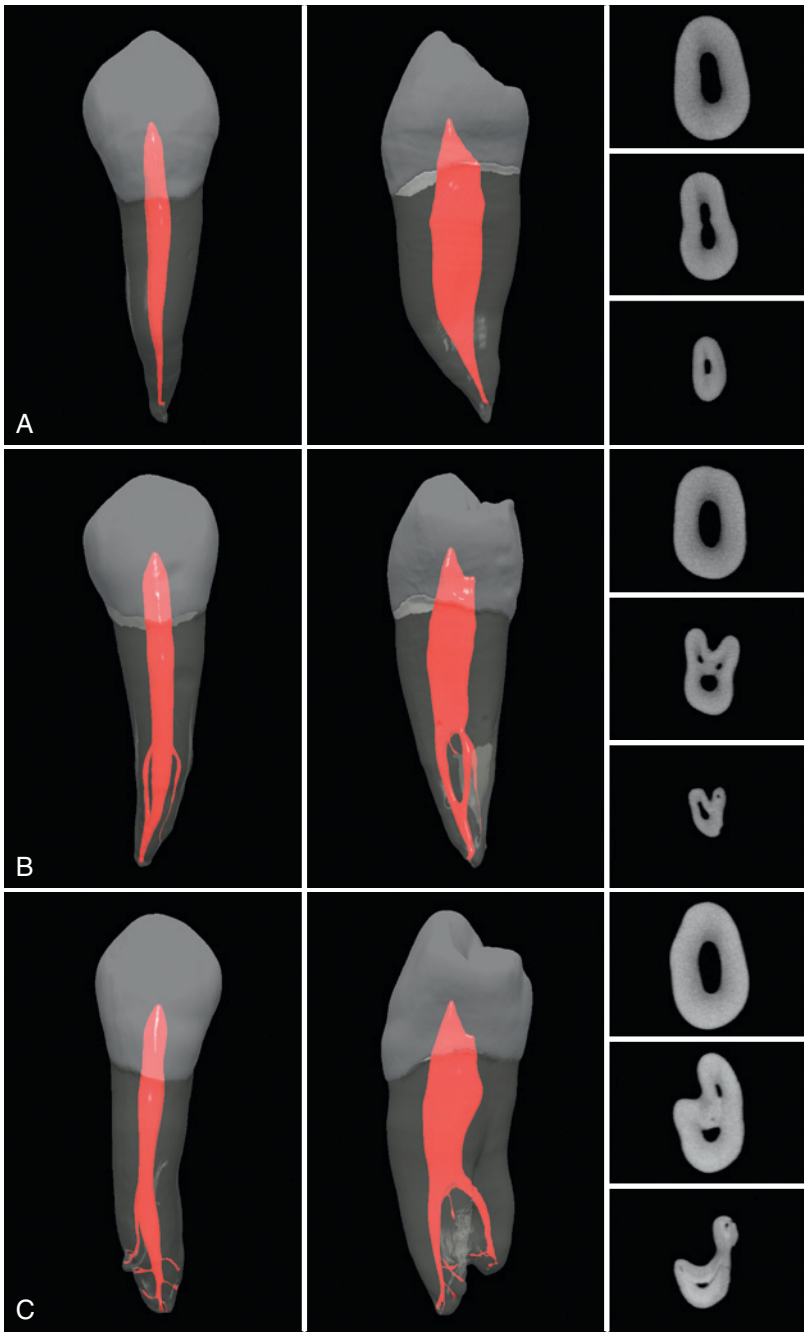

FIG. 5-137 μCT scans of mandibular first preomlars. **A**, Common anatomic presentation. **B**, First premolar with significant canal deviations in the middle to apical third before returning to a single large canal apically and a small deviating canal to the proximal. **C**, First premolar with a branching main canal lingually and multiple accessory canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-12 online at the Expert Consult site for rotational views of these teeth.)

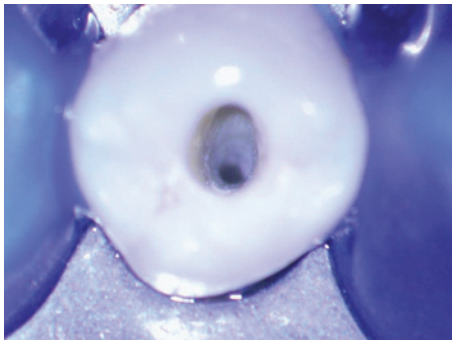

FIG. 5-138 Access cavity for a mandibular first premolar as viewed through the dental operating microscope: one orifice. (×5.1.)

FIG. 5-139 Two canals. (Courtesy Dr. Raed Kasem, Clearwater, Fla.)

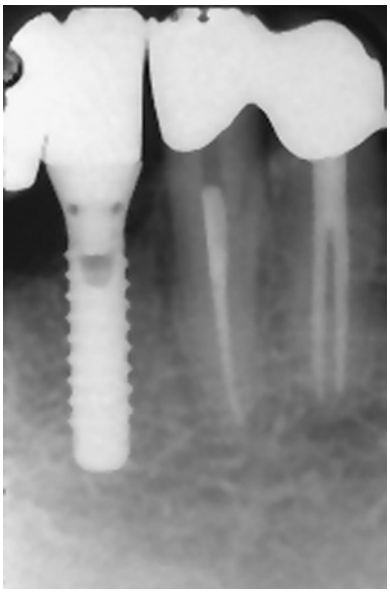

FIG. 5-140 Single canal that has divided into two.

FIG. 5-141 Three canals.

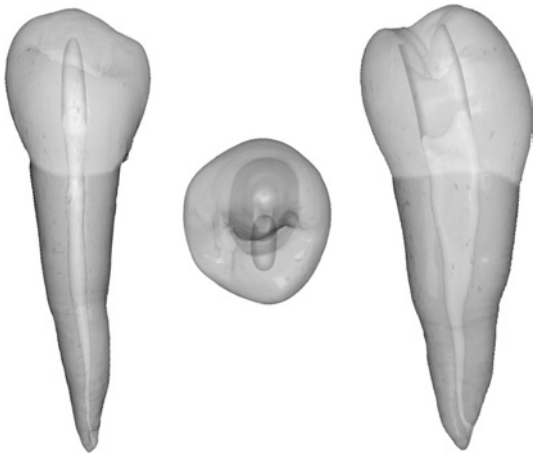

FIG. 5-142 Mandibular second premolar. Average time of eruption—11 to 12 years; average age of calcification—13 to 14 years; average length—22.3 mm. Root curvature (most common to least common): straight, distal, buccal.

barely invades the poorly developed lingual cusp incline. Mesiodistally the access preparation is centered between the cusp tips. Often the preparation must be modified to allow access to the complex root canal anatomy frequently seen in the apical half of the tooth root.

Mandibular Second Premolar

The mandibular second premolar is similar to the first premolar, with the following differences: the lingual pulp horn usually is larger; the root and root canal are more often oval than round; the pulp chamber is wider buccolingually; and the separation of the pulp chamber and root canal normally is distinguishable compared with the more regular taper in the first premolar (Fig. 5-142). The canal morphology of the mandibular second premolar is similar to that of the first premolar with its many variations: two, three, and four canals and a lingually tipped crown. Fortunately, these variations are found less often in the second premolar (see Table 5-23 online at the Expert Consult site). The μCT scans for the mandibular second premolar can be seen in Fig. 5-143. (See Video 5-13 online at the Expert Consult site for rotational views of these teeth.) The access cavity form for the mandibular second premolar varies

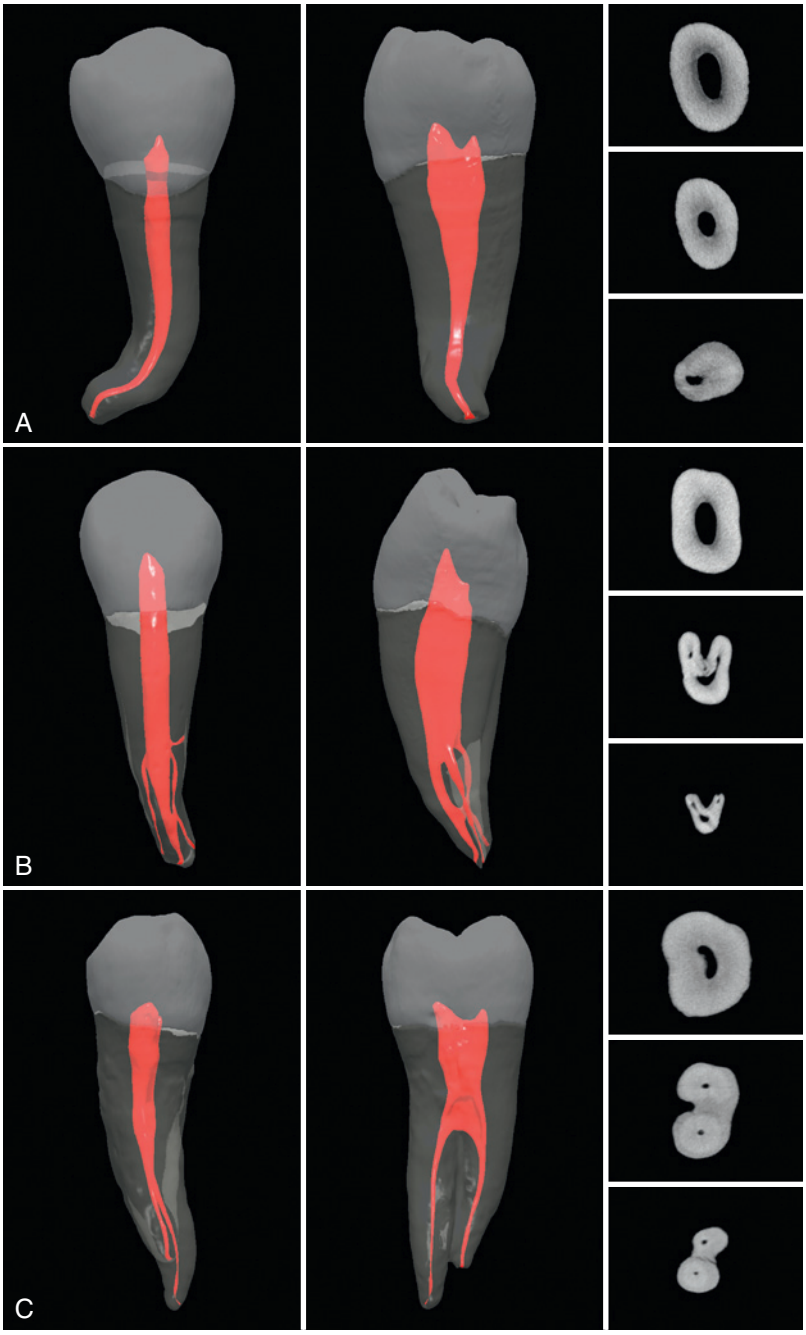

FIG. 5-143 μCT scans of mandibular second premolars. **A**, Common anatomic presentation. **B**, Second premolar with significant canal deviations in the middle to apical third. **C**, Second premolar with fused root that exhibits two distinct canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-13 online at the Expert Consult site for rotational views of these teeth.)

in at least two ways in its external anatomy. First, because the crown typically has a smaller lingual inclination, less extension up the buccal cusp incline is required to achieve straight-line access. Second, the lingual half of the tooth is more fully developed; therefore, the lingual access extension typically is halfway up the lingual cusp incline.[54] The mandibular second premolar can have two lingual cusps, sometimes of equal size. When this occurs, the access preparation is centered mesiodistally on a line connecting the buccal cusp and the lingual groove between the lingual cusp tips. When the mesiolingual cusp is larger than the distolingual cusp, the lingual extension of the oval outline form is just distal to the tip of the mesiolingual cusp (Figs. 5-144 through 5-149).

Mandibular First Molar

The earliest permanent posterior tooth to erupt, the mandibular first molar seems to be the tooth that most often requires an endodontic procedure (vital pulp capping, pulpotomy, root

TABLE 5-23

Studies of Apical Canal Configurations for the Mandibular Second Premolar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)	Four Canals (%)
Vertucci[223]	Clearing	United States	97.5	2.5	—	—
Pineda and Kuttler[170]	Radiographic	Mexico	98.8	1.2	—	—
Calişkan et al.[33]	Clearing	Turkey	93.6	6.4	—	—
Kerekes and Tronstad[103]	Sections	Norway	85	15	—	—
Okumura[154]	Clearing	Japan	98.2	1.8	—	—
Zillich and Dowson[263]	Radiographic and file	United States	88.4 (1)	11.2	0.4	—
Trope et al.[219]	Radiographic	United States				
Black patients			92.2	—	7.8	—
White patients			97.2	—	2.8	—
Holtzman[91]	Clinical	Israel	—	—	—	Case report
El Deeb[56]	Clinical	USA	—	—	Case report	—
Rödig and Hülsmann[181]	Clinical	Germany	—	—	Case report	—
Bram and Fleisher[28]	Clinical	USA	—	—	—	Case report
Rhodes[178]	Clinical	England	—	—	—	Case report
Macri and Zmener[125]	Clinical	Argentina	—	—	—	Case report (5 → 4)‡
Green[81]	Ground sections and microscope	USA	94 (4)	6 (2)	—	—
Sert and Bayirli[190]	Clearing (men)	Turkey	71 (14)	29 (11)	—	—
	Clearing (women)	Turkey	92 (7)	8 (8)	—	—
Nallapati[146]	Clinical	United States	—	—	Case report	—
Fischer and Evans[69]	Clinical	United States	—	—	Case report	—
Sachdeva et al.[185]	Clinical (spiral computed tomography)	India	—	—	—	Case report
Al-Qudah and Awawdeh[9]	Clearing	Jordan	76.8 (4.8)	22.8 (15.3)	0.5	—
DeMoor and Calberson[54]	Clinical (endoscope and DOM)	Belgium	—	—	Case report	—

DOM, Dental operating microscope.
*Percentage of cases in which one canal divided to form two.
†Percentage of cases in which two canals joined to form one.
‡Five canals reconfigured to form four.

FIG. 5-144 Access cavity for a mandibular second premolar as viewed through the dental operating microscope: one canal orifice. (×5.1 magnification with cervical fiberoptic transillumination.)

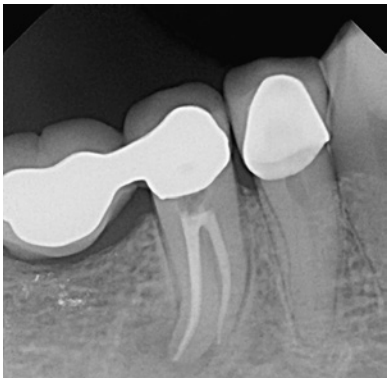

FIG. 5-145 Two canals. (Courtesy Dr. Haider Al Zubaidi, Ocala, Fla.)

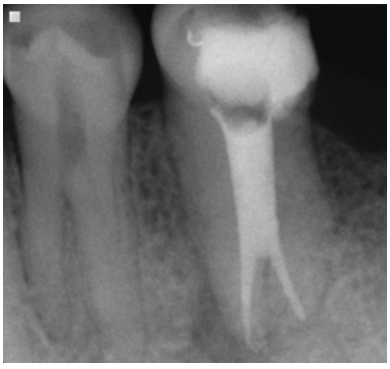

FIG. 5-146 Single canal that has divided at the apex. (Courtesy Dr. Haider Al Zubaidi, Ocala, Fla.)

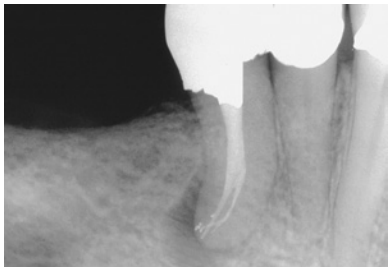

FIG. 5-147 Single canal that has divided and crossed over at the apex.

FIG. 5-148 Three separate canals. (Courtesy Dr. Haider Al Zubaidi, Ocala, Fla.)

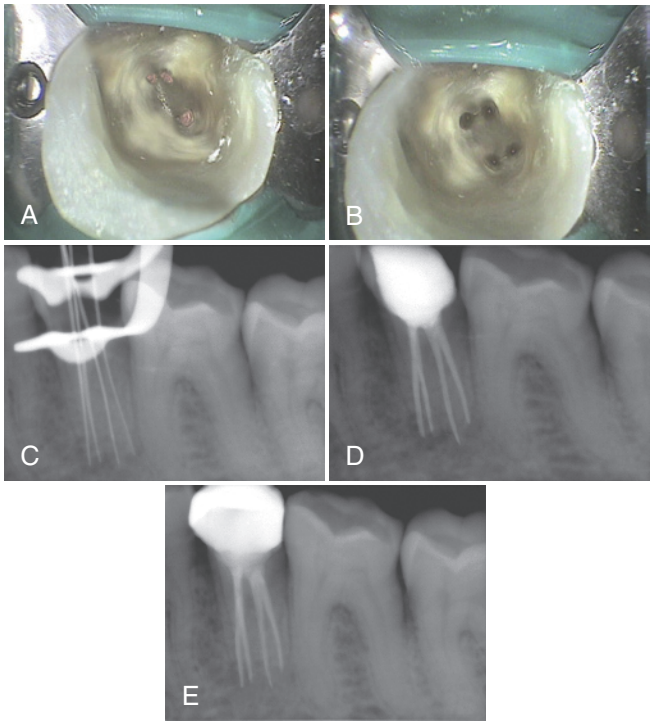

FIG. 5-149 A, Initial access opening. B, Access opening showing orifices of the four root canals. C, Working length determination. D, Immediate posttreatment radiograph. E, Recall radiograph 1 year posttreatment. (From Sachdeva GS, Ballal S, Gopikrishna V, Kandaswamy D: Endodontic management of a mandibular second premolar with four roots and four root canals with the aid of spiral computed tomography: a case report, *J Endod* 34:104, 2008.)

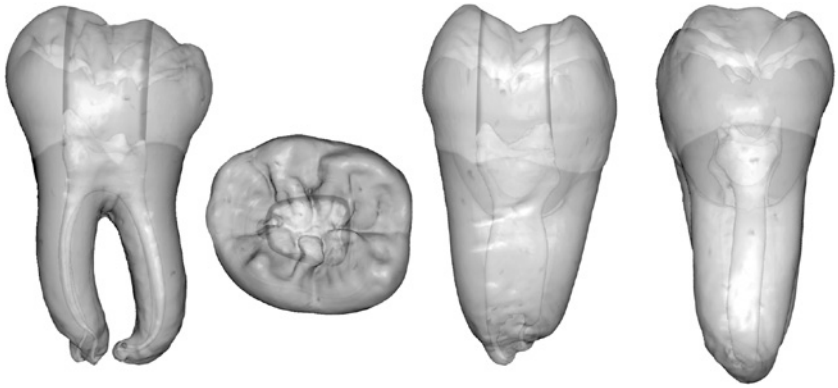

FIG. 5-150 Mandibular first molar. Average time of eruption—6 years; average age of calcification—9 to 10 years; average length—21 mm. Root curvature (most common to least common): mesial root—distal, straight; distal root—straight, distal.

canal); therefore, its morphology has received a great deal of attention (Fig. 5-150).[261] It often is extensively restored, and it is subjected to heavy occlusal stress. Consequently, the pulp chamber frequently has receded or is calcified. The tooth usually has two roots, but occasionally it has three, with two or three canals in the mesial root and one, two, or three canals in the distal root (see Tables 5-24 and 5-25 online at the Expert Consult site). The μCT scans of the mandibular first molar can be seen in Fig. 5-151. (See Video 5-14 online at the Expert Consult site for rotational views of these teeth.)

The canals in the mesial root are the mesiobuccal and mesiolingual canals. A middle mesial (MM) canal sometimes is present in the developmental groove between the other mesial canals,[148] but it may only represent a wide anastomosis between the two mesial canals.[54] The incidence of an MM canal ranges from 1%[223] to 15%.[77,209] The canals in the distal root include the distal canal (if only one canal is present) and the distobuccal and distolingual and middle distal canals (if more than one is present).[75] The orifices to these canals are connected by a developmental groove. Orifices to all canals usually are located in the mesial two thirds of the crown, and the pulp chamber floor is roughly trapezoid or rhomboid. Usually four pulp horns are present.

The presence of two separate distal roots is rare, but this does occur. In such cases the distolingual root is smaller than the distobuccal root and usually more curved. Also, the distolingual root often has a sharp apical hook toward the buccal side that is not obvious on radiographs (Fig. 5-152). The mesial root, the wider of the two roots, curves mesially from the cervical line to the middle third of the root and then angles distally to the apex. The buccal and lingual surfaces are convex throughout their length, whereas the distal surface of the mesial root and the mesial surface of the distal root have a root concavity, which makes the dentin wall very thin.

The mesial canal orifices usually are well separated in the main pulp chamber and connected by a developmental groove.[40] The mesiobuccal orifice is commonly under the mesiobuccal cusp, whereas the mesiolingual orifice generally is found just lingual to the central groove. On occasion an middle mesial canal orifice is present in the groove between the two mesial orifices (Figs. 5-152 through 5-159). The clinician must always check for such an orifice after shaping and cleaning the main root canals. A bur is used to remove any protuberance from the mesial axial wall that would prevent direct access to the developmental groove between the two mesial orifices. Magnification is a tremendous aid during the exploration of this developmental groove with the sharp tip of an endodontic explorer. If a depression or orifice is located, the groove can be troughed with ultrasonic tips, at the expense of the mesial aspect, until a small file can negotiate the space.

When only one distal canal is present, the orifice is oval buccolingually and the opening generally is located distal to the buccal groove. This orifice usually can be explored from the mesial with either an endodontic explorer or a small K-file. If the file tip takes a sharp turn in a distobuccal or distolingual direction, the clinician should search for yet another orifice; in rare cases a mesiodistal canal orifice is present.

If three main root canals are present in this tooth, each is oval in the cervical and middle thirds of the root and round in the apical third. If two canals (distobuccal and distolingual) are present in the distal root, they usually are more round than oval for their entire length. The mesial root canals usually are curved, with the greatest curvature in the mesiobuccal canal. This canal can have a significant curvature in the buccolingual plane that may not be apparent on radiographs. Such a curvature usually can be detected with precurved pathfinder instruments.

Multiple accessory foramina may be located in the furcation of the mandibular molars. These foramina usually are impossible to clean and shape directly; they are rarely seen, except occasionally on a posttreatment radiograph if they have been filled with root canal sealer or thermoplastic filling material. Because sodium hypochlorite solutions can dissolve organic debris, the pulp chamber should be thoroughly exposed to allow the solution to reach the tiny openings. Fractures occasionally occur, which are quite visible with magnification, on proximal marginal ridges and extend down the root or under the lingual cusps.

The access cavity for the mandibular first molar typically is trapezoid or rhomboid, regardless of the number of canals present.[247] When four or more canals are present, the corners of the trapezoid or rhombus should correspond to the positions of the main orifices. Mesially the access need not invade the marginal ridge, except if access to the tooth itself is compromised. Distal extension must allow straight-line access to the distal canal or canals. The buccal wall forms a straight connection between the mesiobuccal and distobuccal orifices, and the lingual wall connects the mesiolingual and distolingual orifices without bowing.

TABLE 5-24

Studies of Apical Canal Configurations for the Mesial Root of the Mandibular First Molar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)	Four Canals (%)
Vertucci[223]	Clearing	USA	40 (28)	59 (8)	1	—
Pineda and Kuttler[170]	Radiographic	Mexico	43 (30)	57 (7)	—	—
Calişkan et al.[33]	Clearing	Turkey	40 (37)	56.6 (7)	3.4	—
Skidmore and Bjorndal[196]	Cast resin	USA	44.5 (38)	55.5	—	—
Pomeranz et al.[172]	Clinical	USA	—	1.6	1.6	—
Wasti et al.[236]	Clearing	Pakistan	(23)	73.7	3.3	—
Zaatar et al.[259]	Clearing	Kuwait	63.3 (57)	34.7	—	—
Sperber and Moreau[203]	Ground sections	Senegal	(16)	84	—	—
Gulabivala et al.[82]	Clearing	Pakistan	43 (29)	50 (6)	7	—
Beatty and Krell[22]	Clinical	USA	—	—	Case report	—
Martinez-Berná and Badanelli[133]	Clinical	Spain	—	—	Case report	—
Fabra-Campos[59]	Clinical	Spain	—	—	Case report	—
Baugh and Wallace[19]	Clinical	USA	—	Case report (3 → 2)§	—	—
Ricucci[179]	Clinical	Italy	—	Case report (3 → 2)§	—	—
DeGrood and Cunningham[51]	Clinical	USA	—	Case report (3 → 2)§	—	—
Jacobsen et al.[95]	Clinical	USA	—	Case report (3 → 2)§	Case report	Case report
Reeh[177]	Clinical	USA	—	—	—	Case report
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	52 (52) 49 (46)	44 (3) 51 (6)	3 —	— —
Walker[232]	Radiographic	Southern China	24	75	1	—
da Costa et al.[47]	Clearing	Brazil	54.6	45.4	—	—
Pattanshetti et al.[159]	Clinical	Kuwait	(100)	—	—	—

*Numbers in parentheses are the percentage of cases in which two canals joined to form one.
†Numbers in parentheses are the percentage of cases in which one canal divided to form two.
‡Studied incidence in middle mesial canals only.
§Three canals reconfigured to form two; 2% represents three canals that join into two.

TABLE 5-25

Studies of Apical Canal Configurations for the Distal Root for the Mandibular First Molar

Authors	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)
Vertucci[223]	Clearing	United States	85 (15)	15 (8)	—
Pineda and Kuttler[170]	Radiographic	Mexico	85.7 (12.7)	14.3 (8.6)	—
Calişkan et al.[33]	Clearing	Turkey	81.4 (33)	16.9 (7)	1.7
Zaatar et al.[259]	Clearing	Kuwait	83.7 (8)	16.3	—
Sperber and Moreau[203]	Ground sections	Senegal	78	(22)	0.2
Gulabivala et al.[82]	Clearing	Pakistan	83.3 (4)	16 (3)	0.7
Wasti et al.[236]	Clearing	Pakistan	56.7 (27)	43.3 (20)	—
Skidmore and Bjorndal[196]	Cast resin	United States	88.8 (18)	11.2	—
Ricucci[179]	Clinical	Italy	Case report	—	—
DeGrood and Cunningham[51]	Clinical	United States	—	Case report (3 → 2)‡	—
Martinez-Berná and Badanelli[133]	Clinical	Spain	Case report (3 → 1)§	Case report (3 → 2)‡	—
Beatty and Krell[22]	Clinical	United States	—	Case report	—
Reeh[177]	Clinical	United States	—	—	Case report
Beatty and Interian[21]	Clinical	United States	—	—	Case report
Friedman et al.[71]	Clinical	Israel	—	—	Case report
Stroner et al.[206]	Clinical	United States	—	—	Case report
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	89 (36) 85 (31)	9 (3) 15 (2)	2 —
Walker[232]	Radiographic	Southern China	72	28	—
da Costa et al.[47]	Clearing	Brazil	90.5	9.5	—
Pattanshetti et al.[159]	Clinical	Kuwait	95.5 (40)	4.5	—

*Numbers in parentheses are the percentage of cases in which two canals joined to form one.
†Numbers in parentheses are the percentage of cases in which one canal divided to form two.
‡Three canals reconfigured to form two.
§Three canals reconfigured to form one.

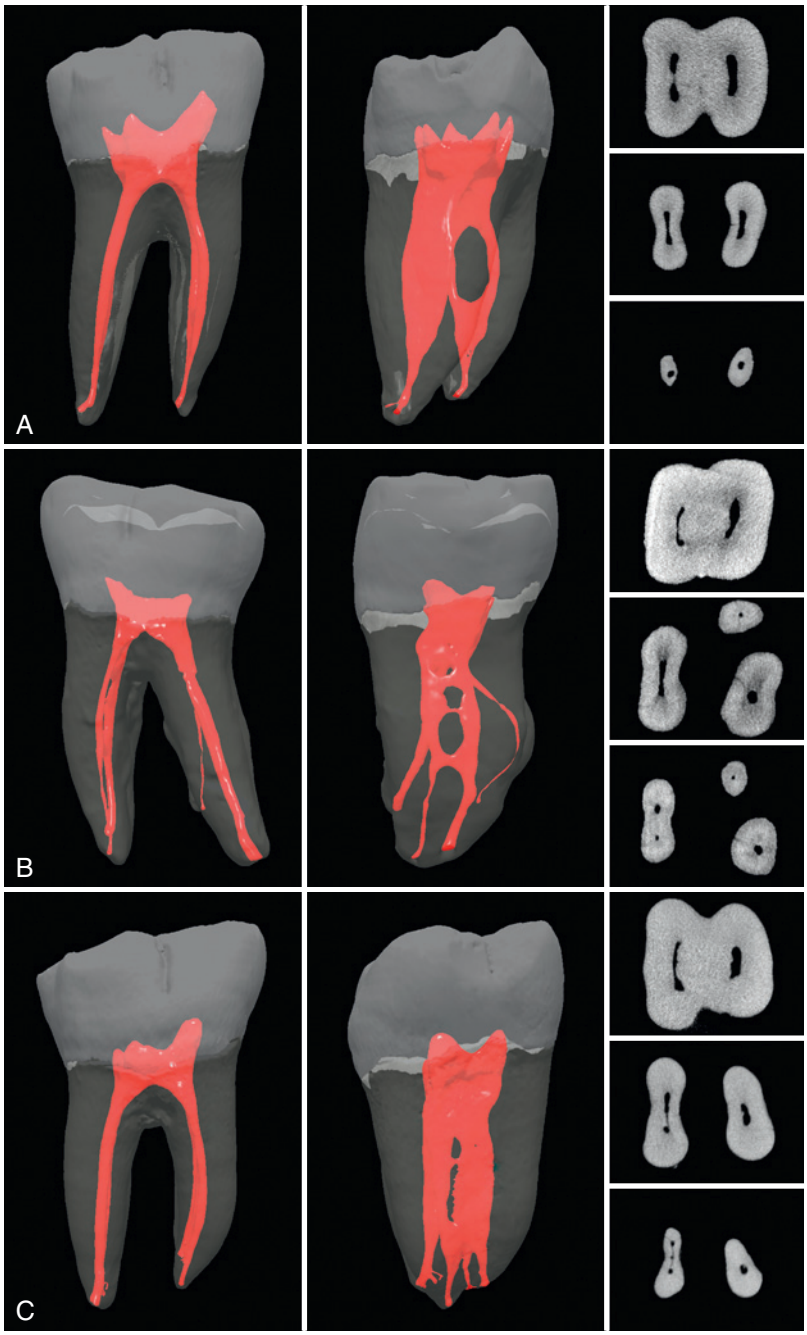

FIG. 5-151 μCT scans of mandibular first molars. **A**, Common anatomic presentation. **B**, First molar with three main canals and a deviant fourth canal/fourth root. **C**, First molar with wide connections or anastomoses between the mesial canals, demonstrating multiple canal exits. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-14 online at the Expert Consult site for rotational views of these teeth.)

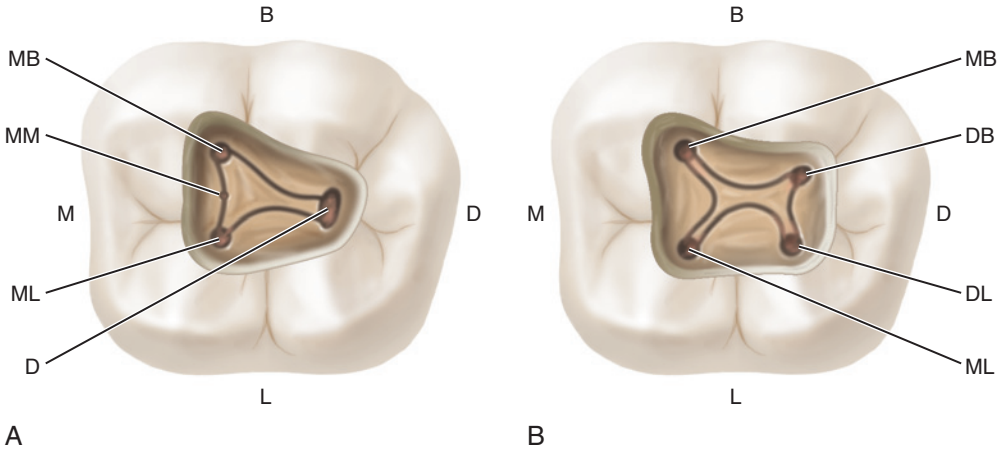

FIG. 5-152 Access cavities for the mandibular first molar. **A,** Three mesial canal orifices and one distal canal orifice. **B,** Two mesial and two distal canal orifices. *B,* Buccal; *D,* distal, distal orifice; ***DB,*** distobuccal orifice; *DL,* distolingual orifice; *L,* labial; *M,* mesial; *MB,* mesiobuccal orifice; *ML,* mesiolingual orifice; *MM,* middle mesial orifice.

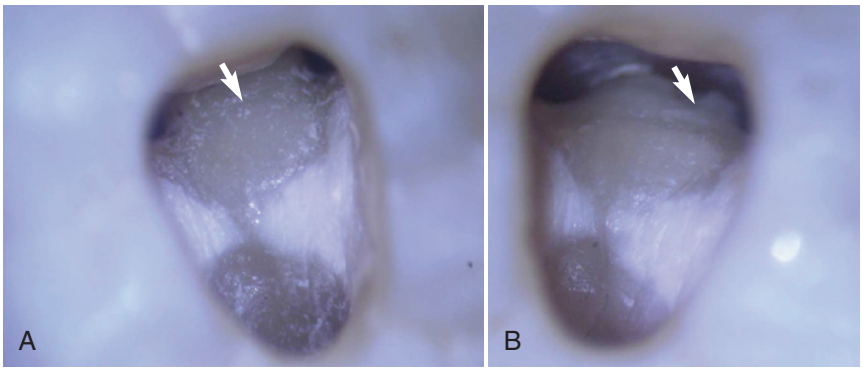

FIG. 5-153 Access cavity for a mandibular first molar as viewed through the dental operating microscope. **A,** Three canal orifices (*MB, ML,* and *D*) and a dentinal projection *(arrow)* can be seen. (×5.1.) A possible middle mesial *(MM)* orifice may be present along the mesial groove. **B,** Troughing of the mesial groove allows identification of the MM canal orifice *(arrow).* (×5.1.)

FIG. 5-154 Two mesial and two distal canals.

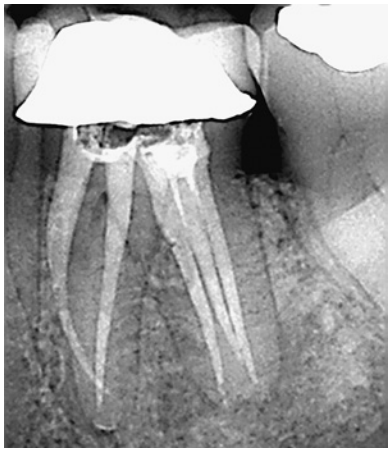

FIG. 5-155 Three canals in the distal root. (Courtesy Dr. Raed Kasem, Clearwater, Fla.)

FIG. 5-156 Three mesial canals.

FIG. 5-157 Three orifices in the mesial root and three orifices in the distal root. (Courtesy Dr. Haider Al Zubaidi, Ocala, Fla.)

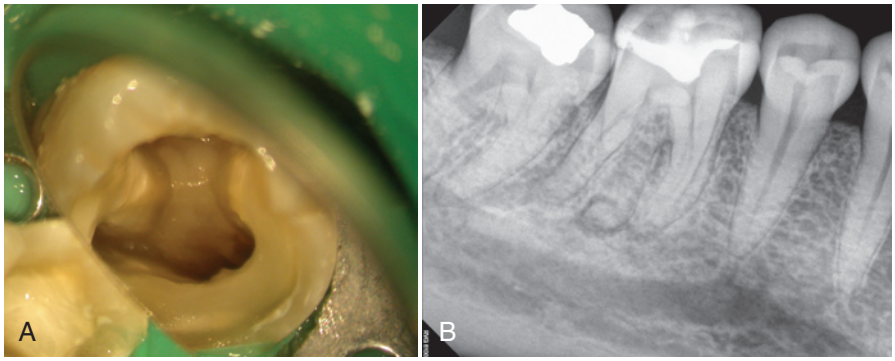

FIG. 5-158 A, Radix entomolaris. Notice lingual position of its orifice in relation to the two canals in the distal root. B, Radiograph with obvious radix entomolaris on mandibular first molar that curves to the buccal. The reader is referred to a recent publication that details extensively this type of anatomy (see following credit for part B). (A courtesy Dr. William J. Aippersbach, Venice, Fla; B from Abella F, Patel S, Durán-Sindreu F et al: Mandibular first molars with disto-lingual roots: review and clinical management, *Int Endod J* 45:963, 2012.)

FIG. 5-159 Pretreatment radiograph. Notice position and appearance of the distolingual root. (Courtesy Dr. William J. Aippersbach, Venice, Fla.)

A variation in root morphology is the presence of an extra distolingual root.[203] Usually this root has a type 1 canal configuration. Two thirds of the first mandibular molars found in a Chinese population had this variation.[251] Similarly, this distolingual root occurred in 4% of mandibular first molars of a Kuwaiti population.[159] These results confirm the observation that East Asian populations have more three-rooted mandibular first molars than do other racial groups.[8]

Mandibular molars, mainly first molars, may also have an additional root located lingually or buccally. Although this is a rare occurrence in Caucasian populations, it is more common in Asian populations.[220] The radix entomolaris (RE) is a supernumerary root located distolingually in mandibular molars (see Fig. 5-158),[10,32] whereas the radix paramolaris (RP) is an extra root located mesiobuccally. Each root usually contains a single root canal. The orifice of the RE is located distolingually to mesiolingually from the main canal or canals of the distal root; the orifice of the RP is located mesiobuccally to distobuccally from the main mesial root canals. A dark line or groove from the main root canal on the pulp chamber floor leads to

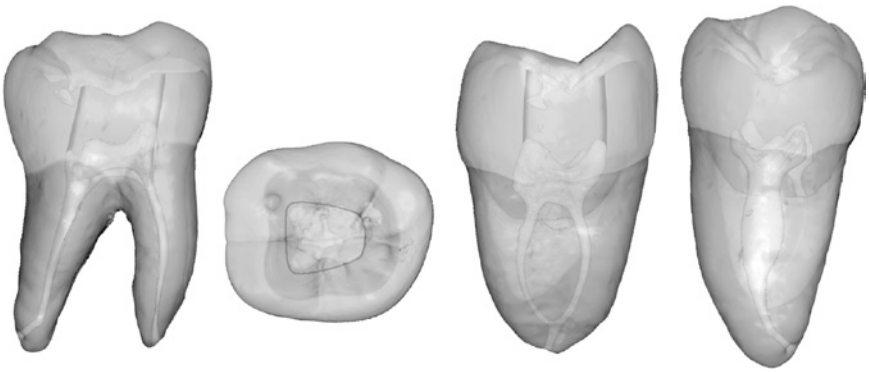

FIG. 5-160 Mandibular second molar. Average time of eruption—11 to 13 years; average age of calcification—14 to 15 years; average length—19.8 mm. Root curvature (most common to least common): mesial root—distal, straight; distal root—straight, distal, mesial, buccal; single root—straight, distal, bayonet, lingual.

these orifices.[32] These anatomic variations present definite challenges to therapy because of their orifice inclination and root canal curvature. The canal can be straight, may have a coronal curvature, or can have separate coronal and apical curvatures.[52]

Mandibular Second Molar

The mandibular second molar is somewhat smaller coronally than the first molar and tends to be more symmetric (Fig. 5-160). This tooth is identified by the proximity of its roots. The two roots often sweep distally in a gradual curve, with the apices close together. In some cases only one root is present. The degree of canal curvature and the configuration were studied in the mesial roots of 100 randomly selected mandibular first and second molars; 100% of the specimens showed curvature in both buccolingual and mesiodistal views.[45]

The pulp chamber and canal orifices of the mandibular second molar generally are not as large as those of the first molar. This tooth may have one, two, three, or four root canals (see Table 5-26 online at the Expert Consult site). The μCT scans for the mandibular second molar can be seen in Fig. 5-161. (See Video 5-15 online at the Expert Consult site for rotational views of these teeth.) Figs. 5-162 through 5-166 provide additional variations and views of this tooth.

The two mesial orifices are located closer together. In some mandibular second molars with single or fused roots, a file placed in the mesiobuccal canal may appear to be in the distal canal. This happens because the two canals sometimes are connected by a semicircular slit, a variation of the C-shaped canal[137,234,262] that often occurs in this tooth. The distal aspect of the mesial root and the mesial aspect of the distal root have concavities.

Mandibular second molars may have one to six canals, although the most prevalent configurations are two, three, and four canals (see Fig. 5-162). When three canals are present, the access cavity is similar to that for the mandibular first molar, although perhaps a bit more triangular and less rhomboid. The distal orifice is less often ribbon shaped buccolingually; therefore, the buccal and lingual walls converge more aggressively distally to form a triangle. The second molar may have only two canals, one mesial and one distal, in which case the orifices are nearly equal in size and line up in the buccolingual center of the tooth. The access cavity for a two-canal second molar is rectangular, wide mesiodistally and narrow buccolingually. The access cavity for a single-canal mandibular second molar is oval and is lined up in the center of the occlusal surface.

Mandibular Third Molar

The mandibular third molar is anatomically unpredictable and must be evaluated on the basis of its root formation (Fig. 5-167).[110] Fused short, severely curved, or malformed roots often support well-formed crowns. This tooth may have one to four roots and one to six canals (see Table 5-27 online at the Expert Consult site). The μCT scans for the mandibular third molar can be seen in Fig. 5-168. (See Video 5-16 online at the Expert Consult site for rotational views of these teeth.) Additional variations are seen in Figs. 5-169 through 5-171.

C-shaped canals also can occur (Fig. 5-172). Most of these teeth can be successfully root treated, regardless of anatomic irregularities; however, the long-term prognosis is determined by the root surface volume in contact with bone. The clinician must weigh the benefit of treatment against the prognosis.

The anatomy of the mandibular third molar is unpredictable; therefore, the access cavity can take any of several shapes. When three or more canals are present, a traditional rounded triangle or rhomboid shape is typical. When two canals are present, a rectangular shape is used. For single-canal molars, an oval shape is customary.

Teeth with C-Shaped Root Canal Systems

The main cause for C-shaped roots and canals is the failure of Hertwig's epithelial root sheath to fuse on either the buccal or lingual root surface (Fig. 5-173). The C-shaped canal system can assume many variations in its morphology (Fig. 5-174, *A*). Further cross section of types I, II, and III are seen in Fig. 5-174, *B* to *D*. The original classification[133] has been modified and has produced a more detailed description of C-shaped root and canal morphology. The C-shaped canal configuration can vary along the root depth so that the appearance of the orifices may not be good predictors of the actual canal anatomy.[62,64,137,234,262]

Category I (C1): The shape is an uninterrupted "C" with no separation or division (see Fig. 5-163, *A*).

Category II (C2): The canal shape resembles a semicolon resulting from a discontinuation of the "C" outline (see

TABLE 5-26

Studies of Apical Canal Configurations of the Mandibular Second Molar

Authors	Type of Study	Country	Root	One Canal (%)*	Two Canals (%)†	Three Canals (%)	Other (%)
Vertucci[223]	Clearing	United States	Mesial	65 (38)	35 (9)	—	—
Pineda and Kuttler[170]	Radiographic	Mexico	Mesial	78.6 (21)	21.4 (8)	—	—
Caliskan et al.[33]	Clearing	Turkey	Mesial	41.2 (19)	56.9	2	—
Weine et al.[242]	Radiographic with file	USA	Mesial	56 (52)	40	—	1.3 (one canal); 2.7 (C shaped)[131]
Manning[130]	Clearing	Australia	Mesial	73.5 (32)	24.5 (10)	2	—
Wells and Bernier[246]	Clinical	USA	Mesial	Case report	—	—	—
Beatty and Krell[22]	Clinical	USA	Mesial	—	—	Case report	—
Pomeranz et al.[72]	Clinical	USA	Mesial	—	2.6 (3 → 2)§	2.6	—
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	Mesial Mesial	70 (56) 61 (50)	30 (7) 39 (6)	— —	
Vertucci[223]	Clearing	USA	Distal	95 (3)	4 (1)	—	
Pineda and Kuttler[170]	Radiographic	Mexico	Distal	96.5 (2)	3.5 (0.5)	—	
Caliskan et al.[33]	Clearing	Turkey	Distal	96.1 (14)	3.9	—	
Weine et al.[242]	Radiographic with file	USA	Distal	94.6 (9)	1.3	1.3 (one canal); 2.7 C shaped	
Manning[130]	Clearing	Australia	Distal	98.3	(1.7)	—	
Beatty and Krell[22]	Clinical	USA	Distal	—	Case report	—	
Wells and Bernier[246]	Clinical	USA	Distal	Case report (M and D join)	—	—	
Sert and Bayirli[190]	Clearing (men) Clearing (women)	Turkey Turkey	Distal Distal	96 (18) 93 (19)	4 (2) 7 (5)	— —	

M, Medial; *D,* distal.
*Numbers in parentheses are the percentage of cases in which two canals joined to form one.
†Numbers in parentheses are the percentage of cases in which one canal divided to form two.
‡Studied incidence in middle mesial canals only.
§Three canals reconfigured to form two.

TABLE 5-27

Studies of Apical Canal Configurations for the Mandibular Third Molar

Author	Type of Study	Country	Number of Roots	C-Shape	Number of Canals (%)					
					1	2	3	4	5	6
Sidow et al.[192]	Clearing	United States	1	2	3.3	6.7	4.7	—	—	—
			2	2	—	10	46.7	15.3	2	0.7
			3	—	—	—	4	0.7	0.7	—
			4	—	—	—	—	0.7	0.7	—
Pineda and Kuttler[170]	Radiographic	Mexico	Mesial		83.5 (18*)	16.5 (3†)	—	—	—	—
			Distal		95.7 (4*)	4.3(2†)	—	—	—	—
Plotino[171]	Clinical	Italy	3 Mesial	—	—	—	Case report	—	—	—
			1 Distal	—	—	—	Case report	—	—	—

*Percentage of cases in which two canals joined to form one.
†Percentage of cases in which one canal divided to form two.

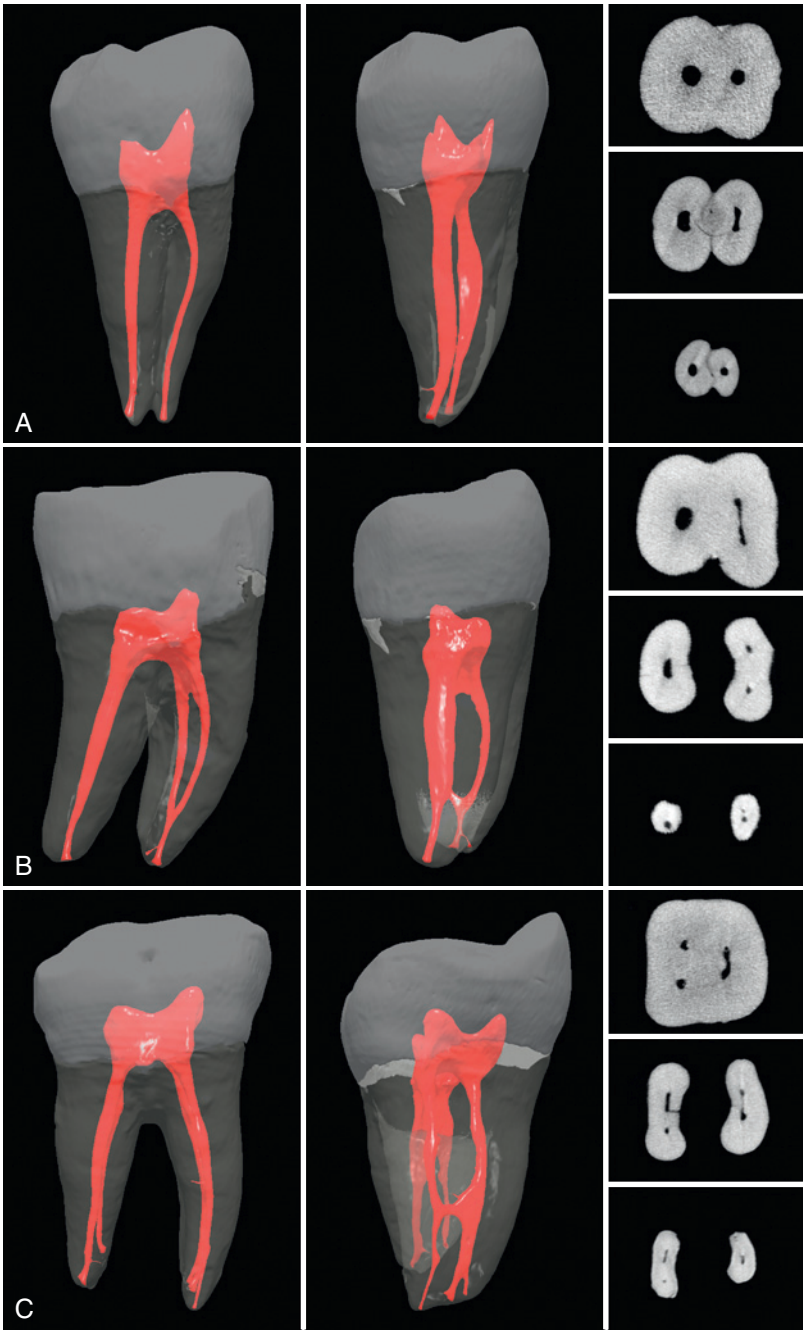

FIG. 5-161 μCT scans of mandibular second molars. **A**, Two-canal second molar with fused roots. **B**, Second molar with three initial canals ending in one canal apically in both roots. **C**, Second molar with four distinct canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-15 online at the Expert Consult site for rotational views of these teeth.)

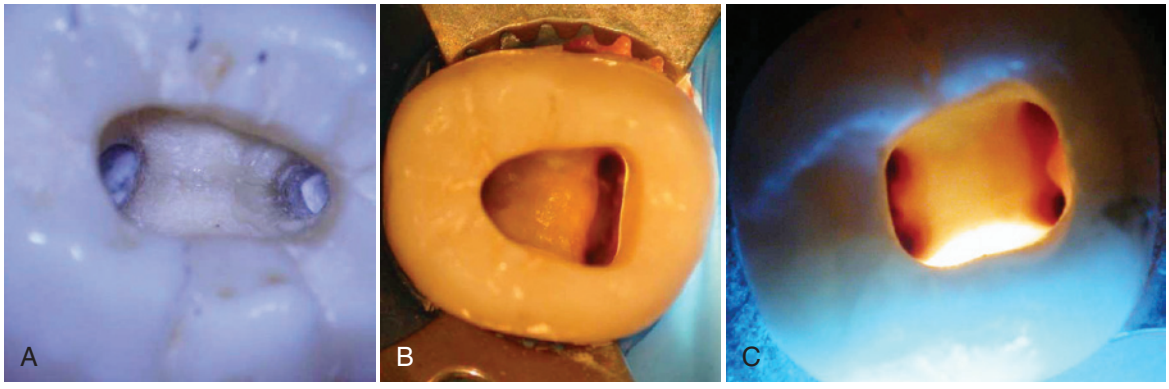

FIG. 5-162 Access cavity for a mandibular second molar as viewed through the dental operating microscope. **A**, Two canal orifices (*M* and *D*). (×5.1.) **B**, Three canal orifices (*MB, ML,* and *D*). (×3.4 magnification with cervical fiberoptic transillumination.) **C**, Four canal orifices identified (*MB, ML, DB,* and *DL*). (×5.1 magnification with cervical fiberoptic transillumination.)

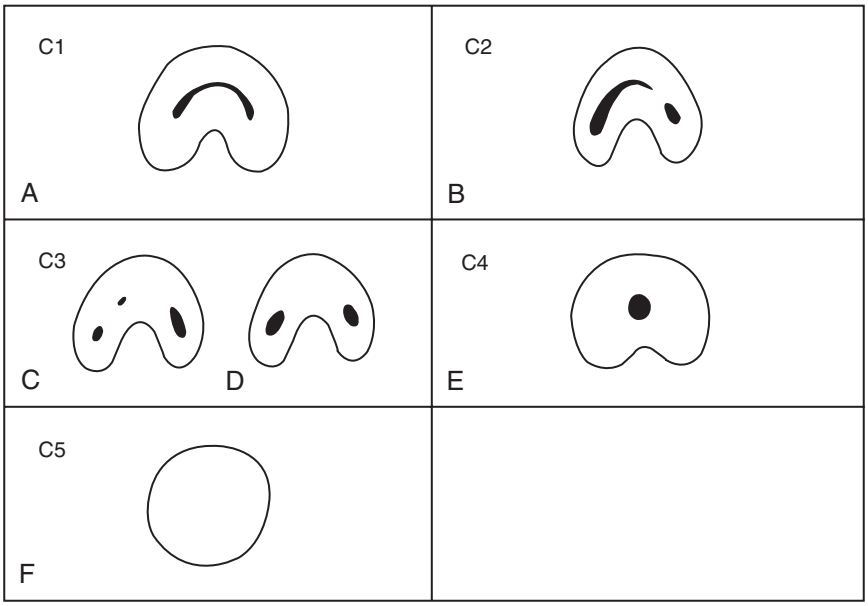

FIG. 5-163 Classification of C-shaped canal configuration. (From Fan B, Cheung G, Fan M, Gutmann J: C-shaped canal system in mandibular second molars. I. Anatomical fractures, *J Endod* 30:899, 2004.)

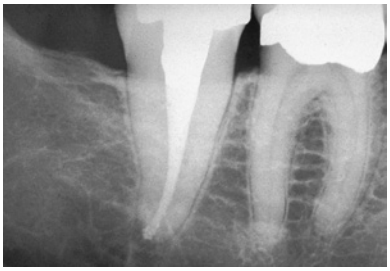

FIG. 5-164 Anastomosis of all canals into one.

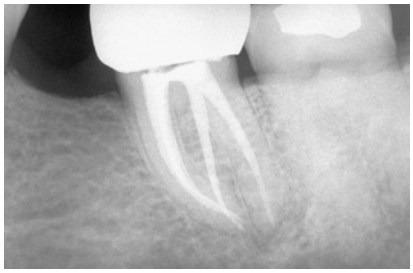

FIG. 5-166 Fusion of mesial canals at the apex.

FIG. 5-165 Two canals with an accessory canal at the distal root apex.

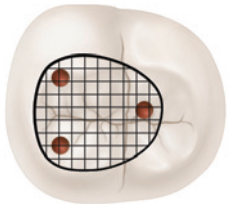

FIG. 5-167 Mandibular third molar. Average time of eruption—17 to 21 years; average age of calcification—18 to 25 years; average length—18.5 mm.

Fig. 5-163, *B*), but either angle α or β should be no less than 60 degrees.

Category III (C3): Two or three separate canals (see Fig. 5-163, *C* and *D*) and both angles, α and β, are less than 60 degrees.

Category IV (C4): Only one round or oval canal is in the cross-section (see Fig. 5-163, *E*).

Category V (C5): No canal lumen can be observed (is usually seen near the apex only) (see Fig. 5-163, *F*).

The C-shaped root canal system was first reported in 1979 in a maxillary molar.[43] Most C-shaped canals occur in the mandibular second molar (Figs. 5-175 through 5-180),[234] but they also have been reported in the mandibular first molar,[146] the maxillary first and second molars, and the mandibular first premolar.* One study reported the incidence of C-shaped canal anatomy in maxillary first molars as 2 in 2175 (0.092%); this study also determined that the DB and palatal orifices were connected by a common groove (see Fig. 5-176, *B*).[53] Investigators who examined 309 Chinese maxillary second molars found C-shaped root canals in 4.9%.[254]

C-shaped mandibular molars are so named because of the cross-sectional morphology of their fused roots and their root

*References 62, 63, 65, 66, 146,150, 252, 253, 261, and 262.

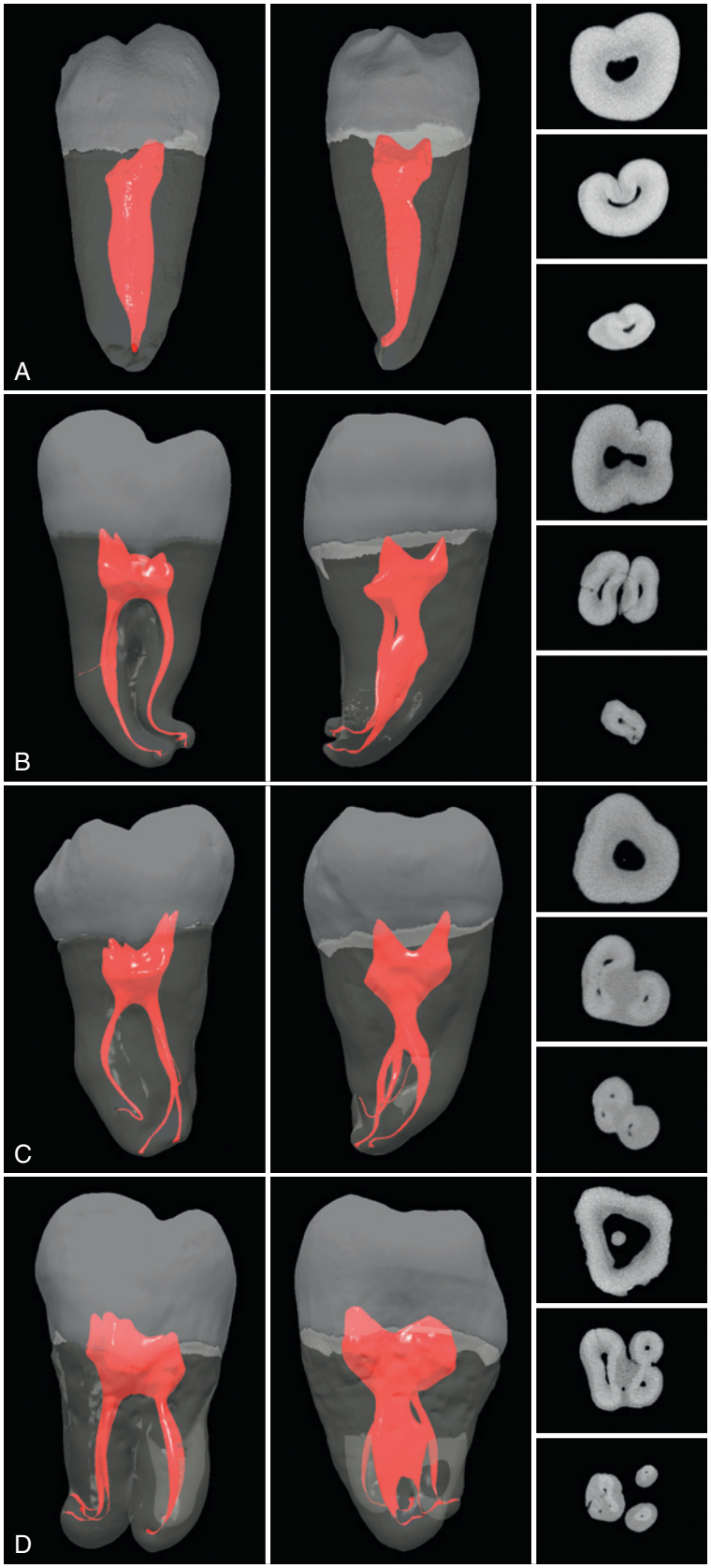

FIG. 5-168 μCT scans of mandibular third molars represent multiple variations for this tooth. **A**, Single C-shaped type canal. **B**, Complex anatomy with significant canal curvatures apically. **C**, Three canals that curve in multiple directions. **D**, Flattened, ribbon-shaped canals with significant apical curvatures. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 5-16 online at the Expert Consult site for rotational views of these teeth.)

FIG. 5-169 Five canals. (Courtesy Dr. Paulo Nogueira, São Paulo, Brazil.)

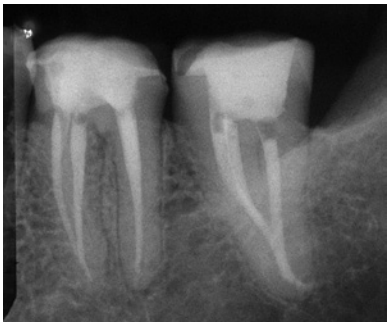

FIG. 5-170 Complex root anatomy. (Courtesy Dr. Paulo Nogueira, São Paulo, Brazil.)

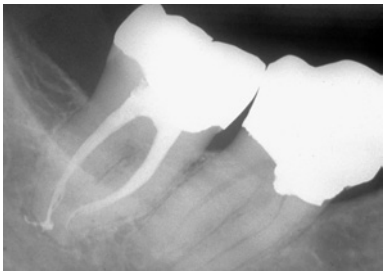

FIG. 5-171 Complex apical anatomy.

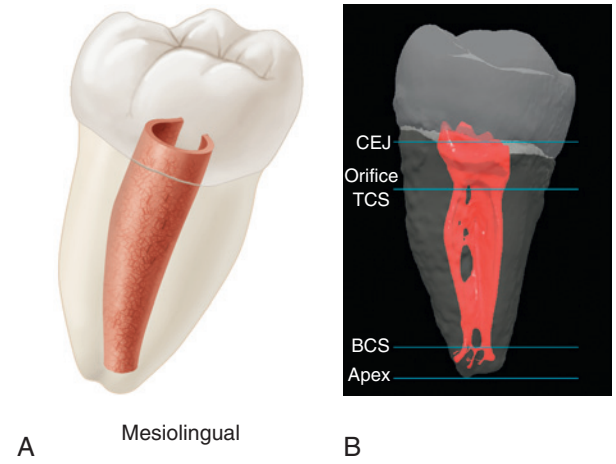

FIG. 5-172 A, Diagram of a C-shaped canal anatomy: one continuous canal from pulp chamber floor to apex. B, One large canal moving from the top of the C-shaped canal *(TCS)* to the apical-most or bottom of C-shaped canal *(BCS),* encompassing most of the canal space.

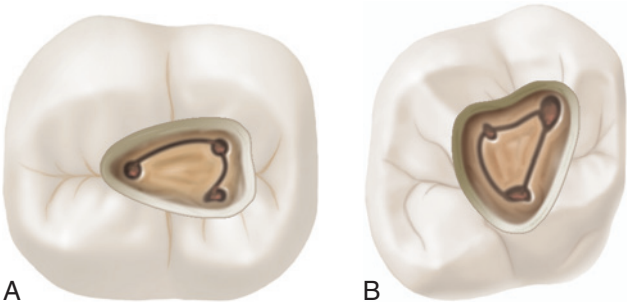

FIG. 5-173 C-shaped canal anatomy. A, Mandibular second molar. B, Maxillary first molar.

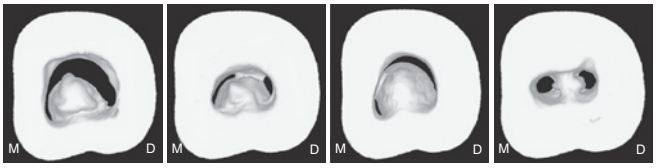

FIG. 5-174 Types of pulpal floor. *M,* Mesial side; *D,* distal side. (From Min Y, Fan B, Cheung G, Gutmann J, Fan M: C-shaped canal system in mandibular second molars. III. The morphology of the pulp chamber floor, *J Endod* 32:1155, 2006.)

canals. Instead of having several discrete orifices, the pulp chamber of a molar with a C-shaped root canal system is a single, ribbon-shaped orifice with an arc of 180 degrees or more. It starts at the mesiolingual line angle and sweeps around either to the buccal or the lingual to end at the distal aspect of the pulp chamber (see Fig. 5-176, *A*). Below the orifice, the root structure can show a wide range of anatomic variations. These can be classified into two basic types: those with a single, ribbon-like, C-shaped canal from orifice to apex, and those with three or more distinct canals below the usual C-shaped orifice. Fortunately, molars with a single swath of canal are the exception rather than the rule. More common is the second type, with discrete canals that take unusual forms.[43] Other investigators determined that C-shaped canals in mandibular second molars can vary in shape and number along the root length.[136]

One study reported a case of a mandibular first molar with a normal mesiolingual orifice and a C-shaped groove that ran continuously from the mesiobuccal orifice along the buccal wall to the distal canal orifice.[18] The groove ran continuously down the root to the apical third, where it divided into two canals. Other researchers reported a C-shaped groove in a mandibular first molar that extended from the DL to the DB orifice and across the buccal surface to the MB orifice.[26] The ML orifice remained separate. Four separate apical foramina were noted. One investigator evaluated 811 endodontically treated mandibular second molars and found that 7.6% had C-shaped canals.[239] Several variants of the C-shaped canal

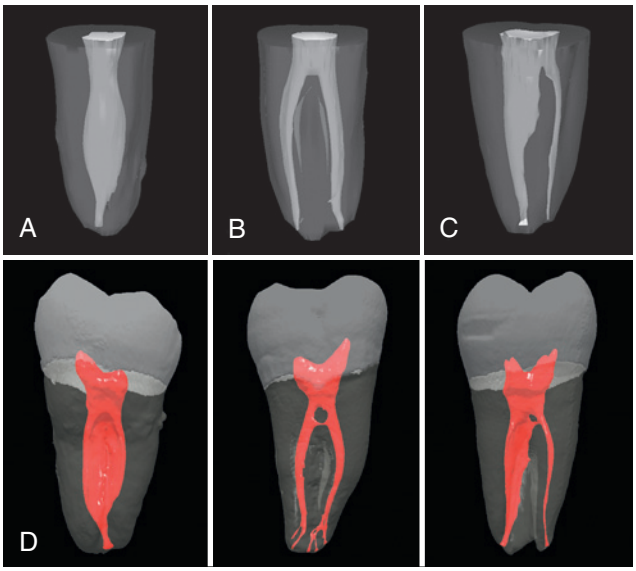

FIG. 5-175 Three-dimensional classification of C-shaped canal configuration. **A,** Merging type. **B,** Symmetrical type. **C,** Asymmetrical type. **D,** Additional C-shaped canal variations. (From Gao Y, Fan B, Cheung G, Gutmann J, Fan M: C-shaped canal system in mandibular second molars. IV. Morphological analysis and transverse measurement, *J Endod* 32:1062, 2006.)

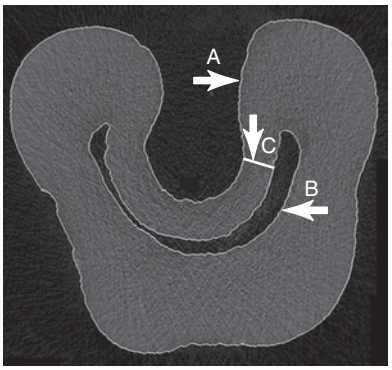

FIG. 5-176 Microcomputed tomographic image of the minimal wall thickness measurement. *A,* Outer root surface. *B,* Inner canal wall. *C,* Thinnest wall thickness. (From Gao Y, Fan B, Cheung G, Gutmann J, Fan M: C-shaped canal system in mandibular second molars. IV. Morphological analysis and transverse measurement, *J Endod* 32:1062, 2006.)

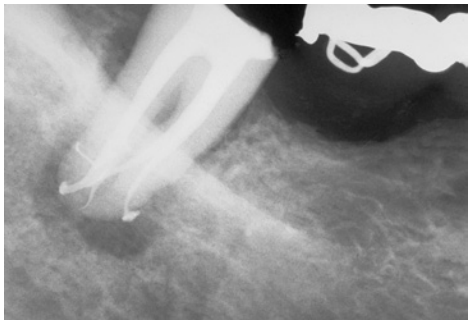

FIG. 5-177 Mandibular second molar with multiple foramina (see also Fig. 5-172, *D*).

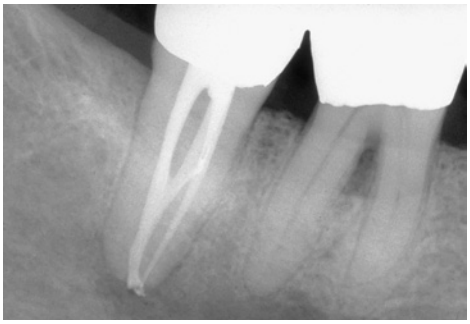

FIG. 5-178 Mandibular second molar with interconnecting canal anatomy.

FIG. 5-179 Pretreatment appearance of a mandibular first molar with a C-shaped canal (see also Fig. 5-175, *D*).

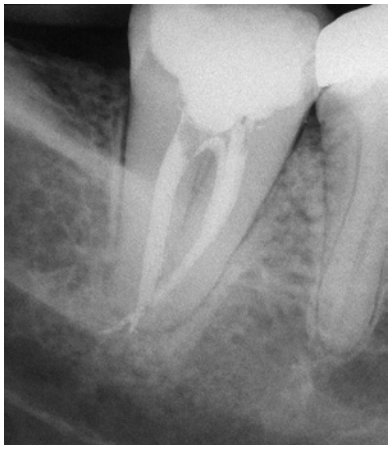

FIG. 5-180 Root canal obturation showing the ribbonlike canal space. (Courtesy Dr. Paulo Nogueira, São Paulo, Brazil.)

morphology were noted, the most common being two or three canals that merged and exited as one canal.[239]

Significant ethnic variation can be seen in the incidence of C-shaped root canal systems. This anatomy is much more common in Asians than in the Caucasian population. Investigators in Japan[106] and China[254] found a 32% incidence of C-shaped canals. Others found the occurrence of C-shaped canals in a Chinese population to be 23% in mandibular first molars and 32% in mandibular second molars.[254,255,257] Another study found a 19% rate in mandibular second molars in Lebanese subjects[85] and an 11% rate in a Saudi Arabian population.[6] Mandibular molars in a Burmese population were found to have C-shaped canals in 22% of teeth.[82] Another investigation

found that 33% of Koreans had a C-shaped canal morphology in mandibular second molars.[105,189]

Four types of pulpal floors were found in mandibular second molars[137]:

Type I: A peninsula-like floor with continuous C-shaped orifice (see Fig. 5-174).

Type II: A buccal, striplike dentin connection between the peninsula-like floor and the buccal wall of the pulp chamber that separates the C-shaped groove into mesial (M) and distal (D) orifices. Sometimes the mesial orifice is separated into a mesiobuccal (MB) orifice and a mesiolingual (ML) orifice by another striplike dentin connection between the peninsula-like floor and the mesial wall of the pulp chamber (most common) (see Fig. 5-174).

Type III: Only one mesial, striplike dentin connection between the peninsula-like floor and the M wall, which separates the C-shaped groove into a small ML orifice and a large MB-D orifice. The MB-D orifice was formed by the merging of the MB orifice and the D orifice (second most common) (see Fig. 5-174).

Type IV: Non–C-shaped floors. One distal canal orifice and one oval or two round mesial canal orifices are present (least common) (see Fig. 5-174).

Not all C-shaped mandibular second molars with C-shaped canal systems have a C-shaped pulpal floor. This makes diagnosis difficult. However, radiographically a tooth with a C-shaped canal system always has a fused root with a longitudinal groove in the middle of the root.[63] Furthermore, there are three types of C-shaped canal systems in mandibular second molars (see Fig. 5-175).[73]

Type I (merging type): Canals merge to one main canal before exiting at the apical foramen

Type II (symmetric type): Separated mesial and distal canals in each root exit as separate canals

Type III (asymmetric type): Separated mesial and distal canals, with the distal canal having a long isthmus across the furcation area

The minimal wall thickness in the middle and apical parts of type III and in the apical part of type II makes these regions danger zones for canal enlargement procedures (see Fig. 5-176).[73]

Another study on mandibular molars found that there is a higher risk of root perforation at the thinner lingual walls of C-shaped canals during shaping and after canal preparation procedures. Both buccal and lingual canal walls were frequently narrower at the mesial locations.[36]

The access cavity for teeth with a C-shaped root canal system varies considerably and depends on the pulp morphology of the specific tooth. Teeth with C-shaped anatomy pose a considerable technical challenge; therefore, use of the DOM during all treatment phases is recommended.

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CHAPTER 6

Cleaning and Shaping the Root Canal System

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CHAPTER OUTLINE

Principles of Cleaning and Shaping
Mechanical Objective
Biologic Objective
Technical Objective
Clinical Issues
Endodontic Instruments
General Characteristics
Manually Operated Instruments
Low-Speed Engine-Driven Instruments
Engine-Driven Instruments for Canal Preparation
Steps of Cleaning and Shaping
Access: Principles
Coronal Preflaring
Patency File
Working Length Determination
Canal Enlargement/Preparation
Final Apical Enlargement
Disinfection of the Root Canal System
Hydrodynamics of Irrigation
Irrigants
Intracanal Medication
Disinfection Devices and Techniques
Criteria to Evaluate Cleaning and Shaping
Well-Shaped Canals
Signs of Mishaps
Sample Protocol for Contemporary Cleaning and Shaping Procedures
Summary

Clinical endodontics encompasses a number of treatments, but they have in common the goal of preventing and treating microbial contamination of pulps and root canal systems. Treatment of traumatic dental injuries and prophylactic treatment of vital pulps are fundamentally different from pulpectomies and root canal instrumentation of teeth with infected pulps (see Chapter 1 for more details on diagnosis).

Endodontic therapy is directed toward one specific set of aims: to cure or prevent periradicular periodontitis.[355,526] The ultimate aim is for patients to retain their natural teeth in function and aesthetics.

To date, many treatment modalities, including the use of nickel-titanium rotary instruments, have not consistently provided a statistically relevant impact on treatment outcomes.[375] This poses a problem in the age of evidence-based therapy, because new therapeutic techniques should deliver improved clinical results over standard procedures. However, the few pertinent clinical trials[99,375,387] and numerous in vitro studies do suggest that certain practices in canal preparation and disinfection are more appropriate than others. This chapter will summarize relevant information.

Orthograde root canal treatment is a predictable and usually highly successful procedure, both in relatively straightforward (Fig. 6-1) and more complex cases (Fig. 6-2). Studies and reviews report favorable outcome rates of up to 95% for the treatment of teeth diagnosed with irreversible pulpitis[39,103,157] and positive outcome rates of up to 85% for infected, necrotic teeth.[102,158,345,375,408]

Microorganisms can breach dental hard-tissue barriers through several avenues, the most common being dental caries (Fig. 6-3). Shaping and cleaning procedures (Box 6-1) as part of root canal treatment are directed against microbial challenges to the root canal system. However, disinfection per se does not guarantee long-term retention of root canal-treated teeth; there is good evidence that this outcome is closely related to placement of an adequate coronal restoration.[21,346,400,425] Moreover, the impact of preservation of radicular structural strength should not be underestimated.[172]

PRINCIPLES OF CLEANING AND SHAPING

Endodontists agree that a major biologic aim of root canal therapy is to address apical periodontitis by disinfection and subsequent sealing of root canal systems. However, considerable disagreement exists over how this goal should be achieved. Although the terms *cleaning* and *shaping* are often used to describe root canal treatment procedures,[417] reversing the order to *shaping* and *cleaning* more correctly reflects the fact that enlarged canals direct and facilitate the cleaning action of irrigants and the removal of infected dentin.

Planktonic microorganisms in the pulp cavity and coronal root canal may be readily killed by irrigants early in a

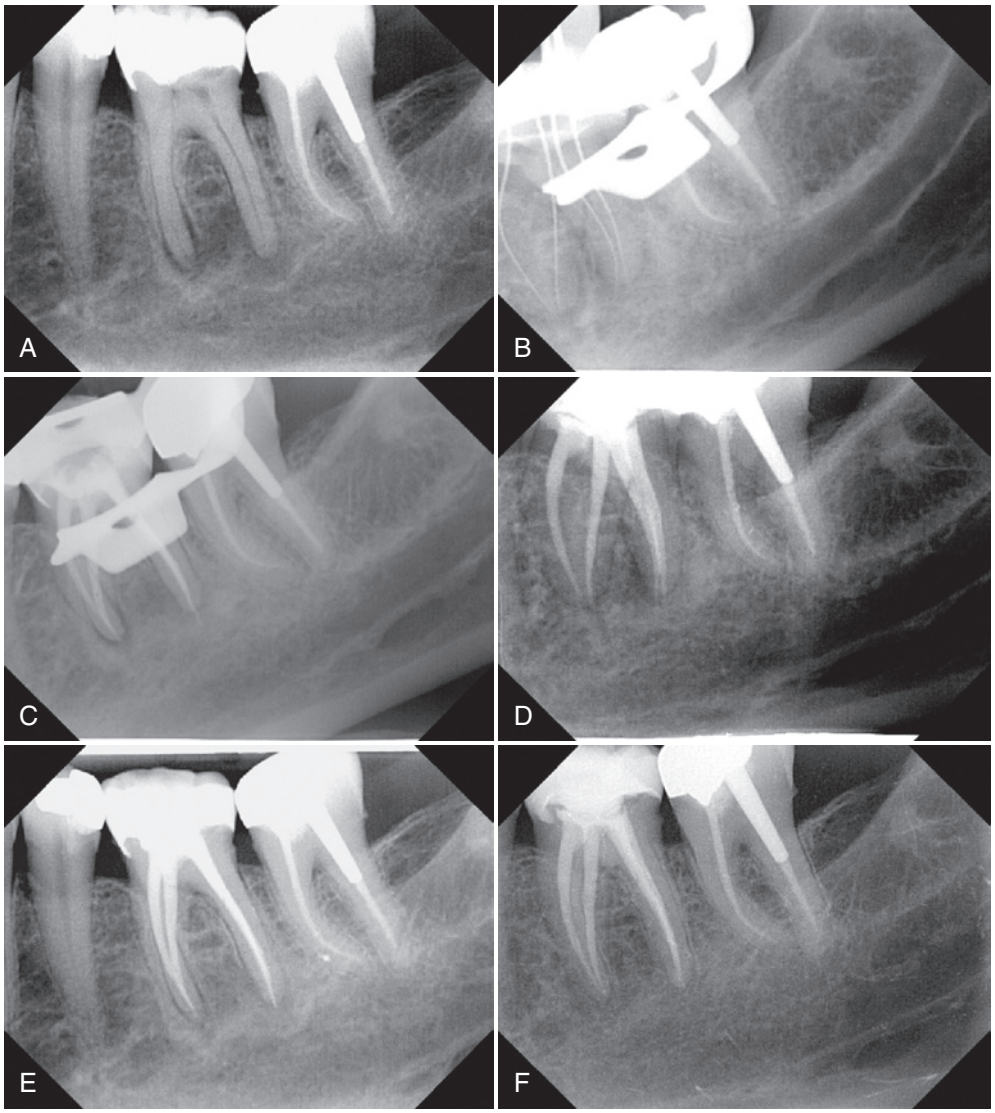


FIG. 6-1 Effect of routine root canal treatment of a mandibular molar. **A**, Pretreatment radiograph of tooth #19 shows radiolucent lesions adjacent to both mesial and distal root apices. **B**, Working length radiograph shows two separate root canals in the mesial root and two merging canals in the distal root. **C**, Posttreatment radiograph after shaping of root canal systems with nickel-titanium rotary files and obturation with thermoplasticized gutta-percha. **D**, Six-month recall radiograph after restoration of tooth #19 with an adhesively inserted full ceramic crown; some periradicular bone fill can be seen. **E**, One-year recall radiograph displays evidence of additional periradicular healing. **F**, Five-year recall radiograph; tooth not only is periapically sound but also clinically asymptomatic and fully functional.

BOX 6-1

Basic Objectives in Cleaning and Shaping

The primary objectives in cleaning and shaping the root canal system are to do the following:

- Remove infected soft and hard tissue
- Give disinfecting irrigants access to the apical canal space
- Create space for the delivery of medicaments and subsequent obturation
- Retain the integrity of radicular structures

procedure, but bacteria in less accessible canal areas or in biofilms still can elicit or maintain apical periodontitis. In everyday practice, these bacteria can be targeted only after mechanical root canal preparation.

Mechanical Objective

An ideal mechanical objective of root canal instrumentation is complete and centered incorporation of the original canals into the prepared shape, meaning that all root canal surfaces are mechanically prepared (*green areas* in Fig. 6-4, *A* and *B*). This goal is unlikely to be met with current techniques.[359,386]

Preparation errors such as deviations, zipping, and perforations should be absent. Although these negative effects of canal

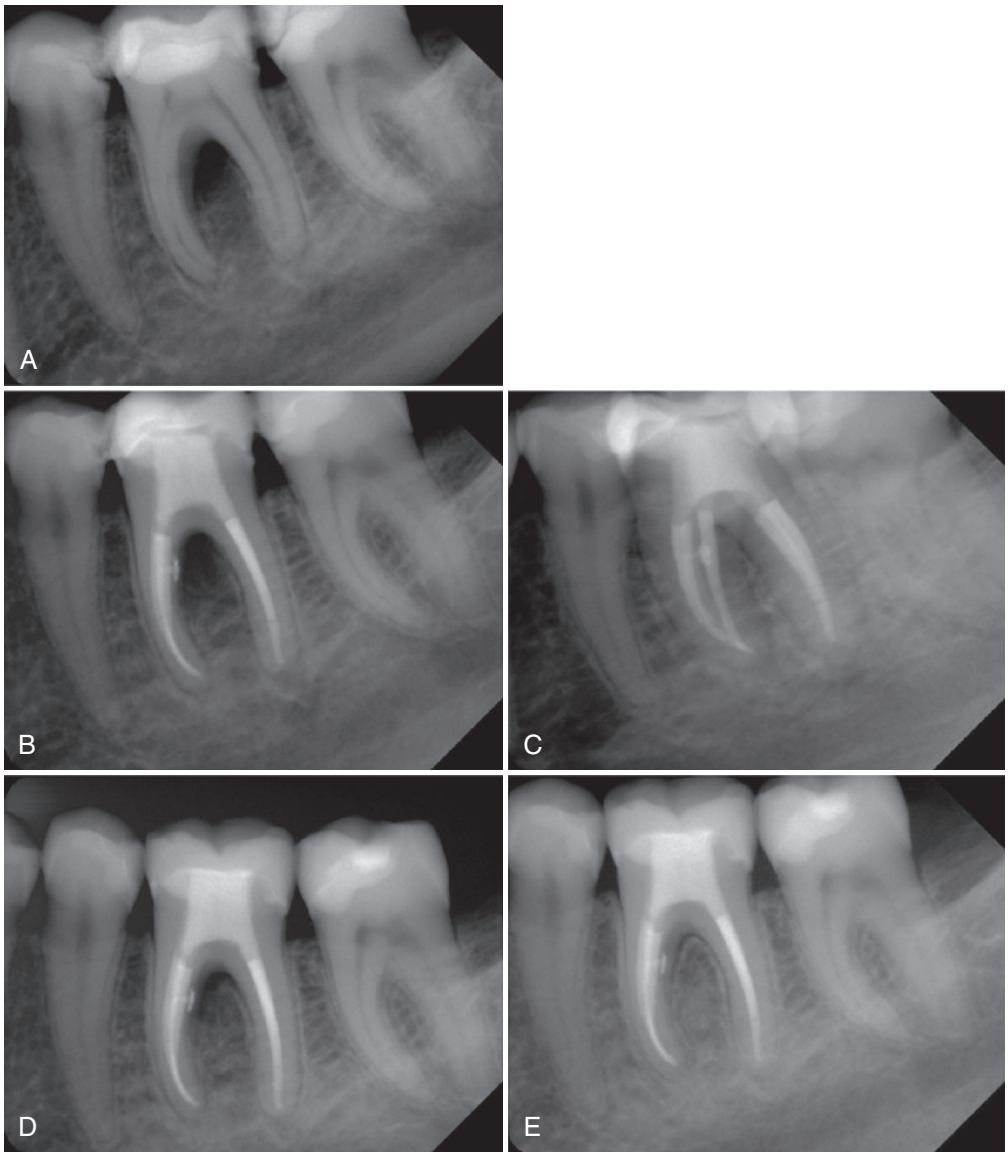


FIG. 6-2 Root canal treatment in a case of apical and interradicular pathosis. **A**, Pretreatment radiograph of tooth #19 shows an interradicular lesion. **B-C**, Posttreatment radiographs after root canal preparation and obturation. Note the lateral canal in the coronal third of the root canal. **D-E**, Two-month recall radiograph suggests rapid healing. (Courtesy Dr. H. Walsch.)

shaping and other procedural mishaps (discussed later) per se may not affect the probability of a favorable outcome,[290] they leave parts of the root canal system inaccessible for disinfection and are undesirable for that reason alone.

Another important mechanical objective is to retain as much cervical and radicular dentin as possible so as not to weaken the root structure, thereby preventing root fractures. Before root canal shaping, dentin wall thickness dimensions of 1 mm and below have been demonstrated in anatomic studies.[127,164] Straightening of canal paths can lead to thinning of curved root walls (Fig. 6-5). Although no definitive minimal radicular wall thickness has been established, 0.3 mm is considered critical by some authors.[287] To avoid overpreparation and outright perforations, adequate access cavity preparation and optimal enlargement of the coronal third of the root canal has to be ascertained (discussed later).

Biologic Objective

Schilder suggested that canals should be prepared to a uniform and continuous taper[445]; however, this guideline was aimed at facilitating obturation rather than targeting antimicrobial efficacy. For optimal disinfection, the preparation shape and antimicrobial efficacy are intimately related through the removal of infected pulp and dentin (Fig. 6-6) and creation of space for delivery of irrigants.

Traditionally, fluids have been dispensed passively into root canals by syringe and needle (Fig. 6-7). When delivered with passive needle irrigation, solutions have been shown to progress only 1 mm farther than the tip of the needle.[189,396,426] Enlarged apical canals and finer needles are likely to allow increasingly deeper needle placement, and this improves debridement and disinfection of canals.[5,12,147,532] However,

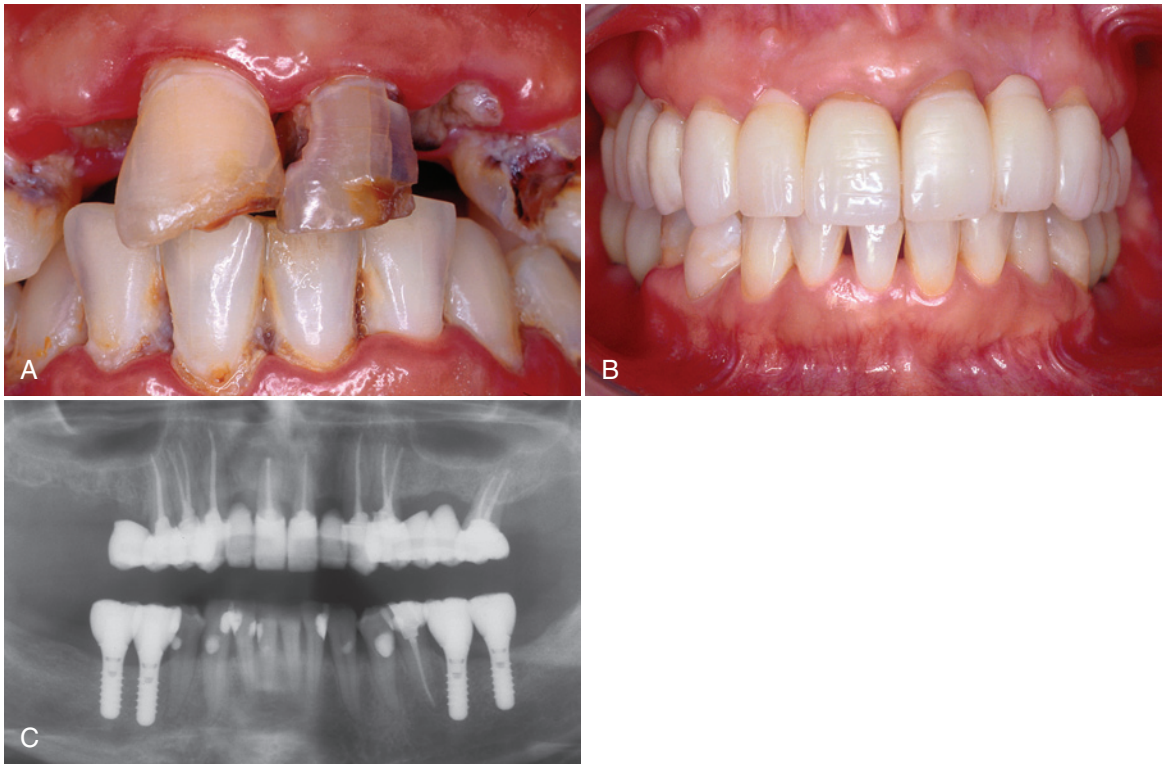


FIG. 6-3 Root canal therapy as part of a comprehensive treatment plan. The patient, who was recovering from intravenous drug addiction, requested restorative dental treatment. Because of extensive decay, several teeth had to be extracted, and nine teeth were treated endodontically. Root canal treatment was aided by nickel-titanium rotary instruments, and obturation was done with lateral compaction of gutta-percha and AH26 as the sealer. Microsurgical retrograde therapy was performed on tooth #8, and the distobuccal root of #14 had to be resected. Metal-free adhesively luted restorations were placed, and missing mandibular teeth were replaced by implants. **A,** Pretreatment intraoral status, showing oral neglect. **B,** Posttreatment intraoral status at 4-year follow-up, showing fully functional, metal-free, tooth-colored reconstructions. **C,** Panoramic radiograph at 4-year recall shows sound periradicular tissues in relation to endodontically treated teeth. (Restorations done by Dr. Till N. Göhring.)

thorough cleaning of the most apical part of any preparation remains difficult,[561] especially in narrow and curved canals.[16,211,404]

Technical Objective

Although a continuous taper that encompasses the original shape and curvature of a given root canal is an accepted goal, final apical preparation size remains a much-disputed entity in root canal therapy, as does final taper of the preparation.[47] Arguments were made for better disinfection with larger sizes (i.e., #50 or greater)[89,415] in combination with smaller tapers of .02 to .05. Others found no difference whether the selected final size was small or large.[105,570] A self-adjusting file was introduced,[327] which does not prepare canals to a specific normed size; its debridement effect is thought to result from a greater radicular wall contact, notably in buccolingually wide canals.[361]

Clinical Issues

A wide spectrum of possible strategies exists for attaining the goal of removing the canal contents and eliminating infection. Lussi and colleagues introduced an approach to removing canal contents and accomplishing disinfection that did not involve the use of a file: the noninstrumentation technique.[303,304] This system consisted of a pump, a hose, and a special valve that was cemented into the access cavity (Fig. 6-8, *A*) to provide oscillation of irrigation solutions (1% to 3% sodium hypochlorite) at a reduced pressure. Although several in vitro studies suggested that canals can be cleaned and subsequently filled using this noninvasive system (see Fig. 6-8, *B* and *C*),[304,305] preliminary clinical results have not been as convincing (see Fig. 6-8, *D*).[25]

At the opposite end of the spectrum is a treatment technique that essentially removes all intraradicular infection through extraction of the tooth in question (see Fig. 6-8, *E* and *F*). Almost invariably, periradicular lesions heal after extraction of the involved tooth.

Clinical endodontic therapy takes place somewhere along this spectrum of treatment strategies. This is reflected in some of the controversies that surround the cleaning and shaping process, such as how large the apical preparation should be and what are the correct diameter, length, and taper.[250] Once the decision has been made to initiate endodontic treatment, the clinician must integrate his or her knowledge of dental anatomy, immunology, and bioengineering science with clinical information.

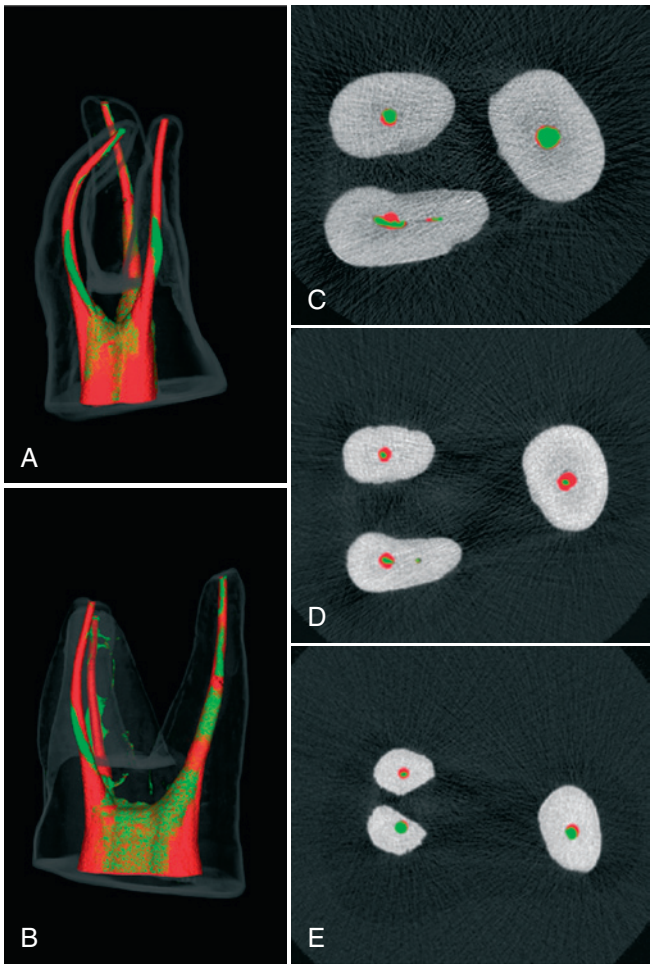


FIG. 6-4 Example of a desired shape, with the original root canal fully incorporated into the prepared outline. **A-B,** Microcomputed tomography reconstructions in clinical and mesiodistal views of a maxillary molar prepared with a NiTi rotary system. The *green area* indicates the pretreatment shape, and the *red area* indicates the posttreatment shape. Areas of *mixed red and green* indicate no change (i.e., no removal of radicular dentin). **C-E,** Cross sections of the coronal, middle, and apical thirds; the pretreatment cross sections *(green)* are encircled by the posttreatment outlines *(red)* in most areas. (*A-B,* From Hübscher W, Barbakow F, Peters OA: Root-canal preparation with FlexMaster: canal shapes analysed by micro-computed tomography, *Int Endod J* 36:740, 2003.)

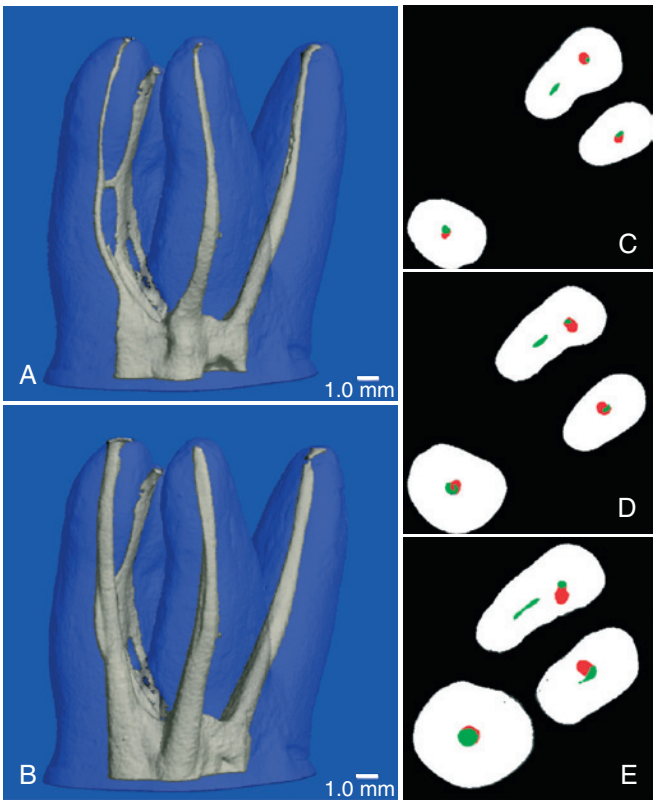


FIG. 6-5 Example of excessive thinning of dental structure during root canal treatment. **A-B,** Microcomputed tomography reconstructions show pretreatment and posttreatment root canal geometry of a maxillary molar. **C-E,** Cross sections of the coronal, middle, and apical thirds with pretreatment canal cross sections. Note the transportation and thinning, in particular, in the main mesiobuccal canal.

Endodontic therapy has been compared to a chain of events, wherein the chain is only as strong as each individual link. For the purposes of this chapter, shaping and cleaning of the root canal system is considered a decisive link, because shaping determines the efficacy of subsequent procedures. It includes mechanical debridement, the creation of space for the delivery of medicaments, and optimized canal geometries for adequate obturation.[373] These tasks are attempted within a complex anatomic framework, as recognized in the early 20th century by Walter Hess[218] (Fig. 6-9; see also Chapter 5 for a complete description of root canal anatomy).

A clinician must choose appropriate strategies, instruments, and devices to overcome challenges and accomplish precise preparation in shape, length, and width. This allows endodontic therapy to address various forms of the disease processes described previously (Fig. 6-10). Recall radiographs taken at appropriate intervals predictably demonstrate longevity and favorable outcomes (see Figs. 6-1, 6-2, and 6-11) if a systematic approach to root canal shaping is adhered to (see Box 6-1).

Endodontic files traditionally have been manufactured according to empiric designs, and most instruments still are conceived based on individual clinicians' philosophies rather than developed through an evidence-based approach. Similar to the development of composite resins in restorative dentistry, the development of new files is a fast and market-driven process. With new instruments becoming available, the clinician may find it difficult to pick the file and technique most suitable for an individual case. Clinicians must always bear in mind that all file systems have benefits and weaknesses. Ultimately, clinical experience, handling properties, usage safety, and case outcomes, rather than marketing or the inventor's name, should decide the fate of a particular design. The following section describes typical instruments used in root canal shaping.

ENDODONTIC INSTRUMENTS

General Characteristics

Design Elements

Root canal preparation instruments such as K-files and nickel-titanium rotary instruments follow certain design principles

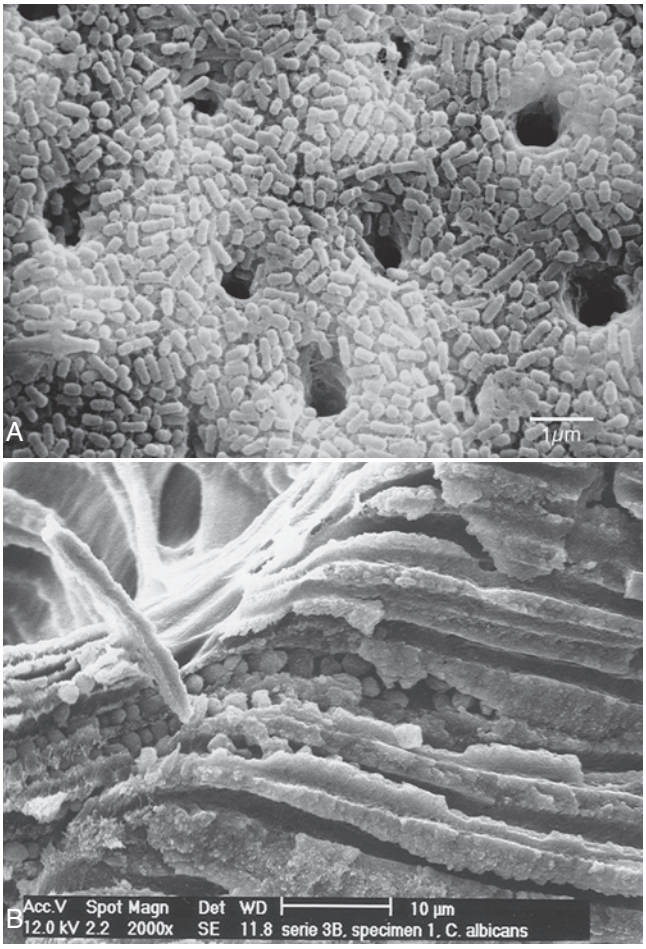


FIG. 6-6 Presence of microorganisms inside the main root canal and dentinal tubules. **A,** Scanning electron micrograph of a root canal surface shows a confluent layer of rod-shaped microbes (×3000). **B,** Scanning electron micrograph of a fractured root with a thick smear layer and fungi in the main root canal and dentinal tubules. (*A,* Courtesy Professor C. Koçkapan. *B,* Courtesy Professor T. Waltimo.)

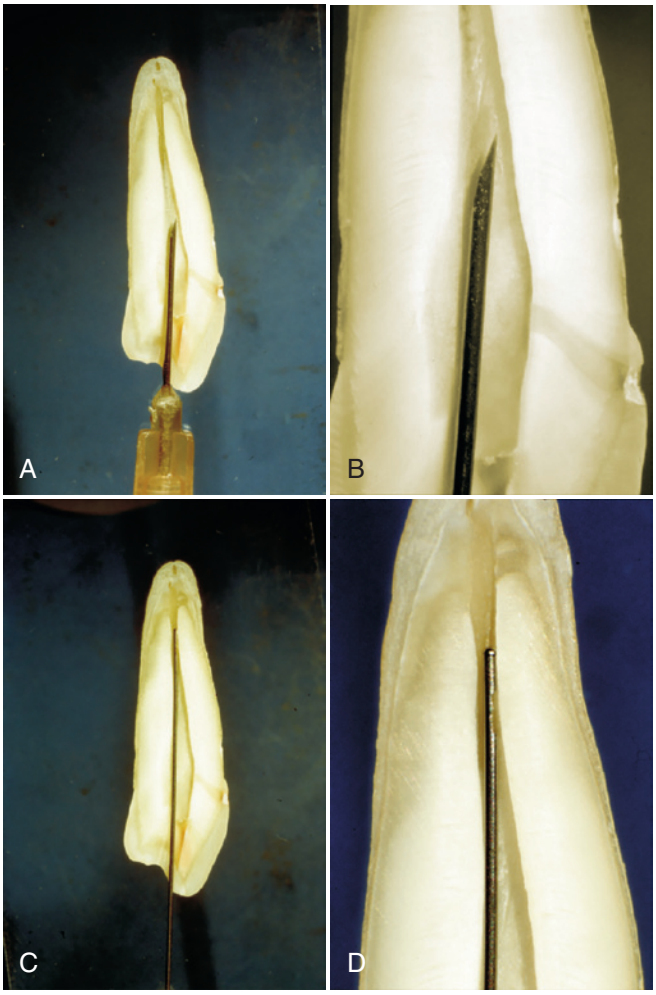


FIG. 6-7 Irrigation needles inserted into prepared root canals. **A-B,** A 27-gauge needle barely reaches the middle third. **C-D,** A 30-gauge, side-venting needle reaches the apical third in adequately enlarged canals.

that relate to drills and reamers used for work in wood and metal, respectively, whereas other instruments such as broaches and Hedström files do not find a direct technologic correlate. Design elements such as the tip, flutes, and cross sections are considered relevant for files and reamers used in rotary motion. These pertinent aspects are briefly described next; for a more detailed review, the reader is referred to the literature.[181,420,427,483]

Tip Design

In root canal preparation, an instrument tip has two main functions: to guide the file through the canal and to aid the file in penetrating deeper into the canal. A clinician unfamiliar with the tip design, in particular of a rotary instrument, may do either of the following: (1) transport the canal (if the tip is capable of enlarging and is used too long in one position in a curved canal) or (2) encounter excessive torsion and break the file (if a noncutting tip is forced into a canal with a smaller diameter than the tip).

The angle and radius of its leading edge and the proximity of the flute to its actual tip end determines the cutting ability of a file tip. Cutting ability and file rigidity determine the propensity to transport the canal. The clinician must keep in mind that as long as a flexible file with a noncutting tip is engaged, 360-degree canal transportation is unlikely to occur.[410]

Studies have indeed shown that tip design affects file control, efficiency, and outcome in the shaping of root canal systems.[330,331] The tip of the original K-file resembled a pyramid; instrument tips have been described as *cutting, noncutting,* and *partially cutting,* although no clear distinction exists among the three types (Fig. 6-12).

Noncutting tips, also called *Batt tips,* are created by grinding and smoothing the apical end of the instrument (see Fig. 6-12, *A*). A tip modification was introduced with the Flex-R file, which was manufactured fully by grinding so that the transitional angles were smoothed laterally between the tip and the instrument's working parts.[411] Similar techniques are required to manufacture NiTi K-files.[512]

For NiTi rotary files, typically rounded noncuttings tips are used (see Fig. 6-12, *B*), which effectively prevent preparation errors that were found with earlier so-called safe cutting tips.[236] One exception to this rule is the type of rotary

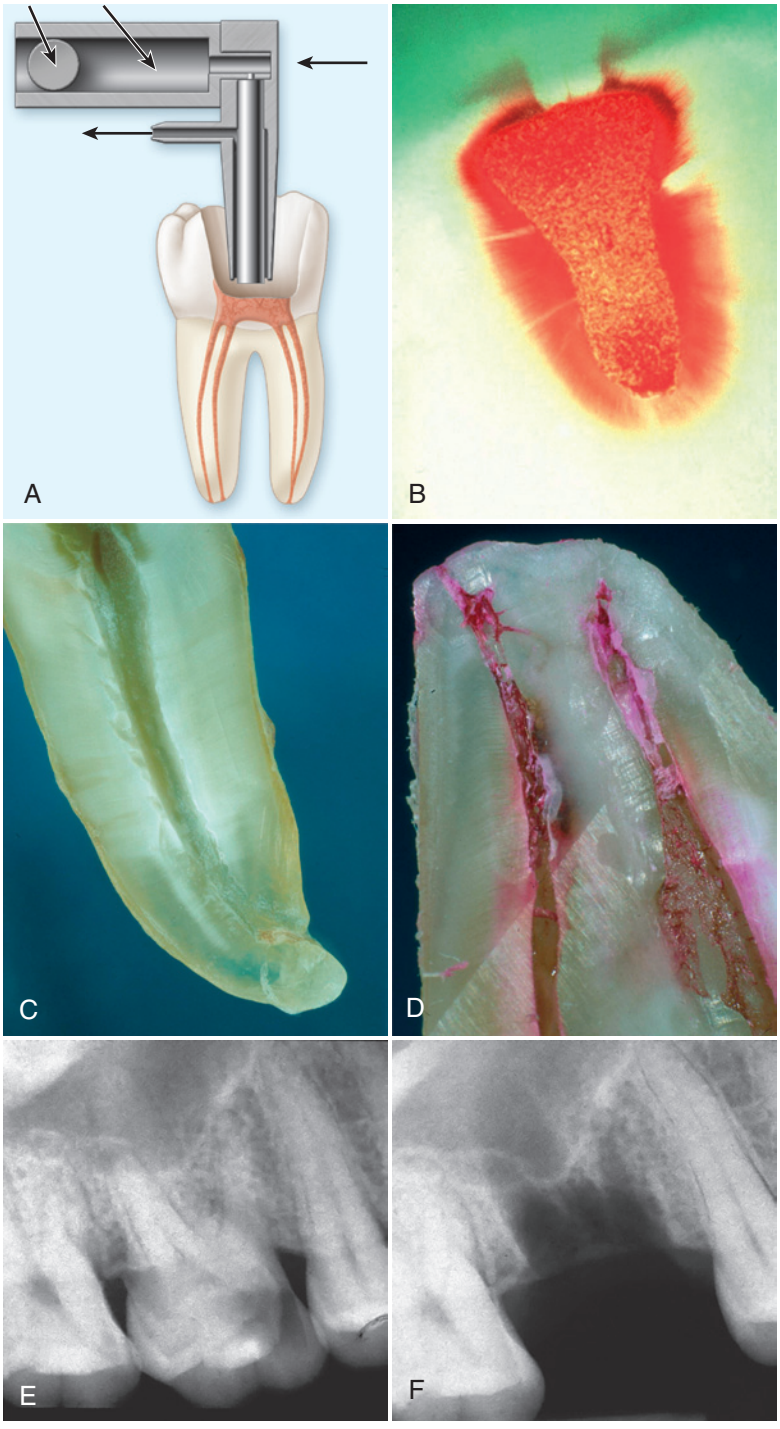


FIG. 6-8 Spectrum of strategies for accomplishing the primary aim of root canal treatment: elimination of infection. **A,** Schematic diagram of minimally invasive therapy using the noninstrumentation technique (NIT). **B,** Example of teeth cleaned in vitro using NIT. Note the clean intracanal surface, which is free of adhering tissue remnants. **C-D,** Examples of teeth cleaned in vivo and later extracted to investigate the clinical effects of NIT. Note the relatively clean, tissue-free canal space in **C** and the significant tissue revealed by rhodamine B staining in **D. E-F,** Course of maximally invasive therapy; apically involved tooth #30 was extracted, effectively removing the source of periradicular inflammation. (*A-B,* Courtesy Professor A. Lussi. *C-D,* Courtesy Professor T. Attin. *E-F,* Courtesy Dr. T. Kaya.)

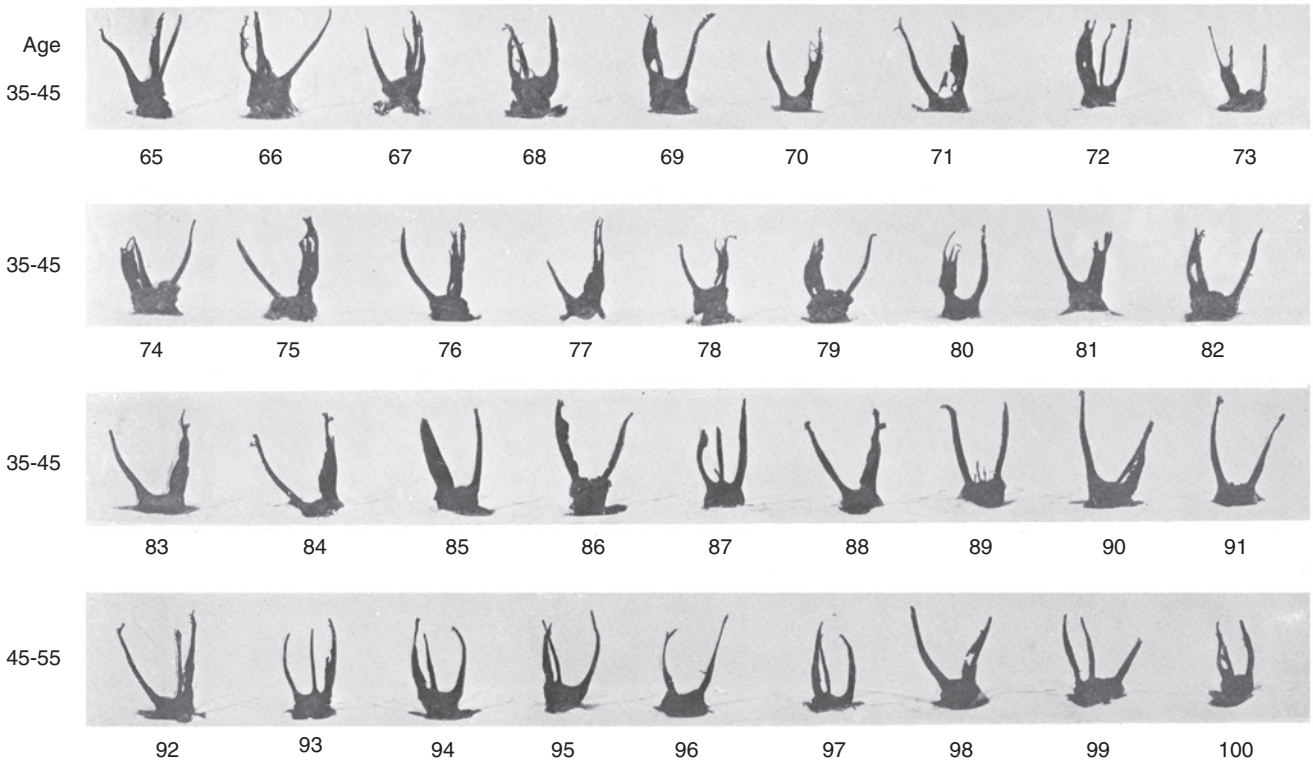


FIG. 6-9 Panel of 36 anatomic preparations of maxillary molars from the classic work by Professor Walter Hess of Zurich. Note the overall variability of root canal systems and the decrease of canal dimensions with age. (From Hess W: *The anatomy of the root canals of teeth of the permanent dentition,* London, 1925, John Bale, Sons & Danielsson.)

specifically designed as a retreatment instrument; cutting tips in that case facilitate removal of the existing root canal filling material and are sufficiently safe.

Longitudinal and Cross-Sectional Design

The *flute* of the file is the groove in the working surface used to collect soft tissue and dentin chips from the wall of the canal. The effectiveness of a flute depends on its depth, width, configuration, and surface finish. The surface with the greatest diameter that follows the groove (where the flute and land intersect) as it rotates forms the *leading (cutting) edge,* or the *blade* of the file. The cutting edge forms and deflects chips along the wall of the canal and severs or snags soft tissue. Its effectiveness depends on its angle of incidence and sharpness.

Some instruments have a feature between trailing and cutting edge that forms a larger contact area with the radicular wall; this surface is called a radial land (Fig. 6-13). Such a land is thought to reduce the tendency of the file to thread into the canal. It also supports the cutting edge and limits the depth of cut; its position relative to the opposing cutting edge and its width determine its effectiveness. On the other hand, landed files are typically less cutting efficient compared to triangular cross sections.[380]

To reduce frictional resistance, some of the surface area of the land that rotates against the canal wall may be reduced to form the *relief*. The angle the cutting edge forms with the long axis of the file is called the *helical angle* (see Fig. 6-13).

If a file is sectioned perpendicular to its long axis, the *rake angle* is the angle formed by the leading edge and the radius of the file through the point of contact with the radicular wall. If the angle formed by the leading edge and the surface to be cut is 90 degrees, the rake angle is said to be neutral. The rake angle may be *negative* or *scraping* (Fig. 6-14, *A*) or *positive* or *cutting* (Fig. 6-14, *C*).

The *cutting angle* is considered a better indication of a file's cutting ability and is determined by measuring the angle formed by the leading edge of the file and a tangent to the radicular wall in the point of contact. The clearance angle corresponds to the cutting angle at the trailing edge of the file and, in case of reciprocating action, becomes the cutting angle. The sum of cutting angle and rake angle is 90 degrees.

The *pitch* of the file is the distance between a point on the leading edge and the corresponding point on the adjacent leading edge (the distance from one "spiral twist" to the next) (see Fig. 6-13). The smaller the pitch or the shorter the distance between corresponding points, the more spirals the file has and the greater the helix angle. Although K-files have a constant pitch typically in the range of 1 mm, many NiTi rotaries have a variable pitch, one that changes along the working surface. When variable pitch is used, usually tighter spirals are located close to the tip of the file and more space between the flutes is located toward the coronal part of the file. A longitudinal section through an instrument reveals the *core* (see Fig. 6-13). The outer diameter of a tapered instrument increases from the file tip toward the handle; depending

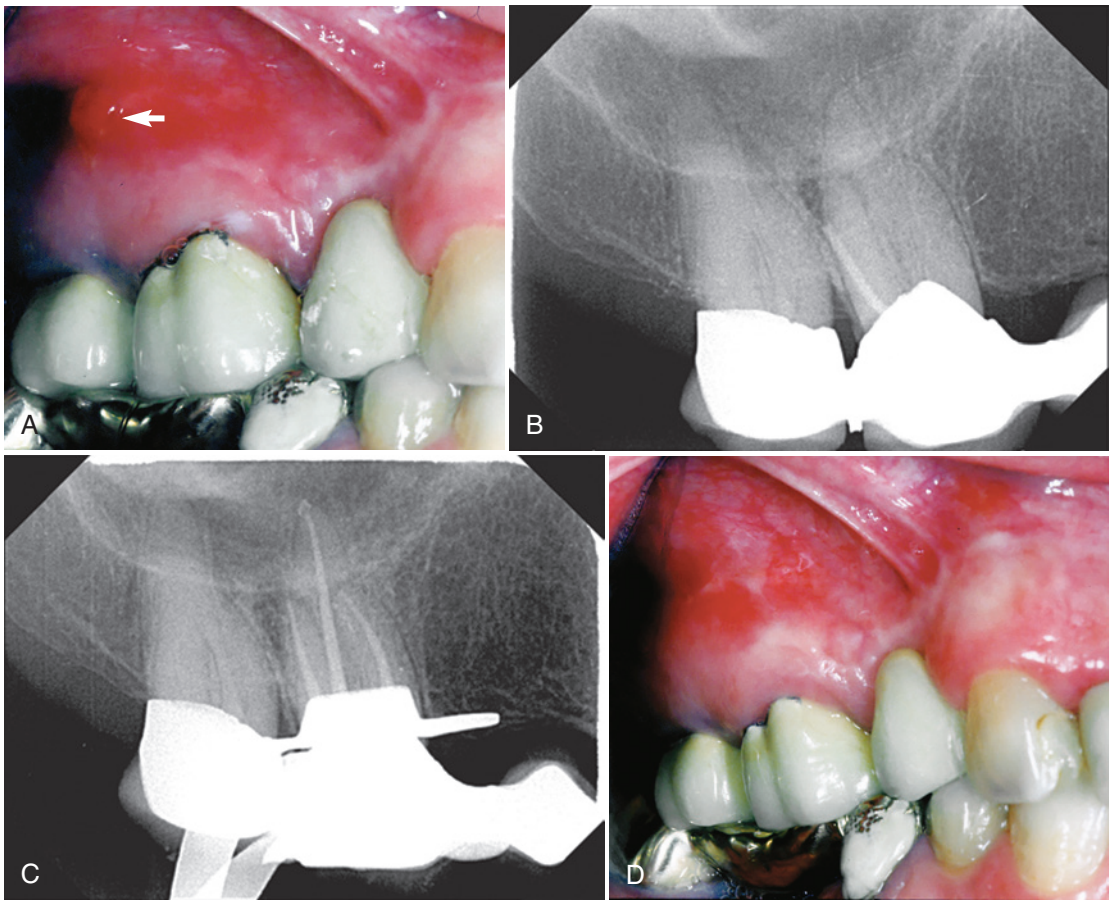


FIG. 6-10 Sinus tract as a sign of a chronic apical abscess and effect of routine root canal treatment. **A,** Intraoral photograph of left maxillary region with draining sinus tract *(arrow)* periapical to tooth #14. **B,** Pretreatment radiograph with gutta-percha point positioned in the sinus tract, pointing toward the distobuccal root of #14. **C,** Finished root canal fillings after 2 weeks of calcium hydroxide dressing. **D,** Intraoral photograph of the same region as in **A,** showing that the sinus tract had closed by the time obturation was performed.

on core dimensions, the flute may become proportionately deeper, resulting in a *core taper* that may be different from the *external taper*.

The cutting angles, helix angles, and external and core tapers may vary along the working surface of the file, and the ratios of these quantities can vary among instruments of the same series. A change in any of these features may influence the file's effectiveness or its propensity for breakage as it progresses into the canal space.

Taper

The taper usually is expressed as the amount the file diameter increases each millimeter along its working surface from the tip toward the file handle. For example, a size #25 file with a .02 taper would have a 0.27-mm diameter 1 mm from the tip, a 0.29-mm diameter 2 mm from the tip, a 0.31 mm diameter 3 mm from the tip, and so forth. Instruments can have constant or variable taper: Some manufacturers express the taper in terms of percentage (e.g., a .02 instrument has been said to have a 2% taper; Fig. 6-15). Current instrument developments include variations in helical angle, pitch, and taper along the cutting portion, which along with variations in alloy and rotational speed (rpm) all affect cutting behavior.[380] The ability to determine cross-sectional diameter at a given point on a file can help the clinician determine the file size in the point of curvature and the relative stress being placed on the instrument. Instruments with greater tapers are designed so that the tip of the instrument functions as a guide and the middle and coronal part of the instrument's working part is the one engaging the canals walls.

ISO Norms

Standardized specifications have been established to improve endodontic instrument quality.[238] For example, the International Standards Organization (ISO) has worked with the Fédération Dentaire Internationale (FDI) to define specifications. These standards are designated with an ISO number. The American Dental Association (ADA) also has been involved in this effort, as has the American National Standards Institute (ANSI); these standards are designated with an ANSI number. However, new instrument designs have resulted in a need for reconsideration of the standards.

Two ISO standards pertain to endodontic instruments. ISO No. 3630-1 deals with K-type files as does ANSI No. 28), Hedström files (ANSI No. 58), and barbed broaches and rasps (ANSI No. 63). ISO No. 3630-3 deals with condensers,

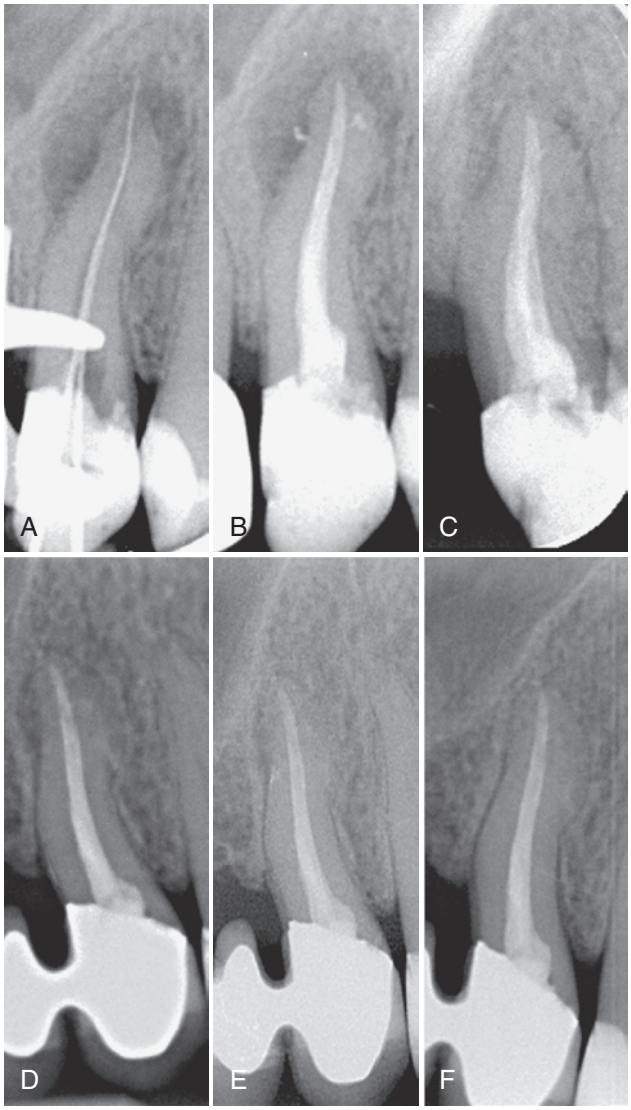


FIG. 6-11 Relationship of radicular anatomy and endodontic disease as shown by filled accessory canals. **A,** Working length radiograph of tooth #13 shows lesions mesially and distally but not apically. **B,** Posttreatment radiograph shows the accessory anatomy. **C,** Six-month recall radiograph before placement of the restoration. **D,** Two-year recall radiograph after resection of the mesiobuccal root of tooth #14 and placement of a fixed partial denture. Excess sealer appears to have been resorbed, forming a distal residual lesion. **E,** Four-year recall radiograph shows almost complete bone fill. **F,** Seven-year recall radiograph; tooth #14 is radiologically sound and clinically within normal limits.

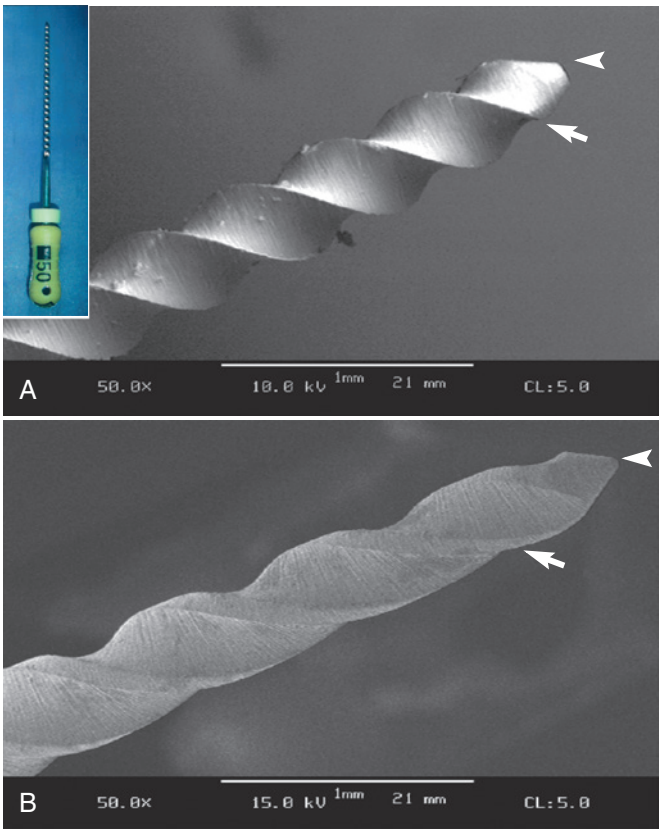


FIG. 6-12 Comparison of the flute geometry and tip configuration of a hand file *(inset)* and a NiTi rotary instrument. **A,** K-file with sharp cutting edges *(arrow)* and Batt tip *(arrowhead). **B**,* GT rotary file with rounded, noncutting tip *(arrowhead),* smooth transition, and guiding radial lands *(arrow).*

pluggers, and spreaders (ANSI No. 71); however, the term *ISO-normed instruments* is often used as a synonym for K-files (see Fig. 6-15).

One important feature of ISO-normed hand instruments is a defined increase in tip diameter of 0.05 or 0.1 mm, depending on the instrument size (Fig. 6-16). ISO-normed K- and Hedström files (Fig. 6-17) are available in different lengths (21, 25, and 31 mm), but all have a 16-mm-long section of cutting flutes (see Figs. 6-12 and 6-15). The cross-sectional diameter at the first rake angle of any file is labeled D0. The point 1 mm coronal to D0 is D1, the point 2 mm coronal to D0 is D2, and so on up to D16. The D16 point is the largest diameter of an ISO-normed instrument. Each file derives its numeric name from the diameter at D0 and is assigned a specific color code (see Fig. 6-15).

Another aspect of ISO files is the standard taper of 0.32 mm over 16 mm of cutting blades, or 0.02 mm increase in diameter per millimeter of flute length (.02 taper). Thus, a size #10 instrument has a diameter of 0.1 mm at D0 and a corresponding diameter of 0.42 mm at D16 [0.1 mm + (16 * 0.02 mm)]. For a size #50 instrument, the diameters are 0.5 mm at D0 and 0.82 mm at D16.

The ISO-normed design is a simplification that has specific disadvantages, and it may explain the clinical observation that enlarging a root canal from size #10 to #15 is more difficult than the step from size #55 to #60. The introduction of K-type files with tip sizes between the ISO-stipulated diameters seemed to solve the problem. However, the use of such files is not universally recommended, perhaps because the approved machining tolerance of ±0.02 mm could negate any intended advantages. Moreover, although ±0.02 mm tolerance is stipulated by the ISO norm, most manufacturers do not adhere to it.[253,449,499,581]

Another suggested modification relates to tips with a constant 29% percentage of diameter increments. This sizing pattern creates smaller instruments that carry less of a workload. However, the intended advantage is offset by larger diameters, because the 29% increase between successive files is

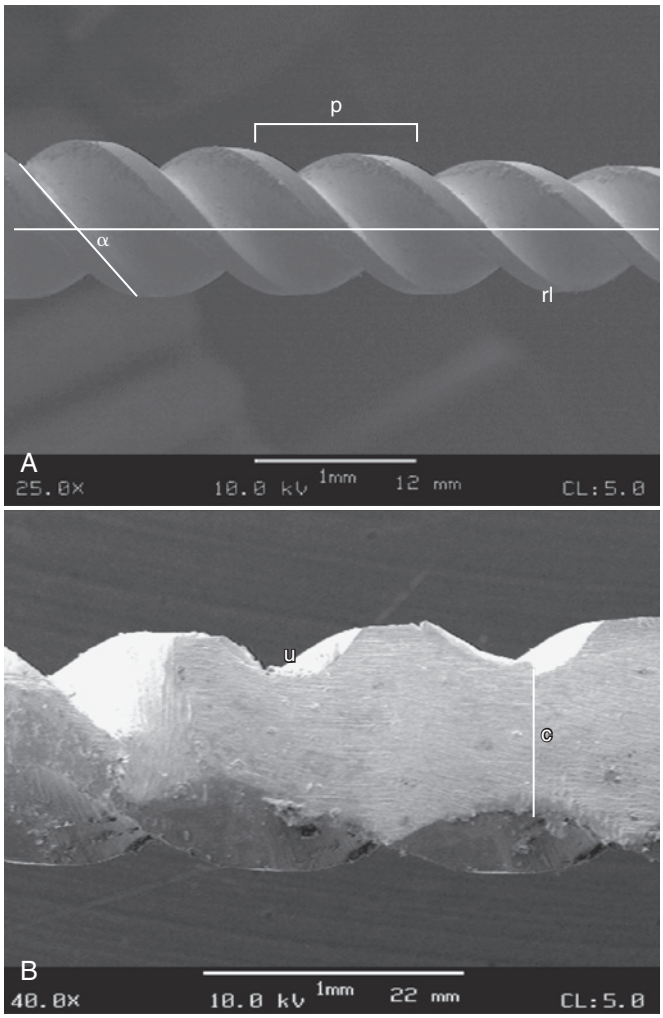


FIG. 6-13 Design characteristics of nickel-titanium rotary instruments. **A**, Lateral view showing the details of the helical angle, pitch *(p)*, and the presence of guiding areas, or radial lands *(rl)* (scanning electron micrograph [SEM], ×25). **B**, Ground working part of the instrument in **A**, showing U-shaped excavations and the dimension of the instrument core *(c)*.

actually greater than the percentage change found in the ISO file series.

Further changes to the numbering system for files with different sizes have been implemented by several manufacturers. One system has introduced "half" sizes in the range of #15 through #60, resulting in instruments in sizes #15, #17.5, #20, #22.5, and so on.

Alloys

There are currently two principally different types of alloys used for endodontic instruments, stainless steel and nickel-titanium. Most manually operated endodontic instruments are fabricated from stainless steel and have considerable resistance to fracture. A clinician who is careful in applying force and adheres to a strict program of discarding instruments after use should have few instrument fractures. Stainless steel files are comparably inexpensive so that adequate cleaning and sterilization for reuse of files in sizes up to #60 may not be cost effective. If this is the case, files in the range up to #60 may be considered disposable instruments.[490]

Several burs and instruments designed for slow-speed handpiece operation such as Gates Glidden drills, Peeso burs, and pilot drills for intraradicular posts are also manufactured from stainless steel. Instruments designed for rotary root canal instrumentation, however, are typically made of nickel-titanium.[454] This alloy offers unique properties, specifically flexibility and corrosion resistance.

Physical and Chemical Properties of Steel and Nickel Titanium Alloys

Basic engineering terms relate to metals and their behavior when used to manufacture endodontic instruments. Stress-strain diagrams describe the response of metal wires under loading depending on their crystal configuration (Fig. 6-18).

During the development of the equiatomic *nitinol* [this acronym is derived from *ni*ckel-*ti*tanium investigated at the *N*aval *O*rdinance *L*aboratory] alloy (55% [by weight] nickel and 45% [by weight] titanium), several effects were noted that relate to its specific crystal arrangement with two stable main phases, *austenite* and *martensite* (Fig. 6-19): a shape memory effect as temperature- and strain-dependent pseudoelasticity, all attributable to specific thermodynamic properties of the new alloy.[77,133,356,517]

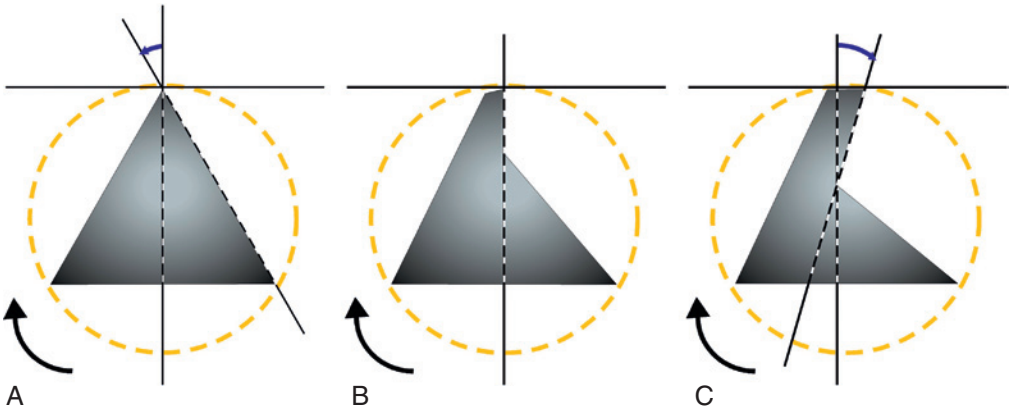


FIG. 6-14 The rake angle of an endodontic file can be negative (**A**), neutral (**B**) or positive (**C**).

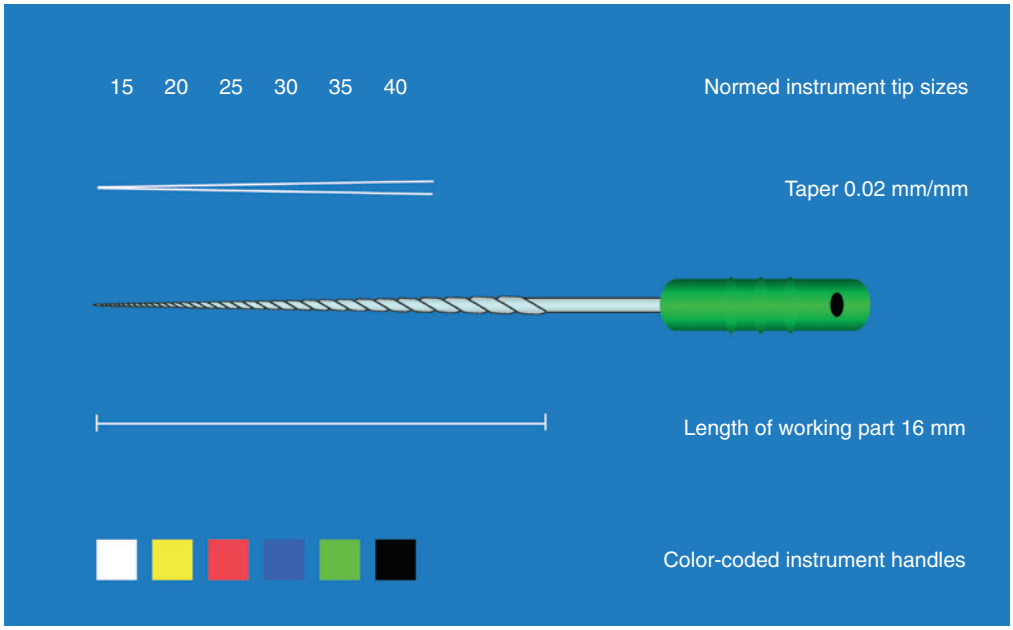


FIG. 6-15 Schematic drawing of an ISO-normed hand instrument size #35. Instrument tip sizing, taper, and handle colors are regulated by the ISO/ANSI/ADA norm.

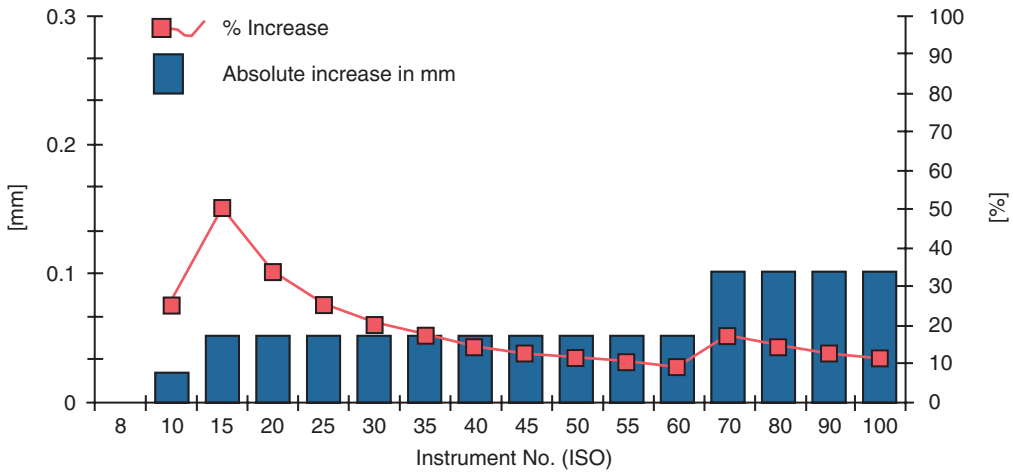


FIG. 6-16 Increase in tip diameter in absolute figures and in relation to the smaller file size. Note the particularly large increase from size #10 to size #15.

Walia and colleagues thought that the pseudoelastic properties of 55-nitinol might prove advantageous in endodontics and initially tested hand instruments.[542] They found that size #15 NiTi instruments were two to three times more flexible than stainless steel instruments; moreover, the instruments showed superior resistance to angular deflection.[542]

Furthermore, hardly any plastic deformation of cutting flutes was recorded when an instrument was bent up to 90 degrees, and forces required to bend endodontic files to 45 degrees were reduced by 50% with NiTi.[454,542] Serene and colleagues speculated that heat, probably during sterilization cycles, could even restore the molecular structure of used NiTi files, resulting in an increased resistance to fracture.[454] Such a behavior is claimed to occur for current martensitic instruments.[377]

These unusual properties are the result of a molecular crystalline phase transformation in specific crystal structures of the austenitic and martensitic phases of the alloy.[517] External stresses transform the austenitic crystalline form of NiTi into a martensitic crystalline structure that can accommodate greater stress without increasing the strain. As a result, a NiTi file has transformational elasticity, also known as *pseudoelasticity,* or the ability to return to its original shape after being deformed (see Fig. 6-19, *B*). This property dictates that typically NiTi instruments are manufactured by milling rather than twisted; twisting incorporates plastic deformation and is used, for example, to produce stainless steel K-files.

Similar to the application of deforming forces, heat can also result in phase transformation (see Fig. 6-19, *A*) from austenite to martensite and vice versa.[209,329] Moreover, thermal conditions during the production of the raw wire can be used to modify its properties, most important of which is its flexibility.[192,461] For austenitic endodontic instruments, a recoverable

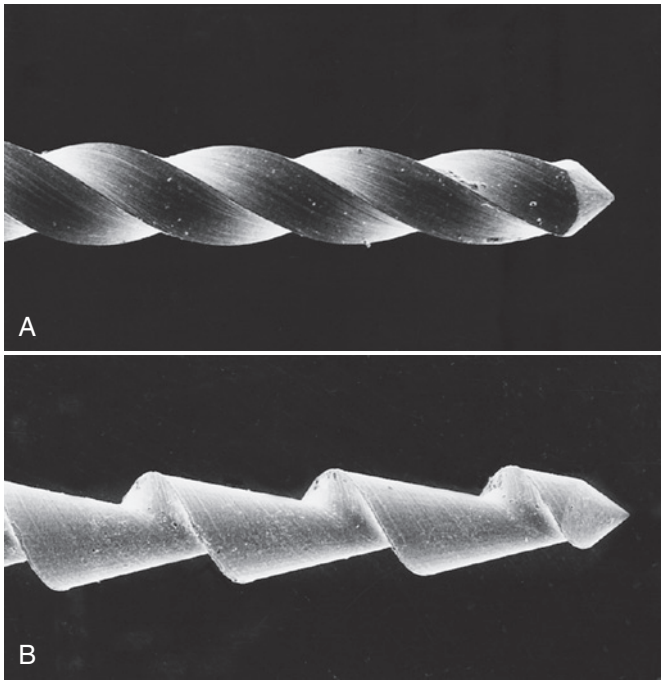


FIG. 6-17 Scanning electron micrographs of endodontic hand files fabricated by twisting (K-file size #40, **A**) and grinding (Hedström file #50, **B**). (Both images courtesy Dentsply Maillefer, Ballaigues, Switzerland).

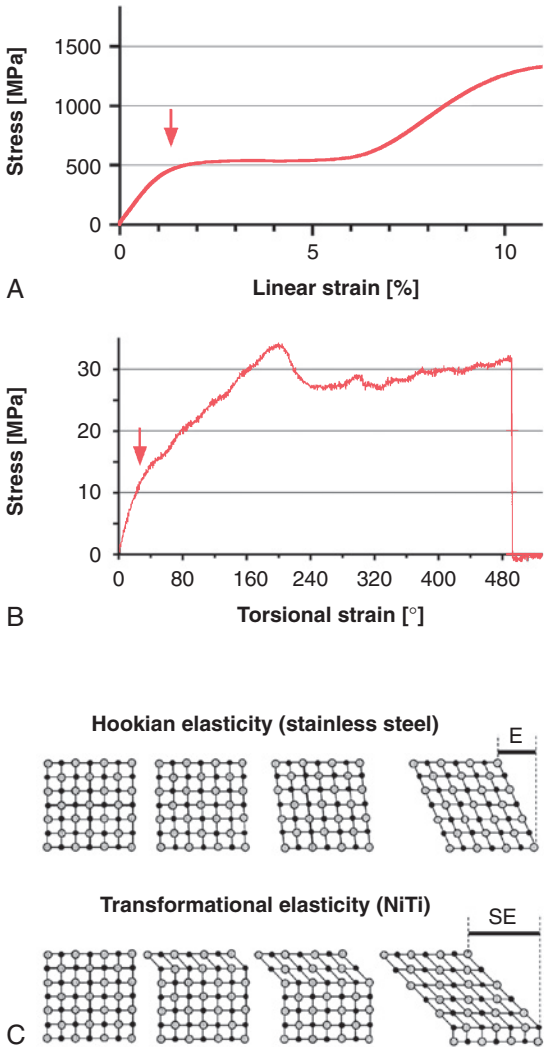


FIG. 6-18 Stress-strain behavior of nickel-titanium alloy. **A,** Schematic diagram of linear extension of a NiTi wire. **B,** Torque to failure test of a size #60, #.04 taper ProFile NiTi instrument. Note the biphasic deformation, indicated by arrows in **A-B. C,** Comparison of stainless steel and nickel-titanium crystal lattices under load. Hookian elasticity accounts for the elastic behavior *(E)* of steel, whereas transformation from martensite to austenite and back occurs during the pseudoelastic *(PE)* behavior of NiTi alloy. (*C,* Modified from Thompson SA: An overview of nickel-titanium alloys used in dentistry, *Int Endod J* 33:297, 2000.)

elastic response of up to 7% is expected (Fig. 6-20). However, more martensitic instruments will have less of an elastic range and are more likely to plastically deform during use.[377,461]

Experiments designed to test fracture resistance demonstrate physical properties of endodontic instruments; following the pertinent ISO norm, 3630-1 graphs such as those shown in Fig. 6-20 are generated and compared among different designs.

Attempts to improve the NiTi alloy continue, and reports indicate that new NiTi alloys may be five times more flexible than currently used alloys.[225,461] NiTi instruments may have imperfections such as milling marks, metal flash, or rollover.[135,454,524,542] Some researchers have speculated that fractures in NiTi instruments originate at such surface imperfections.[11]

Surface irregularities may provide reservoirs of corrosive substances, most notably sodium hypochlorite (NaOCl). Chloride corrosion may lead to micropitting[428] and possibly subsequent fracture in NiTi instruments.[202] Immersion in various disinfecting solutions for extended periods (e.g., overnight soaking) produced corrosion of NiTi instruments and subsequent decreased torsional resistance.[350,486] For ProTaper,[52] RaCe, and ProFile385 instruments, 2-hour immersion damaged the integrity of the alloy. Other authors did not find a corrosion-related effect on K3-files[36] or ProFile instruments.[120]

Regular reprocessing procedures do not seem to significantly affect NiTi rotary instruments.[293,330,503] In one study, only limited material loss occurred when NiTi LightSpeed instruments were immersed in 1% and 5% NaOCl for 30 to 60 minutes.[84] Corrosion of NiTi instruments used in the clinical setting, therefore, might not significantly contribute to fracture except when the instruments are immersed in warmed NaOCl for longer than 60 minutes. In the majority of studies, sterilization procedures do not appear to negatively impact torsional strength[221,464] or fatigue resistance[79,220] of most NiTi instruments: austenitic[538] and martensitic[92] alloy behaved grossly similarly in this aspect.

There is an ongoing discussion over the impact of other aspects of clinical usage on the mechanical properties of NiTi rotaries. Most likely, clinical usage leads to some changes in the alloy, potentially through work hardening.[10,254]

Another strategy to improve surface characteristics is electropolishing; also surface coatings and ion implantation have been tried. Electropolishing is a process that removes surface irregularities such as flash and bur marks. It is thought to improve material properties, specifically fatigue and corrosion resistance; however, the evidence for both these claims is

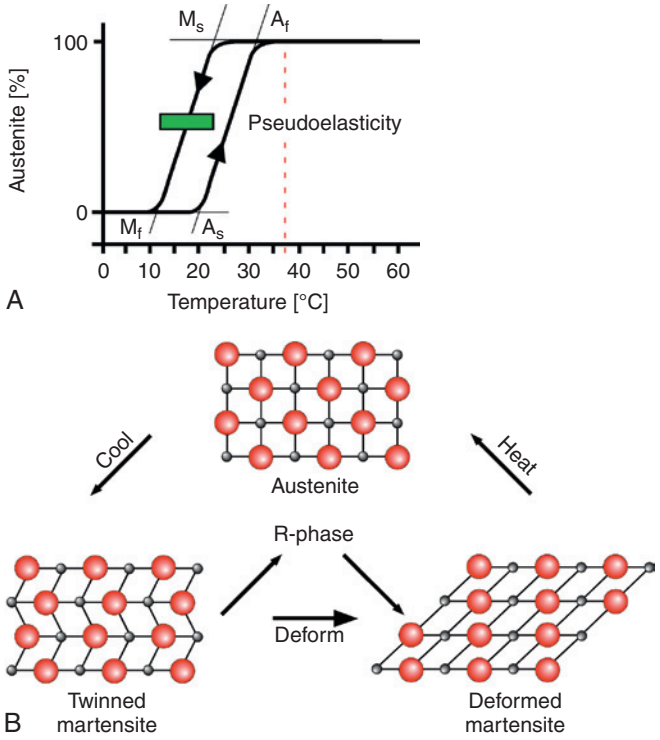


FIG. 6-19 Pseudoelastic behavior of Nickel titanium is based on the two main crystal configurations, martensite and austenite, which depend on temperature (**A**) and applied strain (**B**). Formation of the respective configuration initiates at the start temperatures, M_s and A_s.

mixed. One study found an extension of fatigue life for electropolished instruments,[20] whereas others found no improvement of fatigue resistance of electropolished instruments.[78,100,217] Boessler and colleagues suggested a change in cutting behavior with an increase of torsional load after electropolishing.[59]

Corrosion resistance of electropolished NiTi rotaries is also controversial. One study found superior corrosion resistance for electropolished RaCe instruments,[63] whereas another study demonstrated similar corrosion susceptibility for RaCe and nonelectropolished ProFile instruments.[385] Attempts have been made to improve surface quality by coating it with titanium nitride.[398,438] The latter process also seems to have a beneficial effect on cutting efficiency.[438]

Perhaps more relevant than surface treatment are modification of the base alloy that significantly alter material properties within the atomic ratio.[369] The first commercialized alloy was M-Wire (SportsWire, Langley, OK), which was shown to have higher fatigue resistance with similar torsional strength.[249] More recently, instrument are produced through production process that include annealing and cooling steps to create instruments that, after cold working during the production process, are more martensitic during dental treatment, as defined by their M_s temperature (see Fig. 6-19, *A*). Under clinical conditions these alloys are more flexible[510] and present higher fatigue resistance.[377] Examples are the newly introduced gold and blue alloys types (Dentsply Tulsa Dental Specialties, Tulsa, OK) or so-called controlled memory alloy used in Hyflex instruments (Coltene Endo, Cuyahoga Falls, OH).

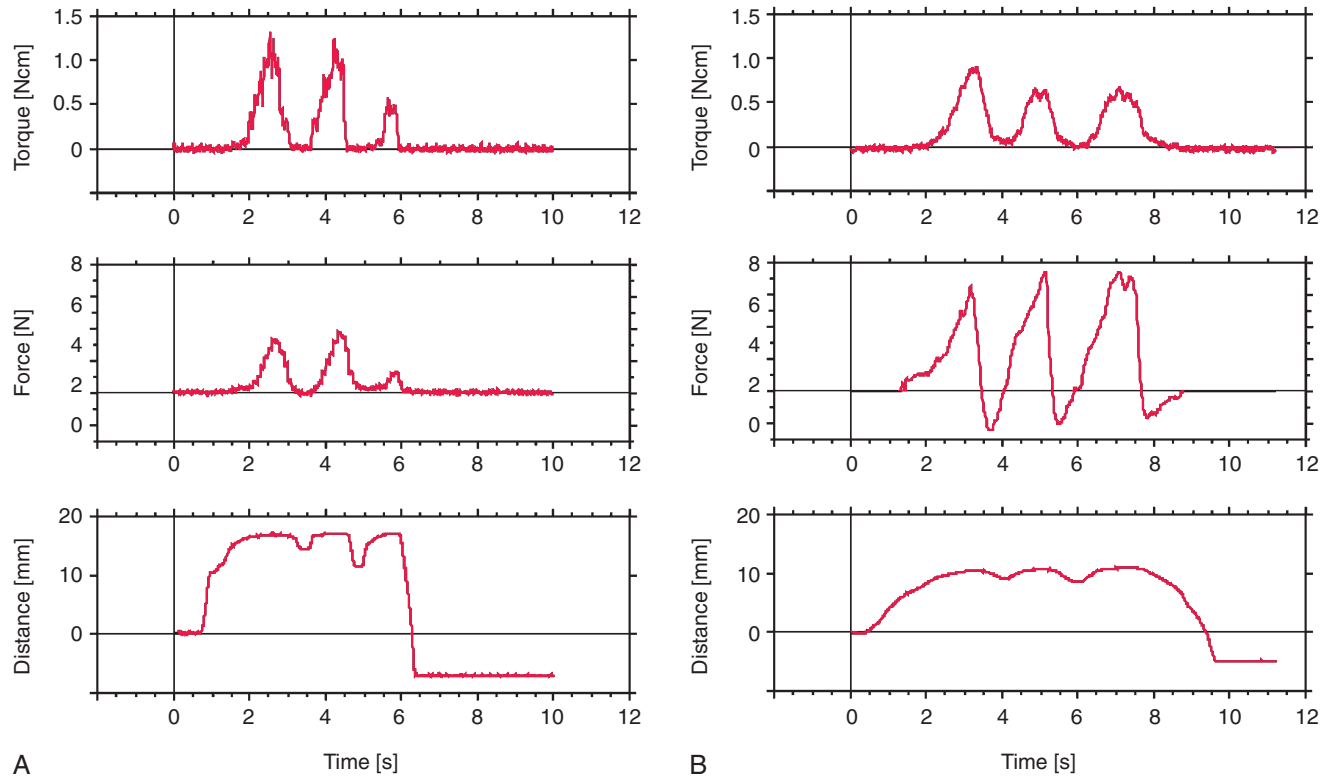


FIG. 6-20 Physical factors (torque, axial force, and insertion depth) that affect root canal instrumentation documented with a torque-testing platform. **A**, ProFile size #45, #.04 taper used in a mildly curved canal of a single-rooted tooth, step-back after apical preparation to size #40. **B**, FlexMaster size #35, #.06 taper used in a curved distobuccal canal of a maxillary first molar, crown-down during the initial phase of canal preparation.

Manually Operated Instruments

Endodontic trays contain many items familiar from general dentistry but certain hand instruments are designed specifically for endodontic procedures. This includes instruments employed for procedures inside the pulp space, for example hand and engine-driven instruments for root canal preparation, and energized instruments for root canal shaping. Special instruments and devices for root canal obturation are selected for filling prepared canal spaces.

K-Type Instruments

Manually operated instruments are generically called *files*. Defined by function, files are instruments that enlarge canals with apico-coronal insertion and withdrawal motions.

Historically, root canal instruments were manufactured from carbon steel. Subsequently, the use of stainless steel greatly improved the quality of instruments. More recently, K-type files manufactured from NiTi were introduced (NiTi-Flex, Dentsply Maillefer, Ballaigues, Switzerland).

Files were first mass produced by the Kerr Manufacturing Co. of Romulus, Michigan, in the early 1900s, hence the name "K-type" file (or K-file) and K-type reamer (K-reamer). K-files and K-reamers were manufactured by the same process—that is, by twisting square or triangular metal blanks along their long axis, producing partly horizontal cutting blades (see Fig. 6-17, *A*). Three or four equilateral, flat surfaces were ground at increasing depths on the sides of a piece of wire, producing a tapered pyramidal shape. The wire then was stabilized on one end, and the distal end was rotated to form the spiral instrument. The number of sides and the number of spirals determine whether the instrument is best suited for filing or reaming. Generally, a three-sided configuration with fewer spirals (e.g., 16 per 16-mm working portion) is used for reaming (i.e., cutting and enlarging canals with rotational motions). A file has more flutes per length unit (e.g., 20) than a reamer, whereas a three- sided or triangular configuration is generally more flexible than a four-sided one.[437]

K-type instruments are useful for penetrating and enlarging root canals. Generally, a reaming motion (i.e., constant file rotation) causes less transportation than a filing motion (translational or "in and out" motion).[171,482] (*Transportation* is defined here as the excessive loss of dentin from the outer wall of a curved canal in the apical segment, as described in more detail later.)

Stainless steel K-files may be precurved by overbending. This procedure subjects the file to substantial strain and should therefore be done carefully. Permanent deformation occurs when the flutes become wound more tightly or opened more widely (Fig. 6-21). When such deformation occurs, the instrument should no longer be used; file fracture is likely to occur during clockwise motion after plastic deformation.[456]

Interestingly, although the force required for failure is the same in both directions of rotation,[271,278] failure occurs in the counterclockwise direction at half the number of rotations required for failure in the clockwise direction. Therefore, K-type instruments should be operated more carefully when pressure is applied in a counterclockwise direction.

Cross-sectional analysis of a K-file reveals why this design allows careful application of clockwise and counterclockwise rotational and translational working strokes: the cross section is symmetrical with negative rake angles, allowing dentin to be adequately cut in both clockwise and counter-clockwise direction.

Reamers are instruments that are similar to K-files in overall design, but they have fewer cutting flutes per mm of the working surface. They are more appropriate for twisting motion and are less frequently used today.[437]

H-Type Instruments

H-type instruments, also known as Hedström files (see Fig. 6-17, *B*), are milled from round, stainless steel blanks. These files are very efficient for translational strokes,[437] because of a positive rake angle and a blade with a cutting rather than a scraping angle. Rotational working movements are strongly discouraged because of the possibility of fracture. Hedström

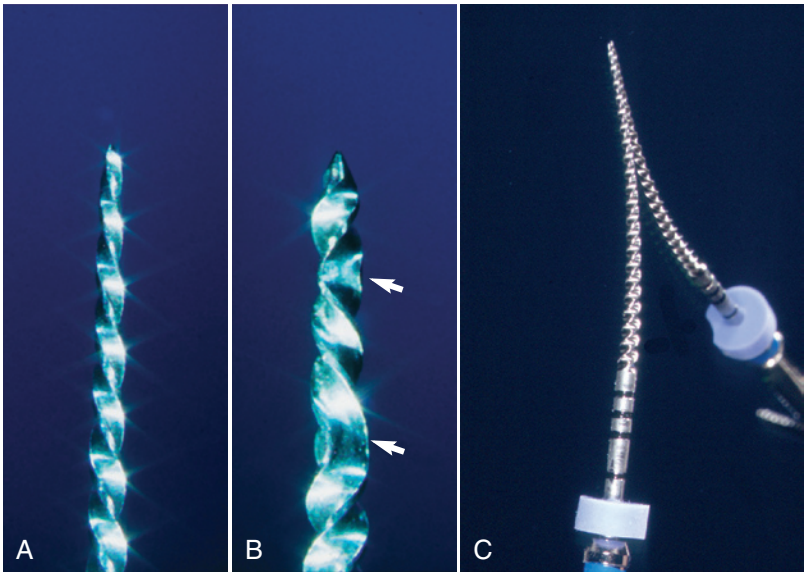


FIG. 6-21 Deformation of endodontic instruments manufactured from nickel-titanium alloy. **A** and **B**, Intact and plastically deformed ProFile instruments (*arrow* indicates areas of permanent deformation). **C**, ProFile instrument placed on a mirror to illustrate elastic behavior.

files up to size #25 can be efficiently used to relocate canal orifices and, with adequate filing strokes, to remove overhangs. Similarly, wide oval canals can be instrumented with Hedström files as well as with rotary instruments. On the other hand, overzealous filing can lead to considerable thinning of the radicular wall and strip perforations (Fig. 6-22). As with stainless steel K-files, Hedström files have been described as disposable instruments.[490]

Bending Hedström files results in points of greater stress concentration than in K-type instruments. These prestressed areas may lead to the propagation of cracks and ultimately fatigue failure.[200] Note that clinically, fatigue fractures may occur without visible signs of deformation.

Hedström files are produced by grinding a single continuous flute into a tapered blank. Computer-assisted machining technology has allowed the development of H-type instruments with complex forms. This process, called *multiaxis grinding,* allows adjustment of the rake angle, helix angle, multiple flutes, and tapers and is also used to fabricate the majority of NiTi instruments. Because the H-file generally has sharper edges than the K-file, it has a tendency to thread into the canal during rotation, particularly if the instrument's blades are nearly parallel. Awareness of threading-in forces is important to avoid instrument failure.

Effectiveness and Wear of Instruments

The ability of an endodontic hand instrument to cut and machine dentin is essential; however, no standards exist for either the cutting or the machining effectiveness of endodontic files, nor have clear requirements been established for resistance to wear. In any study of the effectiveness of an instrument, two factors must be investigated: (1) effectiveness in cutting or breaking loose dentin and (2) effectiveness in machining dentin.

Attempts have been made to evaluate the effectiveness of an instrument when used with a linear movement.[437,442] Collectively, these studies showed that instruments might differ significantly, not only when comparing brands and types but also within one brand and type. For K-files, effectiveness varies 2 to 12 times between files of the same brand. This variation for Hedström files is greater, ranging from 2.5 to more than 50 times.[498] The greater variation among Hedström files is easy to understand because the H-file is the result of more individual grinding during manufacture than the conventional K-file, which is difficult to alter much during the manufacturing process. For example, during the grinding of a Hedström file, the rake angle can be modified to neutral or even slightly positive; this is impossible to achieve with a K-file.

During canal preparation, a file's rake edge shaves off dentin that accumulates in the grooves between the rake edges. The deeper and larger this space, the longer the stroke can be before the instrument is riding on its own debris, making it ineffective.

These design variations and the rake angle of the edges determine the effectiveness of a Hedström file. Of the hybrid files, the K-Flex (SybronEndo, Orange, CA) file has properties similar to those of K-files. The Flex-R file (Integra Miltex, York, PA), which is a ground instrument with a triangular cross section similar to a K-file, more closely resembles a Hedström file in its variations in cutting behavior. It also is more effective at substrate removal than the K-files but cannot measure up to the H-files' ability to machine radicular dentin.[498]

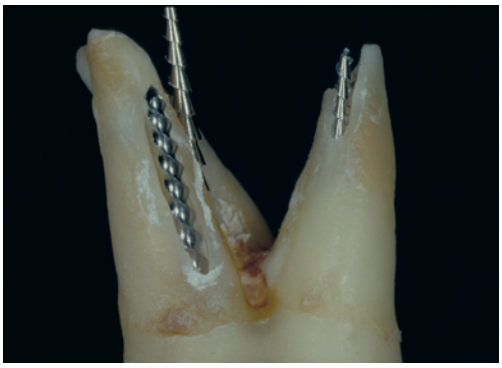


FIG. 6-22 Result of an overenthusiastic attempt at root canal treatment of a maxillary second molar with large stainless steel files. Multiple strip perforations occurred; consequently, the tooth had to be extracted.

Barbed Broaches

Barbed broaches (Fig. 6-23) are produced in a variety of sizes and color codes. They are manufactured by cutting sharp, coronally angulated barbs into metal wire blanks. Broaches are intended to remove vital pulp from root canals, and in cases of mild inflammation, they work well for severing pulp at the constriction level *in toto*. The use of broaches has declined since the advent of NiTi rotary instruments, but broaching occasionally may be useful for expediting emergency procedures (see Chapter 18) and removing materials (e.g., cotton pellets or absorbent points) from root canals.

Low-Speed Engine-Driven Instruments

Burs

Specialized burs are available for endodontic access cavities. These burs are used in both high-speed and slow-speed handpieces and are manufactured from stainless steel. Access cavity preparation and used materials are described in detail in Chapter 5).

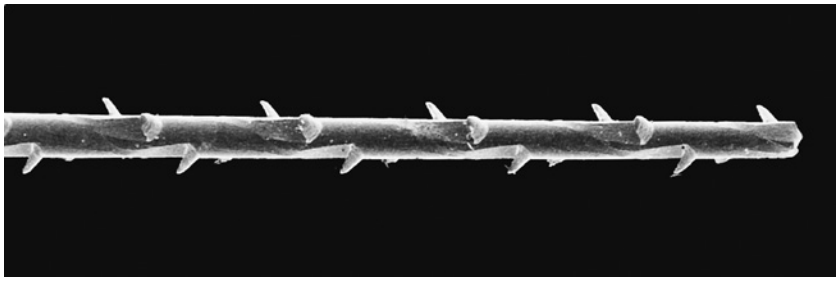


FIG. 6-23 Scanning electron micrograph of a barbed broach. (Moyco Union Broach, York, PA.)

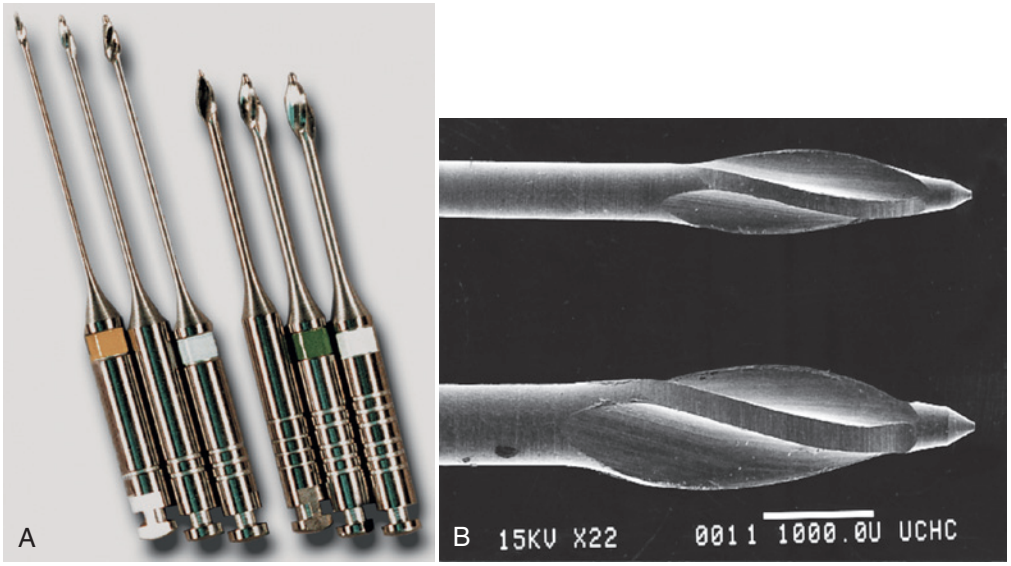


FIG. 6-24 A, Various Gates-Glidden (GG) burs made of stainless steel and scanning electron micrograph (B, working tip). (*A*, From Johnson WT: *Color atlas of endodontics,* St Louis, 2002, Saunders.)

Gates-Glidden Drills

Gates-Glidden (GG) drills (Fig. 6-24) have been used for more than 100 years without noteworthy design changes. Gates Glidden drills are typically used to enlarge coronal canal areas.[119] When misused, GG drills can dramatically reduce radicular wall thickness.[173,242]

GG drills are sized from 1 to 6 (with corresponding diameters of 0.5 to 1.5 mm); the number of rings or color-coding on the shank identifies the specific drill size. GG instruments are available in various lengths and by several manufacturers. Each instrument has a long, thin shaft with parallel walls and a short, oval cutting head with safety tips (Fig. 6-24, *B*); these drills are produced in stainless steel and NiTi varieties. Because of their design and physical properties,[68] GG drills are side-cutting instruments; they can be used to cut dentin as they are withdrawn from the canal (i.e., on the outstroke).[417] Used this way, their cutting action can deliberately be directed away from external root concavities in single-rooted and furcated teeth. GG drills should be used only in the straight portions of a canal.[522]

Two procedural sequences have been proposed: with the step-down technique, the clinician starts with a large drill and progresses to smaller ones; conversely, with the step-back technique, the clinician starts with a small drill and progresses to larger ones. With the step-down approach, the clinician must select a GG instrument with a diameter that allows introduction into the respective orifice and progression for about 1 mm. The subsequent smaller instruments progress deeper into the canal until the coronal third has been preenlarged. This technique efficiently opens root canal orifices and works best when canals exit the access cavity without severe angulations. Opened orifices simplify subsequent cleaning and shaping procedures and help establish a smooth glide path from the access cavity into the root canal system.

With the step-back approach, a small GG instrument is introduced into the canal, and dentin is removed on the outstroke. This process is repeated with the next larger GG instrument, which is again worked shorter than the preceding smaller one. In this way, the coronal third of the root canal is enlarged and dentin overhangs are removed.

When used adequately, GG instruments are inexpensive, safe, and clinically beneficial tools. Gates-Glidden drills may be used safely and to their fullest potential at 750 to 1500 rpm. High revolutions per minute (rpm), excessive pressure, an incorrect angle of insertion, and the use of GG instruments to aggressively drill into canals have resulted in mishaps such as strip perforation. The preferred mode of action for GG drills is against the outer canal wall, away from the canal curvature. Also, cyclic fatigue may cause GG instruments to fracture when used in curved canal areas, and the short cutting heads may fracture with high torsional loads. As with nickel-titanium rotary instruments, GG drills work best when used in electric gear reduction handpieces rather than with air motors.

Peeso Drills (Reamers)

Peeso drills are typically used in root canal preparation either for coronal flaring or during post preparation. These drill are at this point manufactured mostly from stainless steel by milling similar to the Gates Gliddens. Peeso drills are also used in the electric slow-speed handpiece; the rotational speed is the range of 800 to 1200 rpm; the cutting flutes are more parallel and longer compared to GG drills but shorter than the 16 mm prescribed for ISO-normed hand files. Peeso drills are classified as type P and type B-l, as defined by ISO norm 3630-2.[299] The sizing for these drills is also numbers 1 to 6, similar to GGs. Peeso drills are available with cutting and noncutting tips and should be used with caution to avoid excessive preparation and thinning of radicular dentin walls.[4]

Engine-Driven Instruments for Canal Preparation

Instrument Types

Engine-driven instruments for root canal preparation made of stainless steel have been in use for more than half a century—for the first decades, mainly in handpieces that permitted reciprocation (alternating clockwise-counterclockwise motion). The major two problems with this type of instrument were canal transportation (discussed later) and file fracture. This

TABLE 6-1

Grouping of Instruments According to Their Mode of Cutting and Details

Group	Enlargement Potential	Preparation Errors	Fracture Resistance	Clinical Performance
I ProFile[1], ProSystem GT, GTX[1], Quantec[2], Pow-R[3], Guidance[4], K3[2] LightSpeed var.[2]	+, Depending in sizes, often time consuming	++ Low incidence, usually <150 µm canal transportation	+/– Fatigue + Torsional load, depending on system	++ Good, depending on treatment conditions; no difference between instruments shown so far, except for inexperienced clinicians, who perform better with landed instruments
II ProTaper var.[1], RaCe[5], Hero 642[6], FlexMaster[7], Mtwo[7], Sequence[8], Alpha[9]… ProFile Vortex[1] Twisted File[1]	+/–, Good with use of hybrid techniques	+/–, Overall more demanding in clinicians' ability	+ Fatigue +/– Torsional load, depending on taper, handling	
III, EndoEZE AET[12], Liberator[11]… WaveOne[1], Reciproc[7] OneShape[6] SAF	Limited	Varies, Liberator—EndoEZE AET—WaveOne, Reciproc+	Varies + with WaveOne, Reciproc	Varies

Manufacturers:

Grouping of instruments according to their mode of cutting and details about manufacturers. Group I consists of radial-landed instruments with reaming action. Group II instruments have a triangular cross section and cutting action. Group III is made up of instruments with unusual geometry, movement, or sequence.

changed with the advent of NiTi rotaries from about the early 1990s; the much more flexible alloy allowed continuous rotation and reduced both canal preparation errors and instrument fracture compared to earlier engine driven techniques.

Currently, more than 50 types of rotary instrument systems have been described and more continue to be developed. The instruments vary greatly in terms of design, alloy used, and recommended cutting movement (Table 6-1). Various built-in features may help prevent procedural errors, increase efficiency, and improve the quality of canal shaping.

For example, a longer pilot tip may guide the instrument and help to stay centered in the canal long axis. Alternatively, a file can be given an asymmetric cross section to help maintain the central axis of the canal.

Another direction of instrument development is the prevention of instrument fractures (Fig. 6-25). There are several ways to modify an instrument to make it less likely to fracture; for example, increasing the core diameter will increase torsional resistance. Another approach is to use a torque-limiting motor (discussed later). Alternatively, a zero taper or nearly parallel and fluted working portion of the file can be provided for curved canals so that the apical portion of the canal can be enlarged without undue file stress and compression of debris. More recently a reciprocation motion was reapplied to NiTi rotary instruments to prevent threading-in and instrument fractures in general.

Yet another, more recent, direction of instrument development is to improve shaping as it relates to circumferential root canal wall contact. One example for this strategy is a file manufactured from an expandable and flexible hollow NiTi tube, the so-called Self-adjusting File (ReDent-Nova, Ra'anana, Israel, Israel) (Fig. 6-26). More recently, a substantial S-shape was superimposed onto a flexible NiTi rotary that provides a larger envelope of motion while maintaining a limited maximum flute diameter (TRUShape, Dentsply Tulsa Dental Specialties).

Marketed files vary greatly in terms of their specific design characteristics, such as tip sizing, taper, cross section, helix angle, and pitch (see Fig. 6-13). Some of the early systems have been removed from the market or relegated to minor roles; others, such as ProFile (Dentsply Tulsa Dental Specialties), are still successfully used. More recently added instruments vary in cross-sectional and longitudinal design (Fig. 6-27). However, the extent to which clinical outcome changes (if any) will depend on design characteristics is difficult to forecast.[375,381]

Most instruments described in the following section are manufactured by a grinding process, although some are produced by laser etching and yet others by plastic deformation under specific heating and cooling processes. Surface quality has been considered an important detail because cracks that arise from superficial defects may play a role in instrument fracture.[11] Moreover, surface defects such as metal flash and rollover are common in unused NiTi instruments.[135,314]

Many variables and physical properties influence the clinical performance of NiTi rotaries.[272,374,454,512] Clinical practice has generated much of what is known about NiTi instruments, including reasons for instrument fracture[33] and instrument sequences. These instruments have substantially reduced the incidence of relevant canal shaping errors,[387] but they are also thought to fracture somewhat more easily than hand instruments.

Table 6-1 and the following sections describe the instrument groups most widely used for root canal preparation at this point in time. Most basic strategies apply to all NiTi rotary instruments, regardless of the specific design or brand. However, three design groups need to be analyzed separately: group I, instruments designed for passive preparation; group II, rotary instruments designed for active cutting; and group III, unique designs that do not fit in either group I or group II.

Group I: Passive Preparation; Presence of Radial Lands

The first commercially successful rotary instruments were ProFile (Dentsply Tulsa Dental Specialties), LightSpeed (marketed in its current form by SybronEndo), and GT rotaries (Dentsply Tulsa Dental Specialties) and have in common a cross section with so-called radial lands. These are created by three round excavations, also known as the U-shape. The

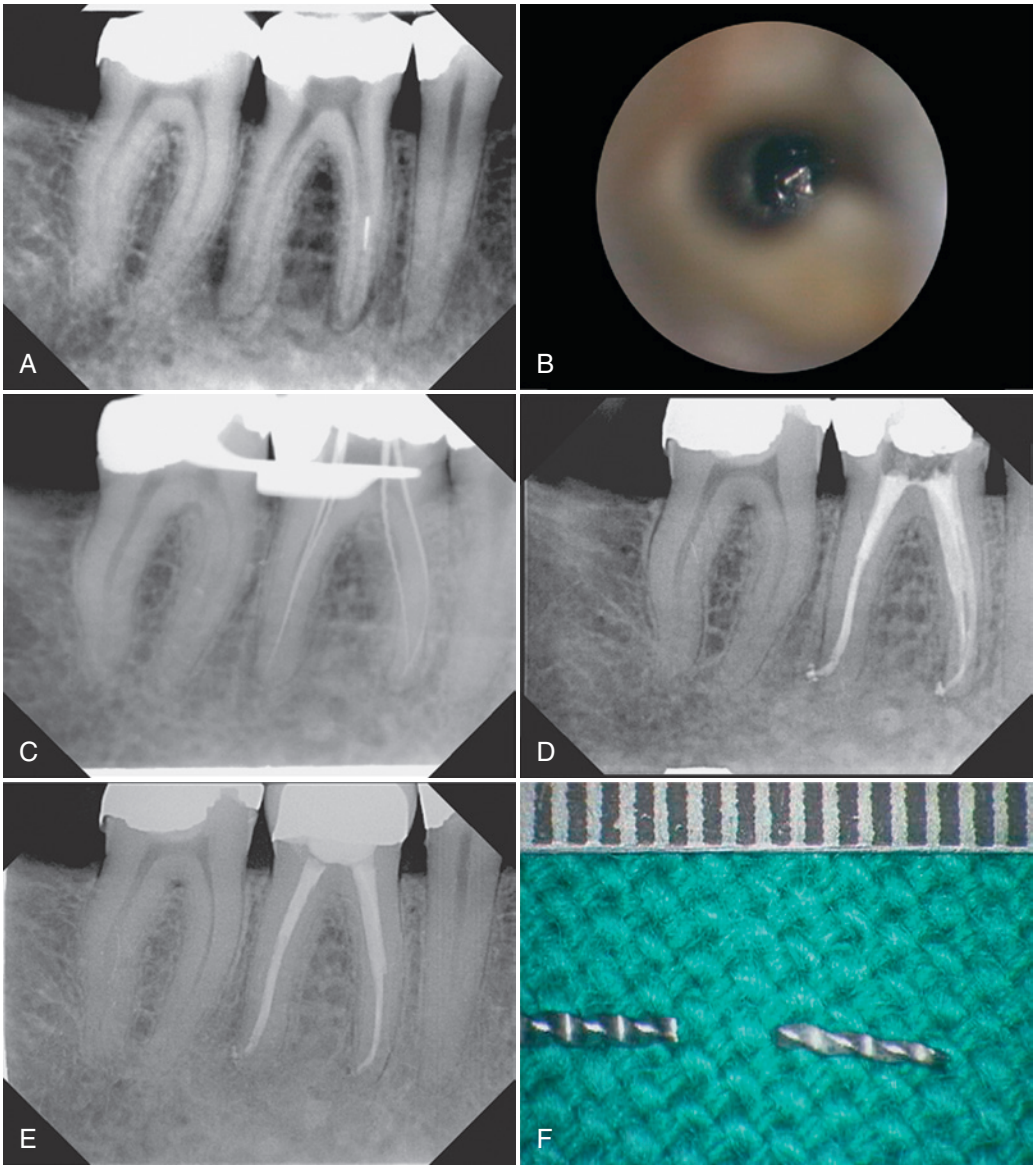


FIG. 6-25 Removal of a fractured NiTi instrument from a mesiolingual canal of a mandibular molar. **A**, Fragment located in the middle third of the root. **B**, Clinical aspect of the fragment after enlargement of the coronal third of the root canal with modified Gates-Glidden drills, visualized with an operating microscope (×25). **C**, Radiograph taken after removal of the fragment; four hand files have been inserted into the canals. **D**, Final radiograph shows slight widening of the coronal third of the mesiolingual canal and fully sealed canal systems. A full crown was placed immediately after obturation. **E**, Recall radiograph 5 years after obturation shows sound periradicular tissues. **F**, Removed fragment and separated file (gradation of ruler is 0.5 mm).

design of the instrument tip and also the lateral file surface (radial land) guide the file as it progresses apically. This makes rotaries listed in group I fairly safe regarding preparation errors. On the other hand, it results in a reaming action rather than cutting of dentin and this makes them inefficient. Moreover, the smear layer produced with radial-landed rotaries is different in consistency and amount compared to the debris and smear created by cutting files.[366,573]

LightSpeed

The LightSpeed file, developed by Drs. Steve Senia and William Wildey in the early 1990s and now also known as LS1, was introduced as an instrument different from all others because of its long, thin, noncutting shaft and short anterior cutting head. The same design principles apply to the currently available LSX instrument (SybronEndo) that is manufactured not by milling but by a stamping process. A full set consists of 25 LightSpeed LS1 instruments in sizes #20 to #100, including half sizes (e.g., 22.5, 27.5); LSX does not have half sizes, and a set includes sizes #20 to #80.

The original LightSpeed is a widely researched NiTi rotary instrument,[50,392,394,459,513,514] and most reports have found that the system has a low incidence of overall and specific preparation errors. One report found similar shaping abilities for LSX and LightSpeed LS1 assessed with a double-exposure technique.[240]

FIG. 6-26 The SAF instrument. The instrument is made as a hollow, thin NiTi lattice cylinder that is compressed when inserted into the root canal and adapts to the canal's cross section. It is attached to a vibrating handpiece. Continuous irrigation is applied through a special hub on the side of its shank. Inset shows the abrasive surface of the instrument. (Courtesy ReDent-Nova, Ra'anana, Israel.)

ProFile

The ProFile system (Dentsply Tulsa Dental Specialties) was introduced by Dr. Ben Johnson in 1994. ProFile instruments have increased tapers compared with conventional hand instruments. The ProFile system was first sold as the "Series 29" hand instruments in .02 taper, but it soon became available in .04 and .06 tapers. The tips of the ProFile Series 29 rotary instruments had a constant proportion of diameter increments (29%). Later, a ProFile series with ISO-sized tips (Dentsply Maillefer) was developed and marketed in Europe.

Cross sections of a ProFile instrument show a U-shape design with radial lands (Fig. 6-28) and a parallel central core. Lateral views show a 20-degree helix angle, a constant pitch, and bullet-shaped noncutting tips (see Fig. 6-12). Together with a slightly negative rake angle, this configuration facilitates a reaming action on dentin rather than cutting. Also, debris is transported coronally and is effectively removed from the root canals.

ProFile instruments shaped canals without major preparation errors in a number of in vitro investigations.[74,75,515,516] A slight improvement in canal shape was noted when .04 and .06 tapered instruments were used in an alternating fashion.[73] Loss

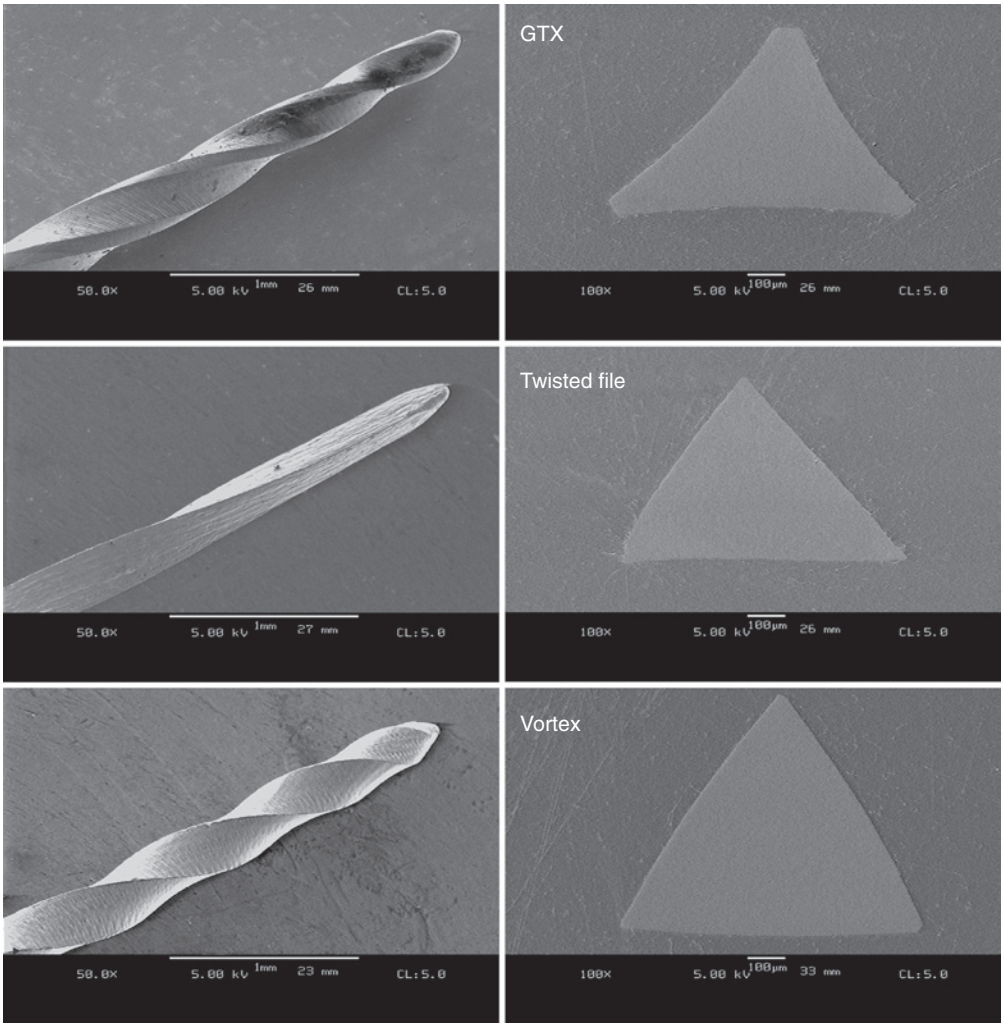
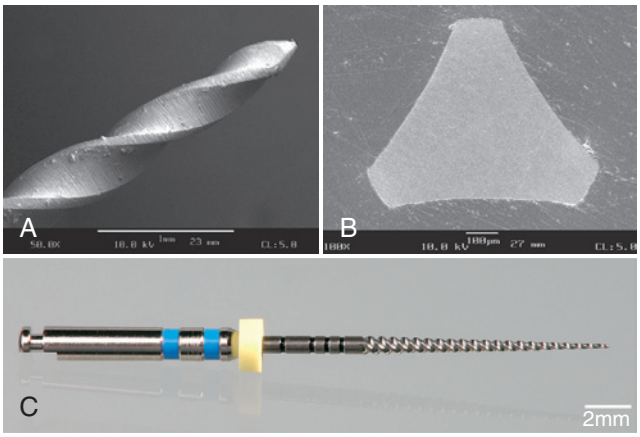


FIG. 6-27 Scanning electron micrographs (SEM) of current nickel titanium rotary instruments, seen laterally (left panel, ×50) and in cross sections (right panel, ×100). Note the radial landed cross section of the GTX instrument



D

No. of instruments/ set	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
Orifice Shapers: 6	20-80	10; from 60: 20	150 to 350, low apical force, torque to fracture and working torque dependent on instrument size	19 mm
ProFile .06: 6	15-40	5		21 mm, 25 mm, some 31 mm
ProFile .04: 9	15-90	5; from #45: 15; from #60: 30		
ProFile .02: 6	15-45	5		
Profile Series 29	13-100	Varies, 29%		21 mm, 25 mm

FIG. 6-28 Design features of a ProFile instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.

of working length did not exceed 0.5 mm[73-75,515,516] and was not affected by the use of .06 tapered instruments.[73] Comparative assessments in vitro suggested that ProFile prepared mesial canals in mandibular molars with less transportation than K3 and RaCe.[9]

A more recent addition to the ProFile family of instruments was Vortex (Dentsply Tulsa Dental Specialties). The major change lies in the nonlanded cross section, whereas tip sizes and tapers are similar to existing ProFiles, hence these files are placed in group 2 (discussed later).

GT and GTX Files

The Greater Taper, or GT file, was introduced by Dr. Steve Buchanan in 1994. This instrument incorporates a radial landed cross-sectional design and was marketed as ProFile GT (Dentsply Tulsa Dental Specialties). The system was first produced as a set of four hand-operated files and later as engine-driven files. The instruments came in four tapers (.06, .08, .10, and .12), and the maximum diameter of the working part was 1 mm. The design limited the maximum shank diameter and decreased the length of the cutting flutes with increasing taper. The instruments had a variable pitch and a growing number of flutes in progression to the tip; the apical instrument diameter was 0.2 mm. Instrument tips were noncutting and rounded; these design principles are mostly still present in the current incarnation, the GTX instrument. The main differences are the M-Wire alloy type used and minor design and handling changes.

Studies on GT files found that the prepared shape stayed centered and was achieved with few procedural errors.[173,208] A shaping assessment using micro-computed tomography [μCT; see Fig. 6-4 as an example)] showed that GT files machined statistically similar canal wall areas compared with ProFile and LightSpeed preparations.[386] These walls were homogeneously planed and smooth.[366,573] The more recently introduced GTX variant is manufactured from M-Wire and does not appear to have significantly improved physical properties[254] or shaping capacity.[241] GT instruments were available for small (tip size #20), medium (tip size #30), and large canal diameters (tip size #40).

K3

In a sequence of design developments by their inventor, Dr. McSpadden, the Quantec 2000 files were followed by the Quantec SC, the Quantec LX, and the current K3 system (all by SybronEndo). The overall design of the K3 is similar to that of the ProFile in that it includes instruments with .02, .04, and .06 tapers. The most obvious difference between the Quantec and K3 models is the K3's unique cross-sectional design: a slightly positive rake angle for greater cutting efficiency, wide radial lands, and a peripheral blade relief for reduced friction. Unlike the Quantec, a two-flute file, the K3 features a third radial land to help prevent threading-in.

In the lateral aspect, the K3 has a variable pitch and variable core diameter, which provide apical strength. This intricate

design is relatively difficult to manufacture, resulting in some metal flash.

Like most other current instruments, the K3 features a round safety tip, but the file is about 4 mm shorter than other files (although it has the same length of cutting flutes) because of the so-called Axxess handle. The instruments are coded by ring color and number.

Tested in vitro, K3's shaping ability seems to be similar to that of the ProTaper[51] and superior to that achieved with hand instruments.[440] More recently, when curved canals in lower molars were shaped to a size #30 .06,[14] K3 files had less canal transportation in a modified Bramante model than RaCe but more than ProFile.

Summary

Radial landed rotary instruments are considered very safe, even when accidentally taken beyond the confines of the root canal. Fracture resistance to torsional loading and cyclic varies depending on specific instrument design. The limited cutting efficacy of these files was perceived as a downside and is a reason that the market share has diminished.[55] However, their excellent track record in clinical applications and research continues to support the use of rotaries listed in group I.

Group II: Active Cutting; Triangular Cross Section

Rotaries in group II all have a more active cutting flute design in common. Radial lands are absent (see Fig. 6-13), and this fact results in a higher cutting efficacy. This translates to a higher potential for preparation errors, in particular when the instrument is taken through the apical foramen, thus eliminating the guide derived from the noncutting tip.

ProTaper Universal

The ProTaper system originally comprised six instruments: three shaping files and three finishing files. This set is now complemented by two larger finishing files and a separate set of three rotaries tailored to retreatment procedures. The instruments were designed by Drs. Cliff Ruddle, John West, and Pierre Machtou. In cross sections, ProTaper shows a convex triangle with sharp cutting edges and no radial lands. The cross section of finishing files F3, F4, and F5 is slightly relieved for increased flexibility. The three shaping files have tapers that increase coronally, and the reverse pattern is seen in the five finishing files.

Shaping files nos. 1 and 2 have tip diameters of 0.185 mm and 0.2 mm, respectively, 14-mm-long cutting blades, and partially active tips. The finishing files (F1-F5) have tip diameters of 0.2, 0.25, 0.3, 0.4, and 0.5 mm, respectively, between D0 and D3, and the apical tapers are .07, .08, .09, .05, and .04, respectively. The finishing files have rounded noncutting tips.

Two aspects of handling have been emphasized for ProTaper. The first is the preparation of a glide path, either manually[368] or with special rotary instruments.[53] An enlargement to a size approaching the subsequent rotaries' tips, at least larger than the file's core diameter, prevents breakage and allows assessment of the canal size.[368] This means that the glide path should correspond to a size #15 or #20. The second specific recommendation is the use of a more laterally directed "brushing" working stroke. Such a stroke allows the clinician to coronally direct larger files away from danger zones and counteract any "threading-in" effect.[58] Both usage elements should be considered good practice for other instruments, particularly for more actively cutting ones.[391]

In a study using plastic blocks, ProTaper created acceptable shapes more quickly than GT rotary, ProFile, and Quantec instruments,[573] but it also created somewhat more aberrations. This was recently corroborated comparing preparations of mesial root canals in mandibular molars ex vivo with ProTaper Universal to Alpha (Gebr. Brasseler GmbH & Co. KG-Komet, Lemgo, Germany).[536] In a comparison of ProTaper and K3 instruments (SybronEndo), Bergmans and colleagues found few differences, with the exception of some transportation by the ProTaper into the furcation region.[51] A study using μCT showed that the ProTaper created consistent shapes in constricted canals, without obvious preparation errors, although wide canals may be insufficiently prepared with this system.[384] It has been recommended that ProTaper be combined with less tapered, more flexible rotaries to reduce apical transportation.[244]

A newer version of this system, called ProTaper Next was introduced in 2013. Current research suggests that mechanical properties of these instruments, manufactured from M-Wire, are better than ProTaper Universal.[23,138,370]

No data are currently available on shaping outcomes or clinical results.

HERO 642, HERO Shaper

Several systems in group II (see Table 6-1) were designed with positive rake angles, which provide greater cutting efficiency. HERO instruments (MicroMega, Besançon, France) are an example. The original version was known as *HERO 642* (the acronym *HERO* stands for *h*igh *e*lasticity in *ro*tation), and the name has now changed into HERO Shaper, with little apparent differences in the instrument design.

Cross sections of HERO instruments show geometries similar to those of an H-file without radial lands. Tapers of .02, .04, and .06 are available in sizes ranging from #20 to #45. The instruments are relatively flexible but maintain an even distribution of force into the cutting areas.[528,529] HERO instruments have a progressive flute pitch and a noncutting passive tip, similar to other NiTi rotary systems. The instruments are coded by handle color.

Research with HERO files indicates a shaping potential similar to that of the FlexMaster[230] (VDW, Munich, Germany) and ProFile,[164] although in one study HERO files induced more changes in cross-sectional anatomy.[179] HERO instruments also caused some aberrations when used in simulated canals with acute curves,[517] but they were safer than Quantec SC instruments (SybronEndo).[236] More recently, HERO Shapers had a better centering ability compared to RaCe instruments in resin blocks.[28] Using a modified Bramante technique in vitro, earlier HERO 642 and current HERO Shaper rotaries showed no differences in canal cross sections before and after shaping.[28]

FlexMaster

The FlexMaster file system is currently unavailable in the United States but popular in Europe. It features .02, .04, and .06 tapers. The cross sections have a triangular shape, with sharp cutting edges and no radial lands. This makes for a relatively solid instrument core and active cutting ability. The overall manufacturing quality seems high, with minimal metal flash and rollover.

FlexMaster files have rounded, passive tips; the tip diameters range from 0.15 to 0.7 mm for .02 instruments and 0.15

to 0.4 mm for.04 and .06-tapered files. In addition to the standard set, the Intro file is available, which has a .11 taper and a 9-mm cutting part. The instruments are marked with milled rings on the instrument shaft, and the manufacturer provides a system box that indicates sequences for narrow, medium-size, and wide canals.

Several studies indicate that the FlexMaster allows centered preparations in both constricted and wider canals[227] and that it performed on par with other systems.[230,550] Clinical studies confirmed that the FlexMaster showed superior shaping characteristics compared with K-files.[441] Also, novice dental students were able to shape plastic blocks successfully with the FlexMaster after a short training period.[484,485] Tested in a well-described model of simulated canals, FlexMaster instruments led to few aberrations but took longer than preparations with RaCe files.[325] Moreover, FlexMaster appeared to be less effective than RaCe in removing dye from the walls of simulated canals prepared to size #30 but were more effective than ProFile.[446]

RaCe, BioRaCe, BT Race

The RaCe file has been manufactured since 1999 by FKG and was later distributed in the United States by Brasseler (Savannah, GA). The name, which stands for *r*eamer with *a*lternating *c*utting *e*dges, describes just one design feature of this instrument. Light microscopic imaging of the file shows flutes and reverse flutes alternating with straight areas; this design is aimed at reducing the tendency of files to thread into a root canal. Cross sections are triangular or square for .02 instruments with size #15 and #20 tips. The lengths of cutting parts vary from 9 to 16 mm.

The surface quality of RaCe instruments has been modified by electropolishing, and the two largest files (size #35, .08 taper and size #40, .10 taper) are also available in stainless steel. The tips are round and noncutting, and the instruments handles are color-coded and marked by and milled rings. RaCe instruments have been marketed in various packages to address small and large canals; recently they are sold as BioRaCe, purportedly to allow larger preparation sizes, with an emphasis on the use of .02 tapered instruments.

Few results of in vitro experiments comparing RaCe to other contemporary rotary systems are available,[443,444] Canals in plastic blocks and in extracted teeth were prepared by the RaCe system with less transportation than with ProTaper files.[443] In another study, ProTaper and RaCe performed similarly when canals were prepared to an apical size #30.[360] When preparing to a size #40, RaCe files prepared canals rapidly and with few aberrations or instrument deformities.[397] The newer BioRaCe instrument sequence utilizes .02 tapered instruments to promote larger apical sizes. As with any rotary system, this is also possible in a hybrid hand-rotary technique. BioRace instruments prepared S-shaped canals in plastic blocks (to size #40) similarly to ProTaper and MTwo but were superior when combined with S-Apex.[62] In a clinical study, Rocas and colleagues found no significant differences between NiTi hand file preparation and BioRace regarding the reduction of bacterial load.[413] A new variant, BT RaCe, has been introduced and incorporates different tip designs, as well as different sequences.

EndoSequence

The EndoSequence rotary instrument is produced by FKG in Switzerland and marketed in the United States by Brasseler. This instrument adheres to the conventional length of the cutting flutes, 16 mm, and to larger tapers, .04 and .06, to be used in a crown-down approach. The overall design, including the available tapers and cross sections, is thus similar to many other files; however, the manufacturer claims that a unique longitudinal design (called alternating wall contact points [ACP]) reduces torque requirements and keeps the file centered in the canal. It also has varying, comparatively small helical angles. Another feature of the EndoSequence design is an electrochemical treatment after manufacturing, similar to that of RaCe files, resulting in a smooth, polished surface. This is thought to promote better fatigue resistance, hence a rotational speed of 600 rpm is recommended for EndoSequence.[264] Most in vitro results, however, suggest that EndoSequence is not superior to other files in cyclic fatigue resistance.[217,277,401]

Twisted File

In 2008, SybronEndo presented the first fluted NiTi file manufactured by plastic deformation, a process similar to the twisting process that is used to produce stainless steel K-files: the Twisted File (TF). According to the manufacturer, a thermal process allows twisting during a phase transformation into the so-called R-phase of nickel-titanium. The instrument is currently available with a size tip from #25 to #50 and in tapers from .04 to .12.

The unique production process is thought to result in superior physical properties; indeed, early studies suggested significantly better fatigue resistance when size #25 .06 taper Twisted Files were compared to K3 instruments of the same size GTX instrument.[163] Moreover, as determined by bending tests according to the ISO norm for hand instruments, 3630-1, Twisted Files size #25 .06 taper were more flexible than ProFiles of the same size.[162] Others found similar levels for fatigue resistance for TF and Profile of similar size.[277]

A more recent development for TF is the use of an electric motor that allows different file movement, both continuous rotation and reciprocation, depending on the clinical situation (TF Adaptive, SybronEndo).

ProFile Vortex

Profile Vortex files are manufactured from NiTi. Two versions are on the market, one made from M-Wire and another from so-called blue wire (Vortex Blue, which showed greater resistance to cyclic fatigue and increased torque resistance), and they have varying helical angles to counteract the tendency of nonlanded files to thread into the root canal. Vortex instruments are recommended to be used at 500 rpm; higher rotational speed results in less torque generated.[37] Canal preparation with ProFile Vortex in vitro was similar to other rotary instruments.[83,563] Vortex instruments are available in sizes #15 to #50 and in .04 and .06 tapers.

MTwo

This instrument, originally sold in Italy by Sweden e Martina, was put onto the European market in 2004. The instrument has a two-fluted S-shaped cross section. The original strategy allowed for three distinct shaping approaches after the use of a basic sequence with tip sizes from #10 to #25 and tapers ranging from .04 to .06. Subsequent enlargement was meant to create apical sizes up to #40 .04 or, alternatively, to #25 .07 or to larger apical sizes with so-called apical files. MTwo is a well-researched and cutting-efficient instrument; clinically it is an example for the so-called single length technique. Canal shapes with MTwo were similar to other

contemporary root canal instruments, either in rotation or reciprocation.[80]

Summary

The market share for rotary files without radial lands continues to expand because of perceived higher efficacy. The overall incidence of clinically relevant preparation errors (details are discussed later) appears to be low, in spite of more aggressive cutting by files without radial lands. Instrument fractures remain a concern, as does the tendency of continuously rotating instruments to thread or pull into the canal, specifically as working length is approached.

Group III, Special Cases

WaveOne, Reciproc

A way to mitigate problems with continuous rotation (e.g., taper lock, fatigue fracture, threading-in) is to return to reciprocation, which had been used decades ago (e.g., in the Giromatic handpiece).[233] A case report[566] described this approach using ProTaper F2 in reciprocation. Based on experiments evaluating the maximal rotational angle before plastic deformation for the selected instrument, a forward angle of 144 degrees followed by a reverse rotation of 72 degrees was recommended.[566] This cycle continues at 400 rpm until working length is reached.

Subsequently two instruments specifically designed for reciprocation were brought to the market: WaveOne (Tulsa Dentsply Dental Specialties) and Reciproc (VDW, currently not available in the United States). WaveOne instruments are available in three tip sizes, #21, #25, and #40, with tapers of .06 and .08, respectively. Corresponding sizes for Reciproc are tips of #25, #40, and #50 with tapers of .08, .06, and .05, respectively. Both instruments feature variable tapers that are largest toward the tips. The main WaveOne cross section is triangular, similar to ProTaper, whereas Reciproc is a two-fluted file with a design similar to MTwo.

Special motors are used for both systems to provide reciprocation action with alternating counterclockwise and clockwise rotations of about 150 to 170 and 30 to 50 degrees, respectively.[151]

Both files are machined with left-leaning flutes; therefore, the cutting direction for both is clockwise. One problem that may occur with this design is the transportation of dentin debris into the apical area, rather than moving debris coronally. There is mixed evidence for this phenomenon[81,124] in vitro. Clinically, frequent careful cleaning of the cutting blades with a moist gauze is recommended.

The shaping ability of these systems, according to current in vitro data, seems similar to that of established systems with continuous rotation.[80,324,537]

Self-Adjusting File

The self-adjusting file (SAF; ReDent-Nova) represents a different approach, both in file design and mode of operation.[327] The file is really a cylindrical, hollow device, designed as a thin-walled NiTi lattice with a lightly abrasive surface (see Fig. 6-26). An initial glide path is established up to a #20 K-file to allow the insertion of the SAF file. The file is proposed to be compressed from its 1.5 mm diameter into dimensions equivalent to those of a #25 K-file. It is operated with a handpiece that generates in-and-out vibrations (4000 per minute) and 0.4 mm amplitudes. As noted, the file is hollow, which allows for continuous irrigation through the file while operated in the root canal.

In vitro data for this system suggest that indeed more wall contact is made compared to rotary files,[361,382] resulting in better debridement and antimicrobial efficacy.[289,472] Shaping quality is also on par with rotary instruments.[382,537]

Endo-Eze

The Giromatic handpiece (MicroMega), a rotary instrument system in use since 1969, delivers 3000 quarter-turn reciprocating movements per minute. Special rasps and barbed broaches made from stainless steel were most often used in Giromatic handpieces, but K-type and H-type instruments may also be used. The preparation quality with this system was insufficient in curved canals, and the technique fell into disregard.[233]

The Endo-Eze file system (Ultradent, South Jordan, UT) is a more recently introduced addition using a similar motion, provided by special equipment or an original Giromatic handpiece. The set has four engine-driven instruments that are designed to clean the middle third of the canal. The sizes and tapers are #10 and #13, with tapers ranging from 0.02 to 0.04. In this system, the use of stainless steel hand instruments is suggested for the apical third of the canal.

Preparation quality in curved canals appeared to be inferior to NiTi rotaries.[349,358] In straight canals, Endo-EZE performed similar to FlexMaster.[419]

Sonic and Ultrasonic Instruments

An alternative way of instrumenting root canals was introduced when clinicians became able to activate files by electromagnetic ultrasonic energy. Piezoelectric ultrasonic units are also available for this purpose. These units activate an oscillating sinusoidal wave in the file with a frequency of about 30 kHz.

Two types of units, ultrasonic and sonic, are marketed. Ultrasonic devices, which operate at 25 to 30 kHz, include the magnetostrictive Cavi-Endo (Dentsply Caulk, Milford, DE), the piezoelectric ENAC (Osada, Tokyo), the EMS Piezon Master 400 (Electro Medical Systems, Vallée de Joux, Switzerland), and the P5 Neutron (Acteon Satelec, Merignac Cedex, France) (Fig. 6-29). Sonic devices, which operate at 2 to 3 kHz, include the Sonic Air MM 1500 (MicroMega), the Megasonic 1400 (Megasonic Corp, House Springs, MO), and the Endostar (Syntex Dental Products, Valley Forge, PA).

FIG. 6-29 Example of an ultrasonic unit. (Neutron P5 piezoelectric, courtesy Acteon Satelec, Merignac Cedex, France).

Ultrasonic device holders can fit regular types of instrument blanks (e.g., K-files), whereas sonic devices use special inserts known as Rispi-Sonic, Shaper-Sonic, Trio-Sonic, or Heli-Sonic files.

Although similar in function, piezoelectric units have some advantages over the magnetostrictive systems. For example, piezoelectric devices generate little heat, so no cooling is needed for the handpiece. Magnetostrictive systems, however, generate considerable heat, and a special cooling system is needed in addition to the cooling effect provided by root canal irrigation. Working without water-cooling becomes essential when using an operating microscope, because the water spray may obstruct visualization.

The piezoelectric transducer transfers more energy to the file than does the magnetostrictive system, making it more cutting efficient. The file in an ultrasonic device vibrates in a sinus wave–like fashion. A standing wave has areas with maximal displacement (i.e., antinodes) and areas with no displacement (i.e., nodes). The tip of the instrument exhibits an antinode. If powered too high, especially with no contact with the canal wall, the instrument may break because of the intense vibration. Therefore, files must be used only for a short time, must remain passive within the canal, and the power must be controlled carefully. The frequency of breakage in files used for longer than 10 minutes may be as high as 10%, and the breakage normally occurs at the nodes of vibrations.[7] Ultrasonic devices have been linked to a higher incidence of preparation errors and to reduced radicular wall thickness.[298,321,582]

Summary

An abundance of NiTi systems is currently on the market. Table 6-1 lists these instruments systematically and illustrates some of the most relevant properties. Most systems included in Table 6-1 have files with tapers greater than the .02 stipulated by the ISO norm. Differences exist in tip designs, cross sections, and manufacturing processes. In vitro tests have continued to identify the effect of specific designs on shaping capabilities and fracture resistance. A desirable goal is the instrumentation of more canal surface, in an effort to make biofilm more susceptible to subsequent chemical disinfection. At the same time, instrumentation paradigms that conserve more dentin appear desirable to promote long-term function.[172] Meaningful differences in clinical outcomes in regard to various specific design variations have yet to be ascertained.[187,375,443]

Motors

Motors for rotary instruments have become more sophisticated since the simple electric motors of the first generation in the early 1990s (Fig. 6-30, *A*). Electric motors with gear reduction are most suitable for rotary NiTi systems, because they ensure a constant rpm level and constant torque. They can also be programmed to provide alternative rotation patterns, for example, reciprocation with freely selectable angles of rotation.[151] Electric motors often have presets for rpm and torque and are capable of delivering torques much higher than those required to break tips. Some authors think that torque-controlled motors (see Fig. 6-30, *B* to *D*) increase operational safety.[160] Others have suggested that such motors may mainly benefit inexperienced clinicians.[569] These motors probably do not reduce the risk of fracture caused by cyclic fatigue, and even if the torque is below the fracture load at D3, a fracture at a smaller diameter (D2) is still possible.

To complicate matters further, a differential exists between torque at failure at D3 and the working torque needed to operate an instrument effectively (Fig. 6-31 and Box 6-2).[57,374,383,432] In many cases the working torque is greater than the torque required to fracture the instrument's tip.

This differential between working torque and tip fracture load is especially large, with files having a taper equal to or greater than .06; therefore, these files are rather ineffective in

FIG. 6-30 Examples of motors used with rotary nickel-titanium endodontic instruments. **A**, First-generation motor without torque control. **B**, Fully electronically controlled second-generation motor with sensitive torque limiter. **C**, Frequently used simple torque-controlled motor. **D**, Newer-generation motor with built-in apex locator and torque control.

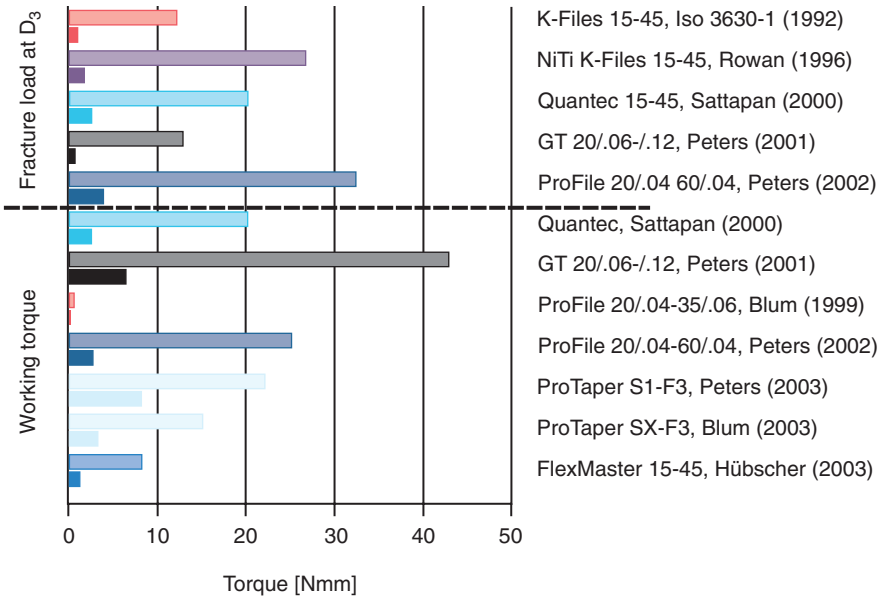


FIG. 6-31 Diagram comparing fracture loads at D_3 *(upper section of graph)* to torques occurring during preparation of root canals *(lower section of graph)*. Filled columns represent the largest file in each set, and open columns show the scores of the most fragile file (see text and Box 6-3 for details).

BOX 6-2

Instrument Breakage with Torsional Load (MacSpadden Factor)

For rotary instrument tips, susceptibility to breakage is governed by the quotient of torque needed to fracture divided by working torque. Simply put, the larger the value, the safer the file.

most torque-controlled motors. Future motors will likely offer more microprocessor controlled features—for example, information about the specific instrument being used, such as the presets and the usage history.

Certain motors have built-in apex locators (see Fig. 6-30, *D*). This type of motor may be programmed to stop a rotary instrument when working length is reached or go into reverse gear. Similar changes in movement can occur in an adaptive fashion, depending on the torsional load that the instrument experiences in the root canal. The TF Adaptive motor (SybronEndo) is an example for this technology.

Some of the factors, besides the motor itself, that may influence the incidence of fracture in engine-driven NiTi rotary instruments are lubrication, specific instrument motion, and speed of rotation. It cannot be overemphasized that NiTi rotary instruments should be used only in canals that have been flooded with irrigant. Although lubricants such as RC-Prep (Premier Dental Products Co., Plymouth Meeting, PA) and Glyde (Dentsply Maillefer) have been recommended in the past, their benefit may be restricted to plastic blocks[19] and less relevant when rotary instruments engage dentin surfaces. Moreover, experimental data for dentin suggest that the use of lubricants fails to reduce torque values during simulated canal preparation.[61,376] Finally, because of chemical interactions between NaOCl and ethylenediamine tetra-acetic acid (EDTA),[183] alternating irrigants and using lubricants that contain EDTA may even be counterproductive. Details in irrigation solution interactions are described later in this chapter.

For instrument motion, some manufacturers recommend a pecking, up-and-down motion. This not only prevents threading in of the file, it is also thought to distribute stresses away from the instrument's point of maximum flexure, where fatigue failure would likely occur.[285,394] However, such in-and-out movements did not significantly enhance the life span of ProFile .04 taper or GT rotary instruments rotated around a 5-mm radius cylinder with a 90-degree curve.[374,379] Furthermore, large variations were noted in the lengths of the fractured segments.[226,530] This suggests that ductile fractures may originate at points of surface imperfections. Files made of more martensitic alloy tend to work better with longer sweeping or brushing strokes. The notion of "brushing" is not directly related to a paintbrush motion, as this would predispose a file to bending and subsequent fatigue.[391] It rather refers to stroking against the wall away from the danger zone, the inner curvature of a curved root.

Rotational speed may influence instrument deformation and fracture. Some studies indicated that ProFile instruments with ISO-norm tip diameters failed more often at higher rotational speed,[131,159] whereas other studies did not find speed to be a factor.[116,252] For Vortex, 300 rpm reduced the generated torque and force; this higher rpm preset did not result in an increase of instrument fractures in vitro.[37]

Clinicians must fully understand the factors that control the forces exerted on continuously rotating NiTi instruments (Box 6-3). To minimize the risk of fracture and prevent taper lock, motor-driven rotary instruments should not be forced in an apical direction. Similarly, acute apical curves limit the use of instruments with higher tapers because of the risk of cyclic fatigue. The incidence of instrument fracture can be reduced to an absolute minimum if clinicians use data from well-designed torque and stress studies. Adequate procedural strategies, such as an adequate glide path, a detailed knowledge of anatomic structures, with avoidance of extreme canal

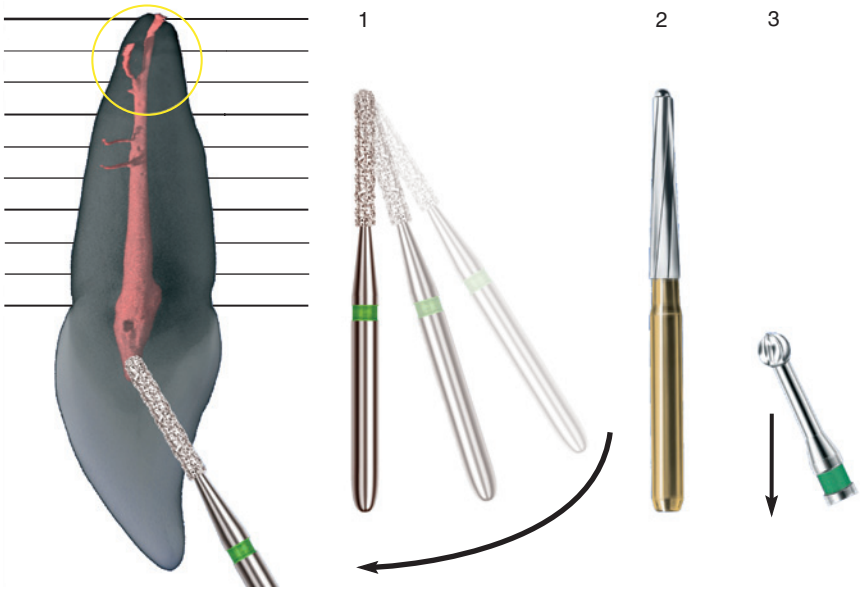


FIG. 6-32 Sequence of instruments used for optimal preparation of an access cavity (e.g., in an incisor). A parallel-sided diamond or steel bur is used to remove overlying enamel in a 90-degree angle toward the enamel surface *(1).* The bur is then tilted vertically to allow straight-line access to the root canal *(arrow).* A bur with a noncutting tip (e.g., Endo-Z bur or ball-tipped diamond bur) is then used to refine access *(2).* Overhangs or pulp horns filled with soft tissue are finally cleared with a round bur used in a brushing or pulling motion *(3).*

BOX 6-3

Factors Governing the Potential for Nickel-Titanium Rotary Instrument Fractures

- Clinician's handling (most important)
- Combination of torsional load, bending, and axial fatigue
- Root canal anatomy
- Manufacturing process and quality

configurations, and specific instrumentation sequences may also improve shaping results.

Certain procedures have evolved for removing fractured instruments from root canals (see Fig. 6-25); these are discussed in detail elsewhere in this book (see Chapters 8 and 19). Most of those methods require the use of additional equipment, such as a dental operating microscope and ultrasonic units. However, the best way to deal with instrument fracture is prevention. An understanding of the anatomy of the root canal system, together with a clear plan for selecting, sequencing, and using shaping instruments, can certainly help prevent procedural mishaps.

STEPS OF CLEANING AND SHAPING

Access: Principles

Access cavity preparation is essential for root canal treatment and is described in detail elsewhere in this book (Chapter 5). It should be emphasized at this point that mishaps during access (e.g., perforation) significantly affect the long-term outcome of a root canal treated tooth. Overenlargement or gouging during access significantly reduces structural strength[402] and may lead to root fracture and nonrestorable conditions.

Rationally, the use of a cylindrical diamond bur, followed by a tapered fluted bur with noncutting tip and perhaps a small round bur, is recommended (Fig. 6-32). One experiment suggested that more conservative access cavities, in particular in premolars, may increase fracture resistance and permit similar shaping outcomes.[270] The use of ultrasonically powered tips under magnification is aimed at achieving ideal access cavities (Fig. 6-33), including discovery and perhaps relocation of the canal orifices.

Coronal Preflaring

The extension of an access cavity into the coronal-most portion of the root canal is called *coronal flaring.* If a canal is constricted, mineralized, or difficult to access, flaring the coronal portion prior to any deep entry into the root canal is beneficial. This canal modification should be preceded by a scouting step, in which a small (e.g., size #10) K-file is passively placed several millimeters into the root canal. Tools for preflaring include Gates Gliddens (Fig. 6-34) and dedicated NiTi instruments (Fig. 6-35). Both step-back and step-down sequences have been recommended for Gates Gliddens, whereas NiTi rotaries are typically used in a crown-down sequence. More recently, specific orifice shaping rotaries have been developed that are used as a single instrument, rather than a sequence.

The use of laterally cutting NiTi rotaries allows the clinician to modify the root canal orifice to form a receptacle for subsequent instrumentation. This is related to removal of coronal mineralization and to relocation of the canal pathway by removal of dentin overhangs. For example, a second mesiobuccal canal typically exits the pulp chamber toward the mesial under a dentin shelf and then curves toward the distal. It is this curve in a small canal that often leads to formation of a ledge, which in turn prevents shaping to length.

Preenlargement of the coronal half to two thirds of a root canal allows files unimpeded access to the apical one third and

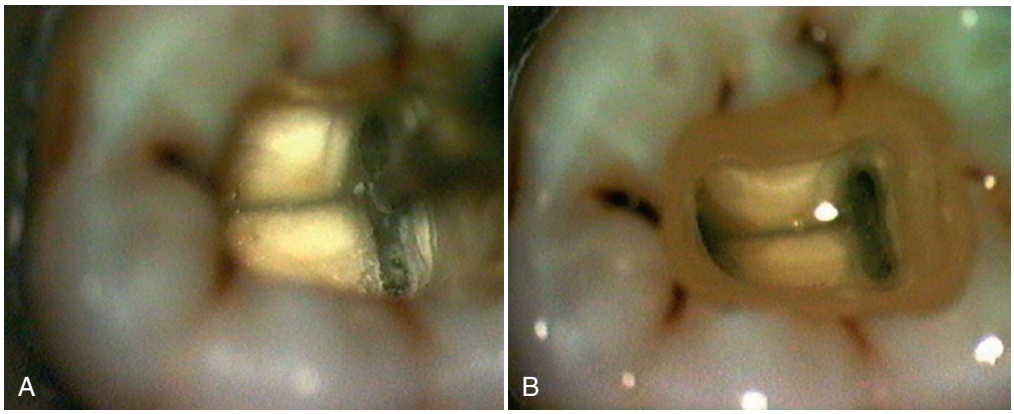


FIG. 6-33 Clinical views of an access cavity in a mandibular molar. **A,** As seen through an operating microscope (×20). **B,** Modification with an ultrasonically activated tip.

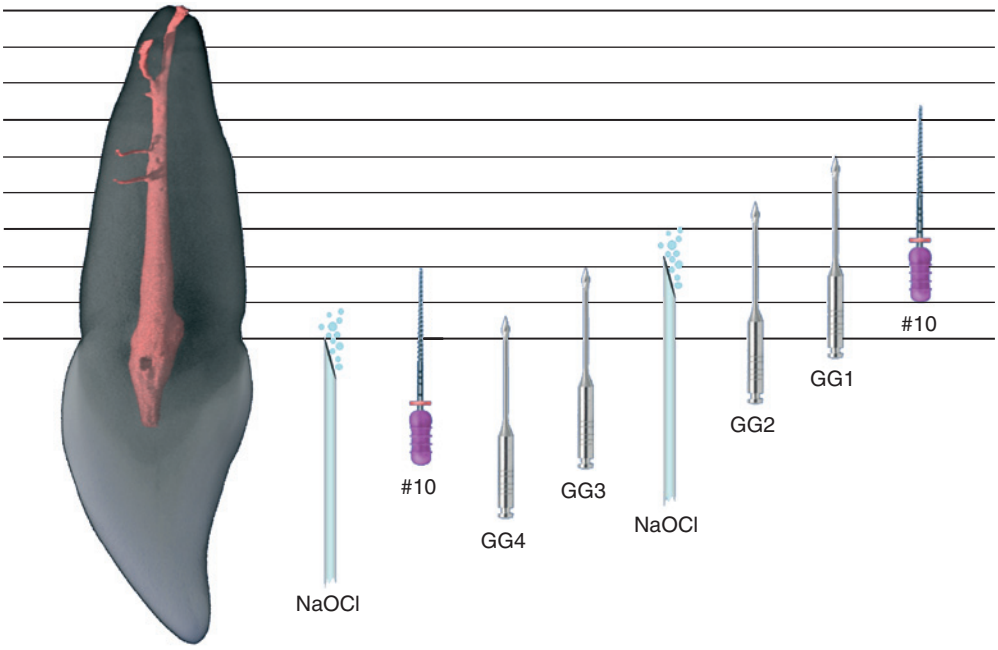


FIG. 6-34 Diagram of coronal enlargement in a maxillary anterior tooth. After preparation of the access cavity (see Fig. 6-32) and copious irrigation, Gates-Glidden burs are used in a step-down manner to enlarge the orifice and provide straight-line access into the middle third of the canal. Precurved size #10 K-files are used to explore the canal path and dimension.

gives the clinician better tactile control in directing small, adequately precurved negotiating files (Fig. 6-36).

One of the purported benefits of early coronal flaring is access of disinfecting irrigation solutions, but this has not been confirmed in experiments. On the other hand, a documented benefit of coronal flaring is mitigation of working length changes during canal preparation.[496]

Comparatively little is known about the efficacy of individual flaring instruments. One study compared the cutting efficacy of radial-landed and triangular cross sections.[338,380] The latter were shown to be more cutting efficient compared to radial-landed design; somewhat surprisingly, flexible martensitic alloy was cutting more rapidly compared to conventional NiTi alloys.

Patency File

A *patency file* is a small K-file (usually a size #10 or #15) that is passively extended slightly beyond the apical foramen. The use of a patency file has been suggested for most rotary techniques. This step is thought to remove accumulated debris, to help maintain working length, and to translate into greater clinical success.[345] One concern with the patency file was that instead of having a cleaning effect, the file would push contaminated debris through the foramen. However, an in vitro study suggested that the risk of inoculation was minimal when canals were filled with NaOCl.[243] Maintaining patency throughout an endodontic procedure does not lead to an increase in posttreatment symptoms.[22] Only initial clinical evidence exists favoring the use of a patency file; however, experience suggests

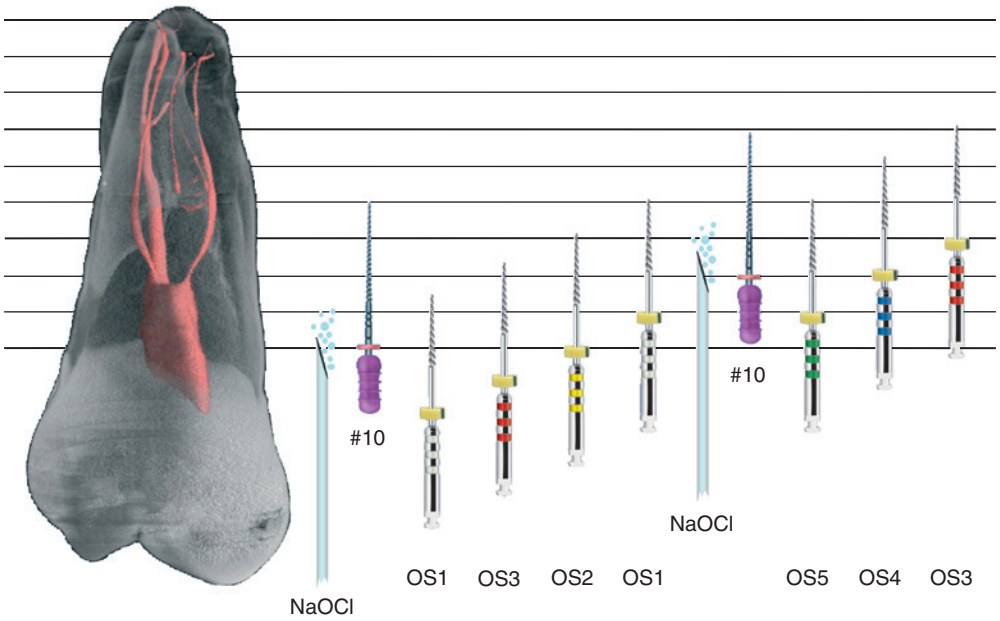


FIG. 6-35 Diagram of coronal enlargement in a more complicated maxillary posterior tooth. This maxillary molar presents several difficulties, including a narrow mesiobuccal canal that exits the pulp cavity at an angle. A possible approach in a case involving difficult entry into the root canal system is to use a small orifice shaper (OS1) after ensuring a coronal glide path with a K-file. Use of a sequence of orifice shapers (OS3 to OS1) then allows penetration into the middle third of the root canal. Wider canals can accept a second sequence of orifice shapers. Copious irrigation and securing a glide path with a size #10 K-file are prerequisites for use of NiTi rotary instruments.

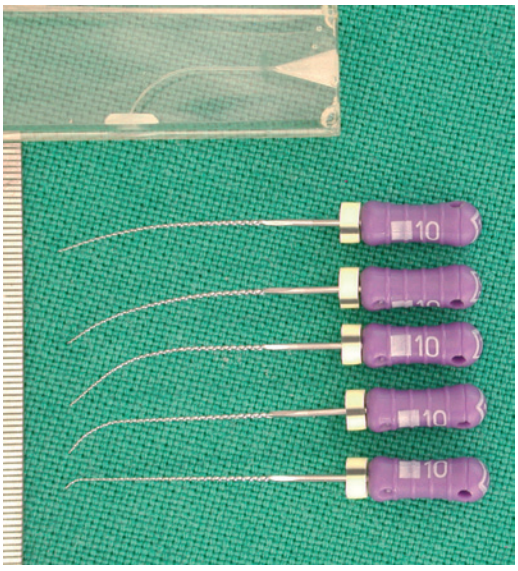


FIG. 6-36 Various precurved, stainless steel hand files for pathfinding and gauging. Compare the curves in the instruments to the ones in a plastic training block (gradation of ruler is 0.5 mm).

that this technique involves relatively little risk and provides some benefit as long as small files are used carefully.

Working Length Determination

Devices

Radiographs, tactile sensation, the presence of moisture on paper points, and knowledge of root morphology have been used to determine the length of root canal systems. Following earlier experiments by Custer,[110] Sunada developed the first commercialized electronic apex locator,[501] suggesting that the apical foramen could be localized using a direct electric current. Currently, the electronic apex locator is considered an accurate tool for determining working length.[153] One study reported that the use of electronic apex locators in a dental student clinic resulted in a higher quality of obturation length control and an overall reduction in the number of radiographs taken.[152] However, these devices must not be considered flawless, because several variables are known to affect their accuracy. For example, immature roots can present problems.[234] Once roots mature (i.e., form a narrow apical foramen) and instruments are able to contact the canal walls, an electronic apex locator's accuracy greatly improves. Some investigators have found no statistical difference between roots with vital and necrotic tissue.[175,319] Because apical root resorption is prevalent in necrotic cases with longstanding apical lesions,[541] it may be concluded that apical resorption does not have a significant effect on the accuracy of electronic apex locators.

Some clinicians have advocated the use of the electronically determined working length in lieu of working length estimations using the placement of a file in the canal and a radiograph. However, combined use of both of these techniques has been shown to result in greater accuracy.[136] Furthermore, radiographs may add essential anatomic information that could be missed if electronic apex locators are used exclusively.

The first two generations of electronic apex locators were sensitive to the contents of the canal and irrigants used during treatment. The development of an algorithm called the *ratio measurement method* distinguished the third generation of apex locators.[263] To arrive at this method, the impedance of the canal was measured with two current sources of different frequencies, and a quotient was determined using the electrical potentials proportional to each impedance.[263] This study found that

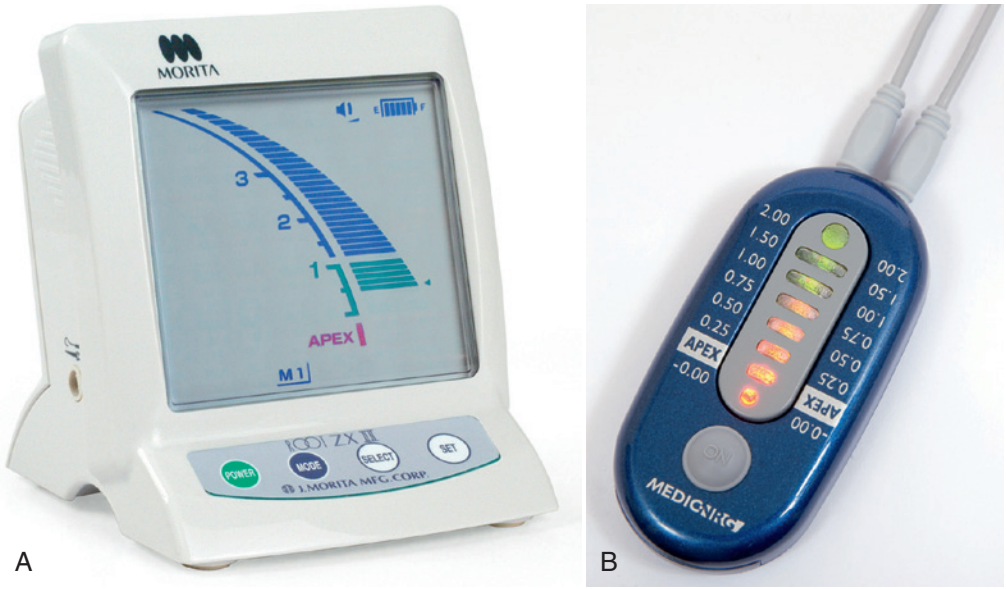


FIG. 6-37 A, Root ZX apex locator with lip clip and file holder. B, NRG miniature electronic apex locator. (*A*, Courtesy J Morita, Irvine, CA. *B*, Courtesy MedicNRG, Kibutz Afikim, Israel.)

electrolytes did not have a significant effect on the accuracy of the unit. This means that clinically canal contents need not be dried, but fluids in contact with crowns or coronal metallic restoration materials could conduct currents and lead to false results.

Some third-generation apex locators are the Endex Plus, or Apit (Osada, Los Angeles, CA), the Root ZX (J. Morita, Kyoto, Japan),[153,276] and the Neosono Ultima EZ (Acteon Satelec). The Endex Plus device uses 1 and 5 kHz and provides apex location based on subtraction. The Root ZX emits currents at frequencies of 8 and 0.4 kHz and provides apex location based on the resulting quotient. In addition to latest model apex locators, manufacturers have developed smaller models (Fig. 6-37, *B*).

Apex locators are generally safe to use; however, manufacturers' instructions state that they should not be used on patients with pacemakers without consulting the patient's cardiologist. However, when connected directly to cardiac pacemakers in vitro, electronic apex locators did not interfere with the function of the pacemaker,[165] and they did not interfere with the functioning of any of the cardiac devices tested in a clinical study under electrocardiogram monitoring.[556]

Strategies

Anatomic studies and clinical experience suggest that typically teeth are 19 to 25 mm long. Most clinical crowns are approximately 10 mm long, and most roots range from 9 to 15 mm in length. Roots, therefore, can be divided into thirds that are 3 to 5 mm long. An important issue in root canal treatment is the apical end point of the prepared shape in relation to the apical anatomy. Traditional treatment has held that canal preparation and subsequent obturation should terminate at the *apical constriction*, the narrowest diameter of the canal (Fig. 6-38). This point is thought to coincide with the cementodentinal junction (CDJ) (see Chapter 12) and is based on histologic sections and ground specimens. However, the position and anatomy of the CDJ vary considerably from tooth to tooth, from root to root, and from wall to wall in each canal. Moreover, the CDJ cannot be located precisely on radiographs. For this reason, some have advocated terminating the preparation in necrotic cases at 0.5 to 1 mm short of the radiographic apex and 1 to 2 mm short[206,407,562] in cases involving irreversible pulpitis. Although there is no definitive validation for this strategy at present,[435] well-controlled follow-up studies seem to support it.[476,478]

Working to shorter lengths may lead to the accumulation and retention of debris, which in turn may result in apical blockage (Fig. 6-39). If the path to the apex is blocked, working to short lengths may contribute to procedural errors such as apical perforations and fractured instruments. Such obstacles (which consist of collagen fibers, dentin mud, and, most important, residual microbes) in apical canal areas are a major cause of persistent or recurrent apical periodontitis,[196,342,466] or *post-treatment disease*[156,559] (also see Chapters 14 and 15).

Using an electronic apex locator has helped clinicians identify the position of apical foramina more accurately and allow safe canal shaping as close as 0.5 mm to the canal terminus.

Canal Enlargement/Preparation

Rationale

Like the position of the apical constriction, apical diameters are difficult to assess clinically.[274] Some have recommended gauging canal diameters by passing a series of fine files apically until one fits snugly. However, such an approach is likely to result in underestimation of the diameter.[555] This is a crucial point because the initial canal size is a major determinant for the desired final apical diameter.

An ongoing debate exists between those who prefer smaller apical preparations combined with tapered shapes and those who favor larger apical preparations for better removal of infected dentin and to allow irrigation fluids access to the apical areas (Table 6-2). Both sides stress the importance of maintaining the original path of the canal during preparation; otherwise, bacteria in the apical one-third of the root canal may not be reached by sufficient amounts of an antimicrobial agent.[335] Investigators demonstrated a higher percentage of

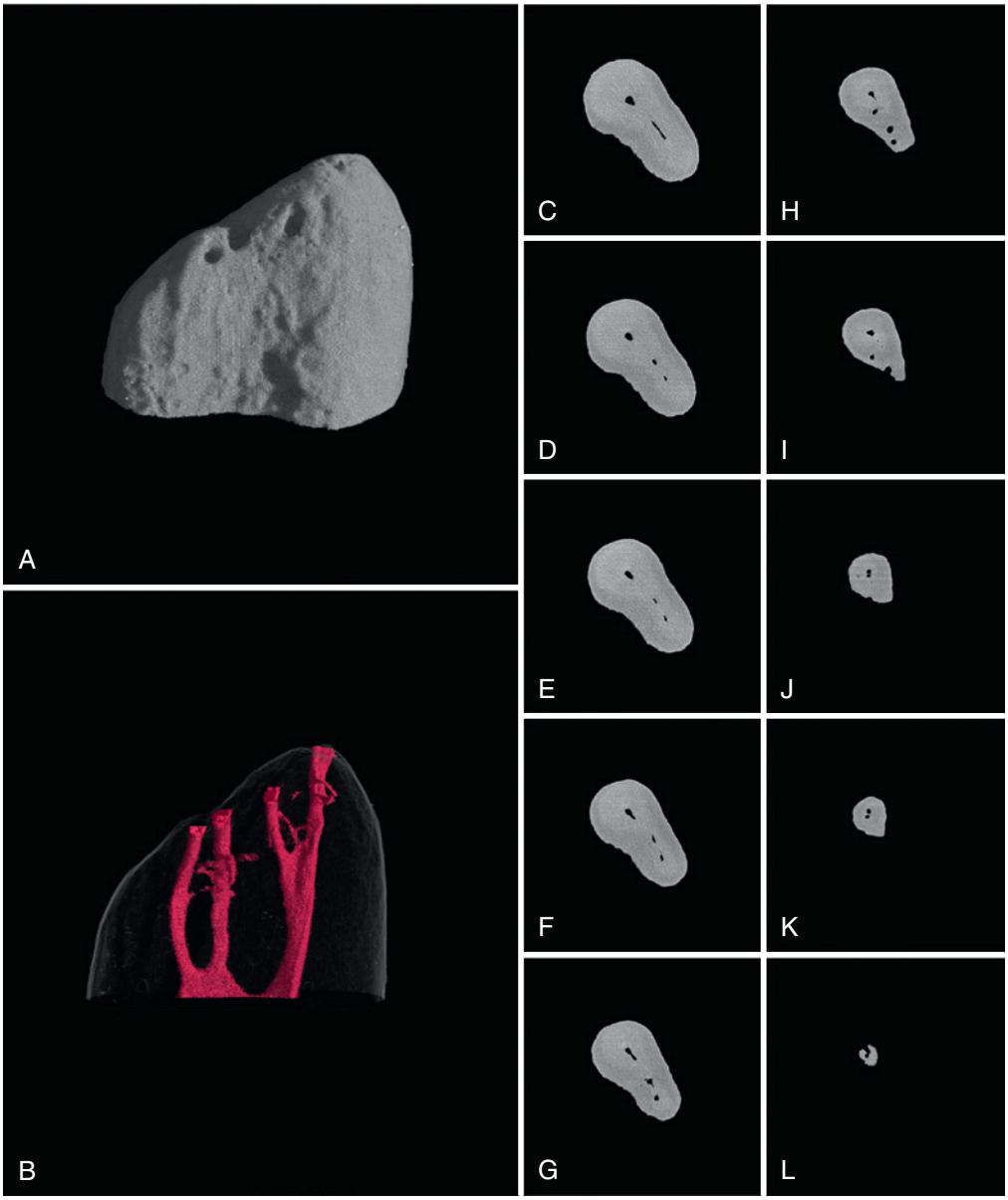


FIG. 6-38 Microcomputed tomographic scan of anatomy of the apical 5 mm of a mesiobuccal root (8 μm resolution). **A-B,** Three-dimensional reconstruction of outer contour and root canal systems. **C-L,** Cross sections of root at 0.5 mm apart.

TABLE 6-2

Characteristics of Wide and Narrow Apical Preparations

Root Canal Preparation	Benefits	Drawbacks
Narrow apical size	Minimal risk of canal transportation and extrusion of irrigants or filling material Can be combined with tapered preparation to counteract some drawbacks Less compaction of hard tissue debris in canal spaces	Little removal of infected dentin Questionable rinsing effect in apical areas during irrigation Possibly compromised disinfection during interappointment medication Not ideal for lateral compaction
Wide apical size	Removal of infected dentin Access of irrigants and medications to apical third of root canal	Risk of preparation errors and extrusion of irrigants and filling material Not ideal for thermoplastic obturation

FIG. 6-39 Presence of dentin dust as a possible source of microbial irritation. Tooth #18 underwent root canal therapy. The clinician noted an apical blockage but was unable to bypass it. Unfortunately, intense pain persisted and at the patient's request, the tooth was extracted a week later. **A**, Mesial root of tooth #18; mesial dentin has been removed. **B**, Magnified view (×125) of rectangle in **A** shows an apical block (gradation of ruler is 0.5 mm).

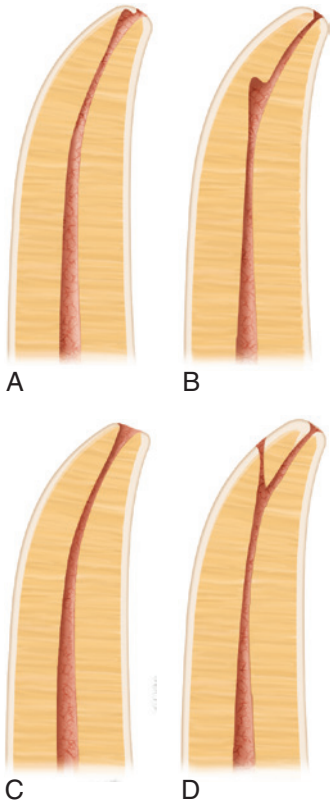


FIG. 6-40 Schematic diagrams showing the most common preparation errors. **A**, Apical zip. **B**, Ledge. **C**, Apical zip with perforation. **D**, Ledge with perforation.

bacterial elimination in single-root canal systems by using a combination of significant enlargement of the apical third and NaOCl irrigation.[89] Preparation errors (e.g., zips, canal transportation) may occur in larger preparations, with both stainless steel and NiTi instruments (Fig. 6-40).

Thorough disinfection of the apical part of a root canal is essential, because this area is most likely to harbor residual intraradicular bacteria.[343] Wider apical preparations remove potentially infected dentin, allowing the delivering needle and subsequently the antimicrobial irrigant to penetrate the root canal more deeply.[101,147]

A study investigating rotary NiTi files of three tapers (.06, .08, and .10) with file tips in sizes #20, #30, and #40 showed that size #20 instruments left significantly more debris in the apical third compared with size #40 instruments.[532] On the other hand, a study in which half the samples were prepared to a size #25 file and the other half to a size #40 file found no statistically significant difference in bacterial growth after instrumentation, with no growth observed after 1 week of treatment with a $Ca(OH)_2$ dressing.[570] Step-down sequences with additional apical enlargement to ISO size #35 and a serial step-back technique with no apical enlargement were compared, using NaOCl and EDTA as irrigants. Here, no significant difference was detected in colony-forming units with or without apical enlargement.[105] These researchers concluded that dentin removal in the apical third might be unnecessary if a suitable coronal taper is achieved.

Despite the disagreement over the appropriate width of a preparation (see Table 6-2), these studies suggest that root canal preparations should be confined to the canal space, should be sufficiently wide, and should incorporate the original root canal cross sections (see Fig. 6-4).

Traditional cleaning and shaping strategies (e.g., the step-back technique) focused immediate preparation of the apical third of the root canal system, followed by various flaring techniques to facilitate obturation.[180,436,518] In an attempt to reach the canal terminus, the clinician first selected a small file, placed an appropriate curve on the instrument, and then tried to work the file to full length. If the terminus could not be reached, the file was removed and, after irrigation, either the same file or a smaller one was inserted. However, not infrequently, full length was not obtained, either because of blockage or because of coronal binding.

Coronal binding is caused by overhangs at the orifice level and when the canal is less tapered than an instrument, making it bind coronally. Moreover, a straight root often contains a curved canal, such as buccal and lingual curvatures that cannot be seen on radiographs.[109,389] In addition, passing a precurved negotiating file through a coronally tight canal will straighten the instrument.[487]

Various instrumentation sequences have been developed for hand and rotary instruments; these are discussed later in this chapter. However, the shape of the access cavity is *the* prerequisite that must be optimized before any canal preparation can take place (see Chapter 5).

As stated previously, preparation of an adequate access cavity (see Fig. 6-32) may involve the use of a cylindrical diamond or fissure bur, a safety-ended drill for additional enlargement, and round burs to remove overhangs on outward strokes. The access cavity shape must allow instruments unimpeded access to the middle third of the root canal system. Ultrasonically powered instruments used under an operating microscope greatly facilitate removal of mesial dentin shelves in mandibular molars (see Fig. 6-33, *A* and *B*) and other teeth. Preexisting restorations allow for ideal access cavities that serve as reservoirs of irrigants (see Fig. 6-33, *C*).

Basic cleaning and shaping strategies for root canal preparation can be categorized as crown-down, step-back, apical widening, and hybrid techniques. In a crown-down approach, the clinician passively inserts a large instrument into the canal up to a depth that allows easy progress. The next smaller instrument is then used to progress deeper into the canal; the third instrument follows deeper again, and this process continues until the terminus is reached. Both hand and rotary instruments may be used in a crown-down manner. However, instrument sets with various tip diameters and tapers allow the use of either decreasing tapers or decreasing diameters for apical progress. Debate continues as to which of those strategies is superior for avoiding taper lock; currently, no compelling evidence favors either of them.

In the step-back approach, working lengths decrease in a stepwise manner with increasing instrument size. This prevents less flexible instruments from creating ledges in apical curves while producing a taper for ease of obturation.

As discussed previously, the aim of apical widening is to fully prepare apical canal areas for optimal irrigation efficacy and overall antimicrobial activity. Apical enlargement has been broken down into three phases: preenlargement, apical enlargement, and apical finishing.[545]

Most rotary techniques require a crown-down approach to minimize torsional loads,[57] and they reduce the risk of instrument fracture. Used sequentially, the crown-down technique can help enlarge canals further. All basic techniques described so far may be combined into a hybrid technique to eliminate or reduce the shortcomings of individual instruments.

Root canal preparation can be broken down into a series of steps that parallel the insertion depths of individual instruments. Anatomic studies and clinical experience suggest that most teeth are 19 to 25 mm long. Most clinical crowns are approximately 10 mm long, and most roots range from 9 to 15 mm in length. Roots, therefore, can be divided into thirds that are 3 to 5 mm long.

Provided adequate tools are used and the access cavity design is appropriate, excessive thinning of radicular structures can be avoided (see Fig. 6-5). Vertical root fractures and perforations are possible outcomes of excessive removal of radicular dentin in zones that have been termed *danger zones*.[15] Overenthusiastic filing, for example, may lead to more procedural errors (see Figs. 6-22 and 6-40). On the other hand, ideal preparation forms without any preparation errors and with circular incorporation of the original canal cross sections may be achieved with suitable techniques (see Fig. 6-4).

Dedicated NiTi instruments have been introduced or suggested for coronal preenlargement, such as the ProFile orifice shapers, GT accessory files, the ProTaper SX, the FlexMaster Intro file, and the size #40, .10 taper or size #35, .08 taper RaCe files. These instruments are better suited than GGs and safer for more difficult cases (see Figs. 6-34 and 6-35).

Techniques

Standardized Technique

The standardized technique adopts the same working length definition for all instruments introduced into a root canal. It therefore relies on the inherent shape of the instruments to impart the final shape to the canal. Negotiation of fine canals is initiated with lubricated fine files in a so-called watch-winding movement. These files are advanced to working length and worked either in the same hand movement or with "quarter-turn-and-pull" until a next larger instrument may be used. Conceptually, the final shape should be predicted by the last instrument used (Fig. 6-41). A single matching gutta-percha point may then be used for root canal filling. In reality, this concept is often violated: curved canals shaped with the standardized technique will be wider than the last used instrument,[14] exacerbated by the pulling portion of the hand movement. Moreover, adequate compaction of gutta-percha in such small a taper (~.02) is difficult or impossible[13] (see Chapter 7).

The standardized technique was hampered by the very standardization of the instruments used to perform the technique. Specifically, the similar size increment by 0.05 mm up to size 55 was clinically more difficult to achieve moving from size #10 to size #15, compared to the step from size #40 to #45. In very small files (sizes #6 to #10), the problem is partly resolved by several key points: (1) apical dimensions are such that a size #6 file does not significantly remove dentin other than in severely calcified cases; (2) a size #8 file taken 0.5 to 1 mm long to establish patency (discussed later in the chapter) contacts the desired end point of the preparation with a diameter approaching the tip size of a #10 file; (3) similarly, placing a size #10 file just minutely through the foramen eases the way for passive insertion of the subsequent #15 file to full length.[417]

Step-Back Technique

Realizing the importance of a shape larger than that produced with the standardized approach, one investigator suggested the step-back technique,[552] incorporating a stepwise reduction of the working length for larger files, typically in 1-mm or 0.5-mm steps, resulting in flared shapes with 0.05 and 0.10 taper, respectively (Fig. 6-42). Incrementally reducing the working length when using larger and stiffer instruments also reduced the incidence of preparation errors, in particular in curved canals. This concept appeared to be clinically very effective.[339]

Although the step-back technique was primarily designed to avoid preparation errors in curved canals, it applies to the preparation of apparently[109,439] straight canals as well. Several modifications of the step-back technique have been described over the years. Another investigator advocated the insertion of progressively larger hand instruments as deep as they would passively go in order to explore and provide some enlargement prior to reaching the working length.[518]

Step-Down Technique

Other investigators described a different approach.[174] They advocated shaping the coronal aspect of a root canal first, before apical instrumentation commenced. This technique is intended to minimize or eliminate the amount of necrotic debris that could be extruded through the apical foramen during instrumentation.[146] Moreover, by first flaring the coronal two thirds of the canal, apical instruments are unimpeded through most of their length. This in turn may facilitate greater control and less chance of zipping near the apical constriction.[280]

Crown-Down Technique

Numerous modifications of the original step-down technique have been introduced, including the description of the crown-down technique.[150,317,433] The more typical step-down technique includes the use of a stainless steel K-file exploring the apical constriction and establishing working length. In

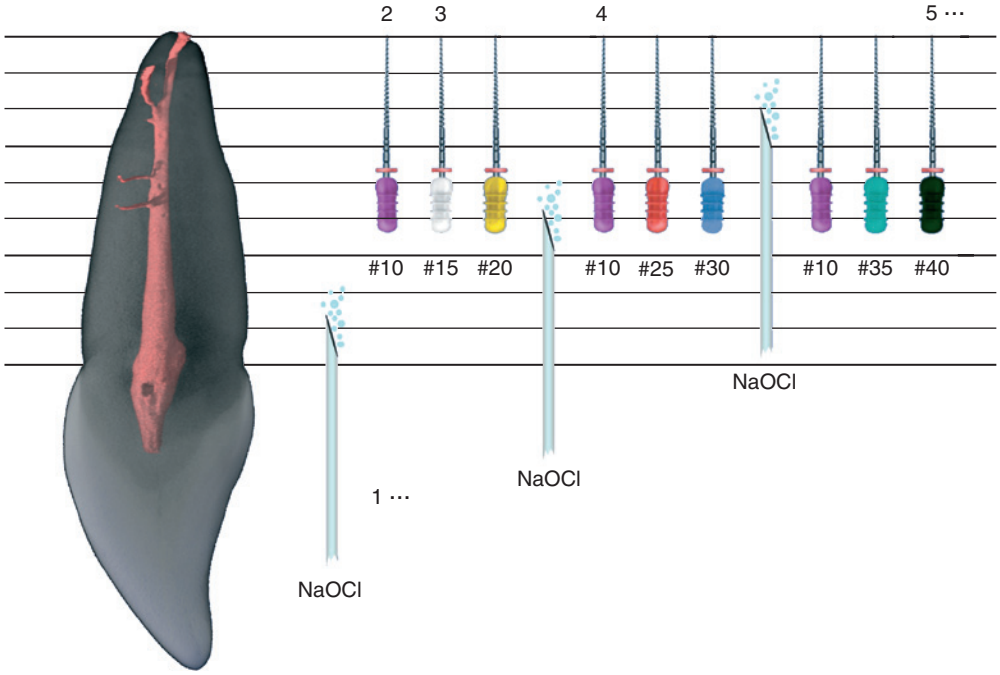


FIG. 6-41 Root canal instrumentation with hand files: Part I. After the orifice has been accessed and copious irrigation performed *(1),* the working length (WL) is determined. A size #10 or #15 K-file is advanced to the desired apical preparation end point, aided by an electronic apex locator *(2).* The apical canal areas are then enlarged with K-files *(3)* used in the balanced force technique (see Fig. 9-43). Frequent, copious irrigation with sodium hypochlorite is mandatory to support antimicrobial therapy. Frequent recapitulation with fine K-files is recommended to prevent blockage *(4).* Apical enlargement is complete to the desired master apical file (MAF) size *(5),* which depends on pretreatment canal sizes and individual strategy. Typically, size #40 or larger may be reached in anterior teeth, as in this example. File sizes larger than #20 may be used with NiTi instruments (e.g., NiTiFlex).

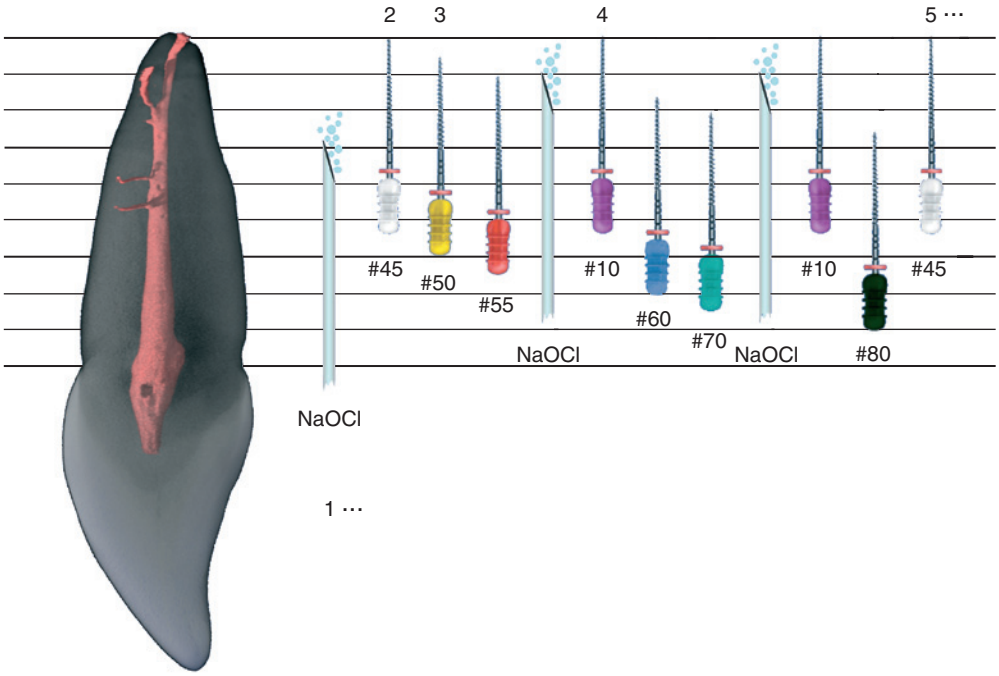


FIG. 6-42 Root canal instrumentation with hand files: Part II. Frequent irrigation with sodium hypochlorite *(1)* is more efficient after the working length (WL) is reached, because irrigation needles may penetrate deeper into the canal. Canal taper is increased to further improve antimicrobial efficiency and to simplify subsequent obturation. Hand instruments are set to decreasing working length in 0.5 mm increments (step-back) from the master apical file (MAF) (*2 and 3*). A fine K-file is used to recapitulate to WL during the procedure *(4),* and the MAF is used as a final recapitulation *(5)* to ensure that remaining dentin chips have been removed.

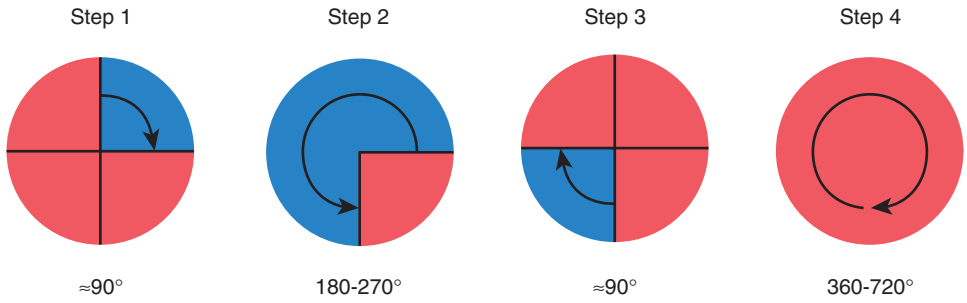


FIG. 6-43 Diagram of handle movements during balanced force hand preparation. **Step 1:** After pressureless insertion of a Flex-R or NiTiFlex K-file, the instrument is rotated clockwise 90 degrees, using only light apical pressure. **Step 2:** The instrument is rotated counterclockwise 180 to 270 degrees; sufficient apical pressure is used to keep the file at the same insertion depth during this step. Dentin shavings are removed with a characteristic clicking sound. **Step 3:** This step is similar to step 1 and advances the instrument more apically. **Step 4:** After two or three cycles, the file is loaded with dentin shavings and is removed from the canal with a prolonged clockwise rotation.

contrast, a crown-down technique relies more on coronal flaring and then determination of the working length later in the procedure.

To ensure penetration during step-down, one may have to enlarge the coronal third of the canal with progressively smaller GG drills or with other rotary instruments. Irrigation should follow the use of each instrument and recapitulation after every other instrument. To properly enlarge the apical third and to round out ovoid shape and lateral canal orifices, a reverse order of instruments may be used starting with a size #20 (for example) and enlarging this region to a size #40 or #50 (for example). The tapered shape can be improved by stepping back up the canal with larger instruments, bearing in mind all the time the importance of irrigation and recapitulation.

The more typical crown-down, or double-flare, technique[150] consisted of an exploratory action with a small file, a crown-down portion with K-files of descending sizes, and an apical enlargement to size #40 or similar. The original technique included stepping back in 1-mm increments with ascending file sizes and frequent recapitulations with a small K-file and copious irrigation. It is further emphasized that significant wall contact should be avoided in the crown-down phase to reduce hydrostatic pressure and the possibility of blockage. Several studies demonstrated more centered preparations in teeth with curved root canals shaped with a modified double-flare technique and Flex-R files compared to shapes prepared with K-files and step-back technique.[434,433] A double-flare technique was also suggested for ProFile rotary instruments.[447]

Balanced Force Technique

Regarding hand movements, a general agreement exists that the so-called balanced force technique creates the least canal aberrations with K-files. This technique has been described as a series of rotational movements for Flex-R files,[412] but it can also be used for K-files and other hand instruments such as GT hand files. Many explanations have been offered for the obvious and undisputed efficacy of the balanced force approach,[94,275,439] but general agreement exists that it provides excellent canal-centering ability, superior to other techniques with hand instruments.[30,71,283]

The balanced force technique involves three principle steps.[412] The first step (after passive insertion of an instrument into the canal) is a clockwise rotation of about 90 degrees to engage dentin (Fig. 6-43). In the second step, the instrument is held in the canal with adequate axial force and rotated counterclockwise to break loose the engaged dentin chips from the canal wall; this produces a characteristic clicking sound. Classically, in the third step, the file is removed with a clockwise rotation to be cleaned; however, because files used with the balanced force technique are not precurved, every linear outward stroke essentially is a filing stroke and may lead to some straightening of the canal path. Therefore, in many cases, the clinician may advance farther apically rather than withdrawing the file, depending on the grade of difficulty.

Rotary Instrumentation

NiTi rotary instruments are an invaluable adjunct in the preparation of root canals, although hand instruments may be able to enlarge some canals just as efficiently when used in appropriate sequences. Hand instruments should be used only after coronal preenlargement (e.g., with GG drills). After preenlargement, the access cavity and canals are flooded with irrigant, and a precurved scouting file is advanced into the canal. A lubricant when using hand instruments can help prevent apical blockage in this early stage. Once the working length has been established (aided by an electronic apex locator and radiographically verified), apical preparation to begins to facilitate a glide path for subsequent rotary instrumentation (Fig. 6-44).

The term *glide path* has been used in endodontics since the early 2000s[384] and relates to securing an open pathway to the canal terminus that subsequent engine-driven instruments can follow. The typical minimum glide path is a size #15 to #20 K-file and should be confirmed with a straight, not precurved file. Various small engine-driven instruments were introduced to simplify this process, such as Pathfiles (Dentsply Maillefer),[53] Scout RaCe (FKG Dentaire SA, La Chaux-de-Fonds Switzerland),[344] and G-file (MicroMega).[112] Copious irrigation and frequent recapitulations with a smaller file to working length may be required, and in some instances, clinicians must devise creative strategies using small crown-down or step-back sequences.

Fig. 6-45 illustrates the development of two different shapes in the mesial root canals of a mandibular molar, clearly showing that substantial areas of the root canal surface in either case are not instrumented, even when apical size #50 or 0.09 taper are reached (see the red areas in Fig. 6-45, *G* and *I*).

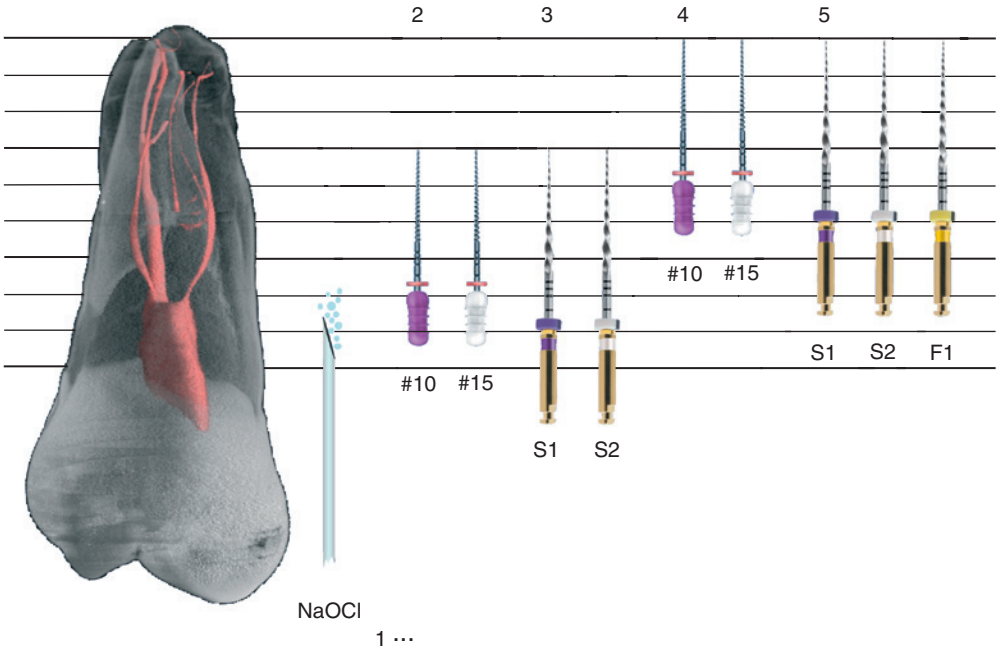


FIG. 6-44 Instrumentation of root canals with ProTaper instruments. After irrigation and scouting (*1* and *2*), the coronal thirds are enlarged with shaping files S1 and S2. Hand files then are used to determine the WL and to secure a glide path. Apical preparation is completed with S1 and S2. Finishing files are used to the desired apical width.

Specific NiTi Instrumentation Techniques

Crown Down

This was the dominant approach for many years and is still being used, for example, for ProFile and several others (ProFile Vortex, HERO 642, K3, and FlexMaster). It must be noted that the manufacturers' instructions for these systems vary somewhat, and the instructions for GT rotary, RaCe, and the Twisted File vary even more. Clinicians should always read the manufacturers' instructions for details on working with those instruments.

Working length is determined after any coronal preenlargement , and an open glide path is secured with K-files up to size #15 or #20, depending on the canal anatomy. If canal size permits, canal preparation begins with .06 taper instruments in descending tip diameters.[57] In more difficult small canals, .06 tapers are followed by .04-tapered instruments, also with descending tip diameters. Apical preparation is performed either with multiple shaping waves, as suggested for GT rotary files,[76] or in a step-back manner.[445] Recapitulation with a small hand file is recommended throughout the preparation.

Single Length

The approach for ProTaper Universal and ProTaper Next instruments differs from that for many other NiTi rotary files (except MTwo, WaveOne and Reciproc) in that no traditional crown-down procedure is performed (see Fig. 6-44).

Size #10 and #15 hand files are passively inserted into the coronal two thirds of a root canal as path-finding files, which confirm the presence of a smooth, reproducible glide path. This step is essential for ProTaper shaping instruments, because they are mostly side cutters and have fine, fragile tips.

Shaping files S1 and S2 are then passively inserted into the scouted canal spaces, which have been filled with irrigant (preferably NaOCl). If necessary, the SX file can be used at this stage to relocate orifices or remove obstructing dentin. After each shaping file is used, the canals are reirrigated, and a size #10 file is used to recapitulate to break up debris and move it into solution. This process is repeated until the depth of the path-finding #10 or #15 file is reached.

After irrigation, the apical third is fully negotiated and enlarged to at least a size #15 K-file, and the working length is confirmed (see Fig. 6-44). Depending on the canal anatomy, the rest of the apical preparation can be done with engine-driven ProTaper shaping and finishing hand files. As an alternative, handles can be placed on these instruments so that they can be used for the balanced force technique.

ProTapers S1 and S2 are then carried to the full working length, still in a floating, brushing motion. The working length should be confirmed after irrigation and recapitulation with a K-file, aided by an electronic apex locator or radiographs. Because of the progressive taper and more actively cutting flutes higher up in the ProTaper design, interferences in the middle and coronal thirds are removed at this stage.

The preparation is finished with one or more of the ProTaper finishing files, used in a nonbrushing manner; because of their decreasing taper, these files will reach the working length passively. Recapitulation and irrigation conclude the procedures (see Fig. 6-44).

LightSpeed Technique

Since the introduction of LightSpeed instruments, the manufacturer's guidelines have changed. This section presents a version used for the original LightSpeed rather than the LS variant (Fig. 6-46).

After access and coronal preenlargement with the instrument of choice, working lengths are obtained, and apical enlargement is done with at least a loose-fitting size #15 K-file. LSX instruments are then slowly advanced to working length

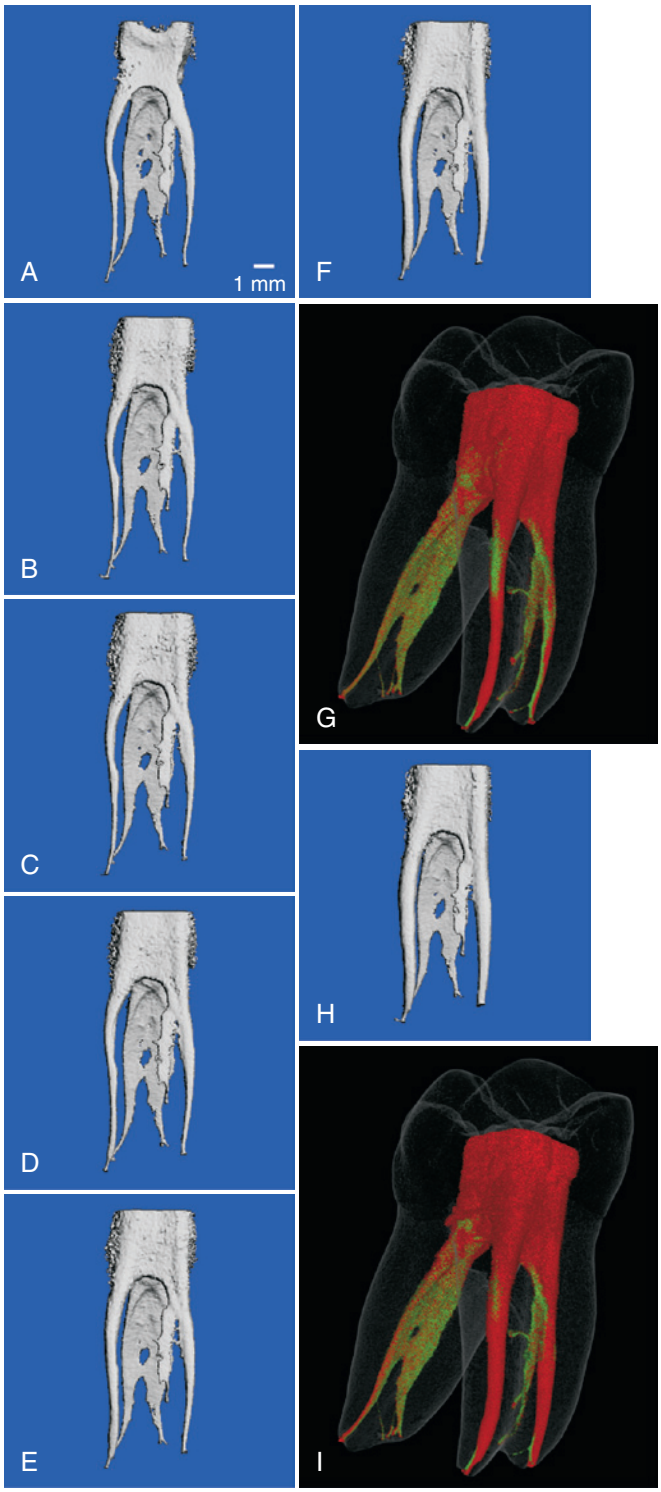


FIG. 6-45 Stepwise enlargement of mesial root canal systems in an extracted mandibular molar demonstrated with microcomputed tomography (μCT) reconstructions. The buccal canal *(left)* was prepared with a LightSpeed (LS) instrument, and the lingual canal *(right)* was shaped with a ProTaper (PT) instrument. **A**, Pretreatment view from the mesial aspect. Note the additional middle canal branching from the lingual canal into the coronal third. **B**, Initial preparation and opening of the orifices, aided by ultrasonically powered instruments. **C**, First step of root canal preparation, up to LightSpeed size #20 and ProTaper shaping file S1. **D**, Further enlargement to LS size #30 and PT shaping file S2. **E**, Apical preparation to LS size #40 and PT finishing file F1. **F**, Additional enlargement to LS size #50 and PT finishing file F2. **G**, Superimposed μCT reconstructions comparing the initial canal geometry *(in green)* with the shape reached after use of the instruments shown in **F**. **H**, Final shape after step-back with LS instruments and PT finishing file F3. **I**, Superimposed μCT reconstructions comparing initial geometry and final shape. Note the slight ledge in the buccal canal after LS preparation and some straightening in the lingual canal after PT preparation.

while registering tactile feedback. The first instrument that experiences resistance 4 mm short of working length is the final apical size; it is then advanced to working length like the smaller instruments before. The next larger instrument is placed to 4 mm short of working length. This prepares the apical 5 mm for a matching SimpliFill obturator (SybronEndo). Midroot shaping is then accomplished with sequentially larger LSX instruments. Finally, the so-called master apical rotary, or MAR, is used to recapitulate to the working length.

All LightSpeed instruments are used in the following way: a slow, continuous apical movement is used until the blade binds; after a momentary pause, the blade is advanced further with intermittent ("pecking") motions.

Technique with the Self-Adjusting File

For this unusual file, a coronal flaring step—for example, with a G—precedes exploration with small K-files, including verification of patency and determining of working length. A glide path to a size #20 is considered appropriate; then the selected self-adjusting file is introduced into the canal with continuous irrigation through the center of the hollow file at a rate of approximately 5 ml/min. This maintains a continuous flow of active irrigant that carries with its outflow tissue debris and the dentin mud generated by the file. One file is used throughout the procedure. It is initially compressed into the root canal and gradually enlarges while cleaning and shaping the canal.

The file can rotate passively around its long axis and is guided by the operator to working length. The file is initially compressed into the root canal and gradually enlarges while cleaning and shaping the canal. The suggested time frame of instrumentation per canal dictates the final size and is approximately 3 to 4 minutes.[60] Obturation may commence with various techniques that allow plastifying gutta-percha (see Chapter 7).

Hybrid Techniques

For some time, combining various NiTi preparation systems have been suggested[89,545] to address certain shortcomings of current instruments (Box 6-4). Although many combinations are possible, the most popular and useful ones involve coronal preenlargement followed by different additional apical preparation sequences. However, clinicians must keep in mind that anatomic variations in each canal must be addressed individually with specific instrument sequences. Most important, oval canals extend deep into the apical area,[505,551,560] and apical foramina may in fact be oval in most cases.[70] Naturally, a rotating file can produce a round canal at best (Fig. 6-47); therefore, a strategy must be devised for adequately shaping oval canals without overly weakening radicular structure (compare Figs. 6-4 and 6-5). One hybrid approach completely prepared 95%

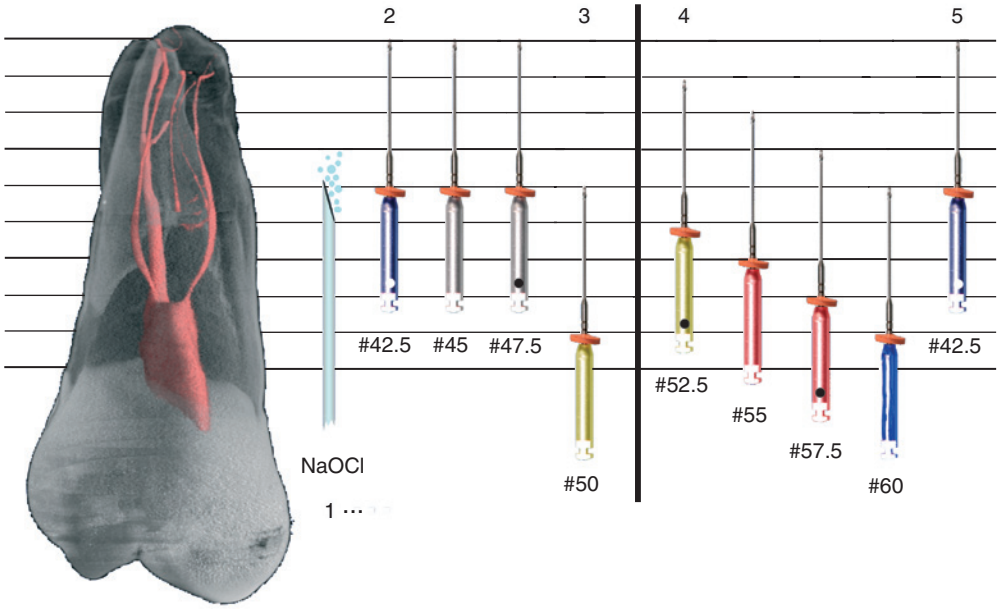


FIG. 6-46 Finishing of LightSpeed preparations to allow obturation. With the canal system flooded *(1)*, apical preparation *(2)* is continued until an LS instrument requires 12 pecks to reach the working length (WL). The next LS instrument *(3)* then is used to a point 4 mm short of the WL to prepare for LightSpeed's SimpliFill obturation system. Alternatively, canals may be flared for other root canal filling techniques by preparing with each subsequent instrument 1 mm shorter *(5)*.

BOX 6-4

Benefits of Using a Combination of Instruments for Endodontic Therapy

- Instruments can be used in a manner that promotes their individual strengths and avoids their weaknesses (most important).
- Hand instruments secure a patent glide path.
- Tapered rotary instruments efficiently enlarge coronal canal areas.
- Less tapered instruments allow additional apical enlargement.

or more of all such canals and resulted in extremely wide apical sizes that may be difficult to achieve with most instrument systems.[255,257,256]

Histologic slides (see Fig. 6-47) and µCT reconstructions show critical areas that were not mechanically prepared despite the use of various individual rotary techniques. The aim of hybridizing NiTi rotary techniques, therefore, is to increase apical size using a fast and safe clinical procedure.

Various clinicians have used this type of hybrid procedure in their practices (see Figs. 6-2 and 6-10). The principle involves the use of a variety of instruments: for example, GG drills and K-files for establishing straight-line access; ProTaper instruments for body shaping and apical preenlargement; NiTi K-files or LightSpeed instruments for apical widening; and various instruments for final smoothing.[545]

After a stainless steel file has confirmed a smooth glide path into the coronal two thirds, irrigation and mechanical preparation with a sequence of ProTaper shaping files opens and preenlarges the apical third (Fig. 6-48). Once the working length has been established, the apical third is flooded with

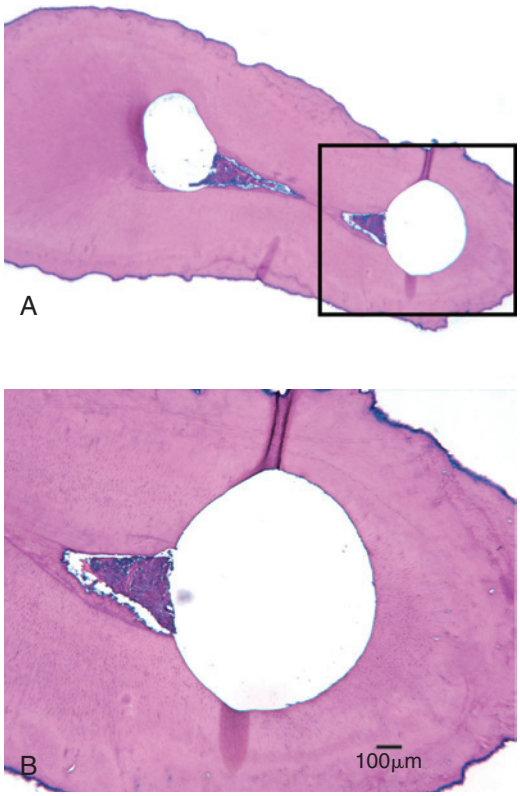


FIG. 6-47 Remaining potentially infected tissue in fins and isthmus configuration after preparation with rotary instruments. **A**, Cross section through a mesial root of a mandibular molar, middle to coronal third of the root. Both canals have been shaped; the left one is transported mesially (×10). **B**, Magnified view of rectangle in **A**. Note the presence of soft tissue in the isthmus area (×63). (Courtesy Professor H. Messer.)

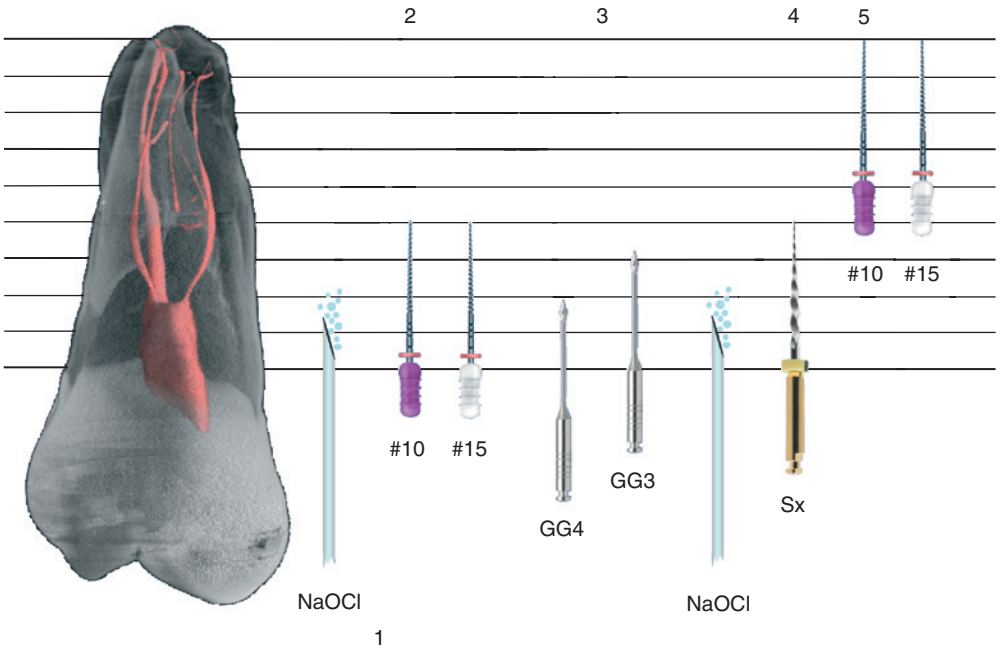


FIG. 6-48 Hybrid technique: Part I. After irrigation *(1)* and scouting *(2)*, GG drills *(3)* or ProTaper SX files *(4)* are used for coronal preenlargement and to secure straight-line access to the middle third. Precurved K-files are then used to explore and determine the working length *(5)*.

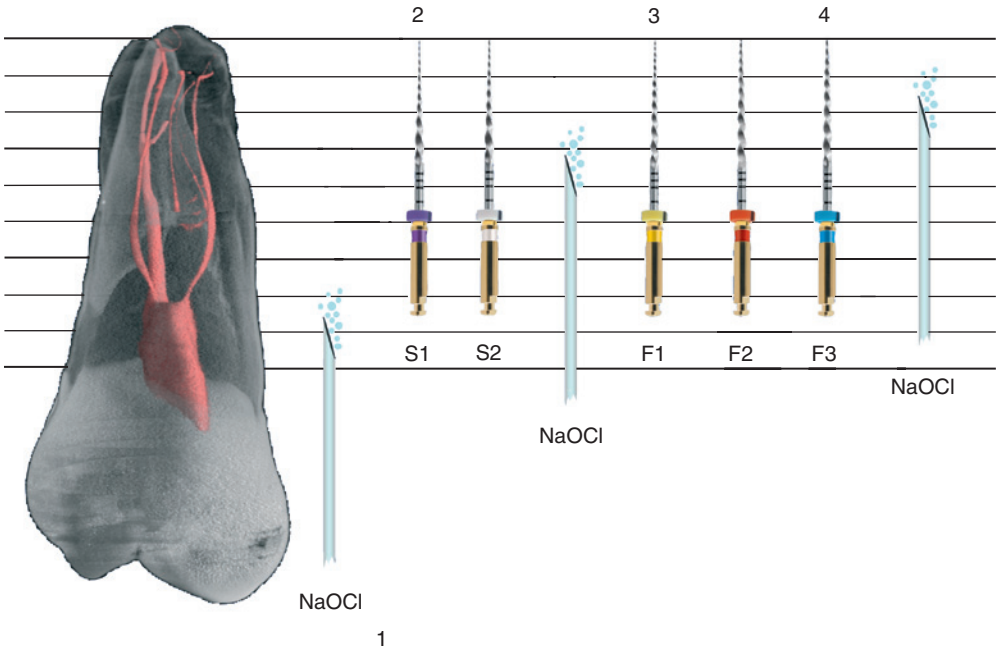


FIG. 6-49 Hybrid technique: Part II. In canal systems flooded with irrigant *(1)*, ProTaper shaping instruments S1 and S2 *(2)* and then finishing instruments F1 and F2 *(3)* are used to preenlarge the apical third, allowing irrigants access to the canals. Finishing instrument F3 may be used if feasible *(4)*.

NaOCl and further enlarged with ProTaper finishing files F1 and F2. The F3 ProTaper finishing file is relatively inflexible, and because of its side-cutting action, it should be used with caution in curved canals (Fig. 6-49). Further enlargement is possible with the F4 and F5 instruments, but these files may not be used in more acutely curved canals. The effectiveness of techniques combining different rotary instruments in enlarging canals recently was documented using superimposed root canal cross sections (Fig. 6-50). This method can help identify insufficiently prepared areas and weakening of the radicular structure.

A different approach, using, for example, NiTi K-files, .02 tapered rotaries (e.g., RaCe), or LightSpeed LSX (Fig. 6-51), may be also advantageous if larger sizes are desired. Finally, the overall shape may be smoothed with either engine-driven or handheld instruments. Handheld ProTaper or GT instruments may aid removal of acute apical curvatures or ledges and provide access to apical canal areas for irrigants.

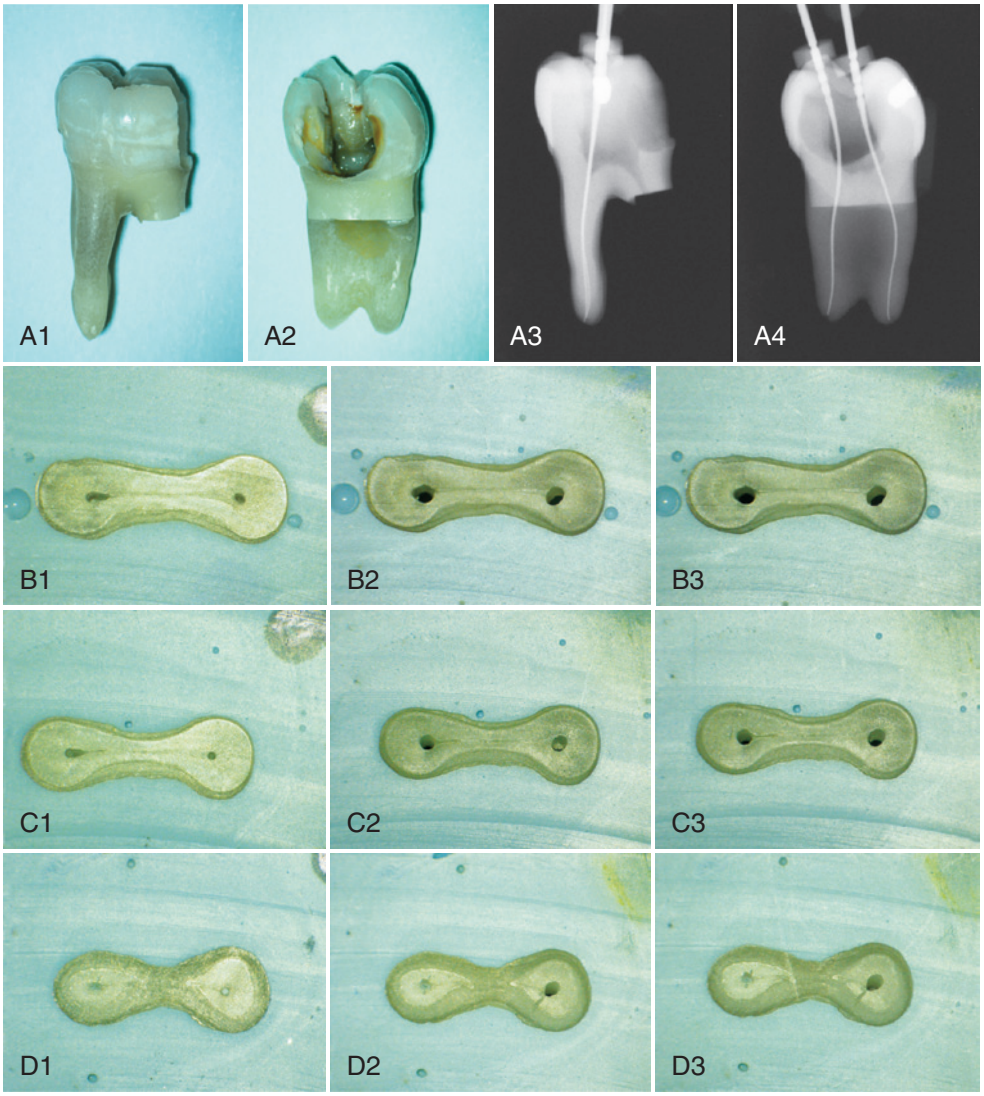


FIG. 6-50 Effect of a hybrid technique on root canal anatomy studied in a Bramante model. **A1-A4**, Both mesial canals of an extracted mandibular molar have been instrumented. Canal cross sections are shown before instrumentation (**B1-D1**). **B2-D2**, Cross sections after preenlargement with a ProTaper F3 file (*left canal*) and a size #45, #.02 taper instrument *(right canal)*. The final apical sizes were LightSpeed (LS) #50 and size #50, #.02 taper in the left and the right canal, respectively. (Courtesy Drs. S. Kuttler, M. Gerala, and R. Perez.)

Some hybrid systems seem to work better than others, but the deciding factors are likely the root canal anatomy and an adequate preparation goal.

Most cases requiring root canal therapy lend themselves to canal preparation with many different systems. Depending on the individual anatomy and the clinician's strategy, various sequences may be used. Mesiobuccal roots of the maxillary molar can show substantial curvature; rotary instrumentation or hybrid techniques allow preservation of the curvature and optimal enlargement. Occasionally hand instruments other than ISO-normed files are used in these cases to ensure a smooth, tapered shape or to eliminate ledges.

Final Apical Enlargement

Conceptually, file sizes during canal preparation were termed *initial apical file, master apical file,* and *final file* (or IAF, MAF, and FF, respectively). The first file to bind, the IAF, is supposed to give clinicians a guide in determining the final size of their canal preparation. On the other hand, many current instrumentation techniques have, due to their design, predefined final sizes—for example, WaveOne shaping aims at final sizes #25 .08 taper and #40, .08 taper.

Whichever apical size was conceptually aimed for, after shaping the overall canal to a tapered form, the apical size at that point should be assessed during apical gauging. K-files or K-Flexofiles are often recommended for this step; the files should be gently moved toward the working length (WL), and the binding point should be noted. The presence of an apical narrowing would translate into the fact that the WL may be reached without shaping effort with the desired size during gauging; however, the next larger size will stay back by a small distance. Finally, it should be verified that patency is maintained.

If apical gauging suggests that the canal is undershaped, additional enlargement is done with any technique appropriate to the specific anatomy of the root canal, often necessitating a hybrid technique.

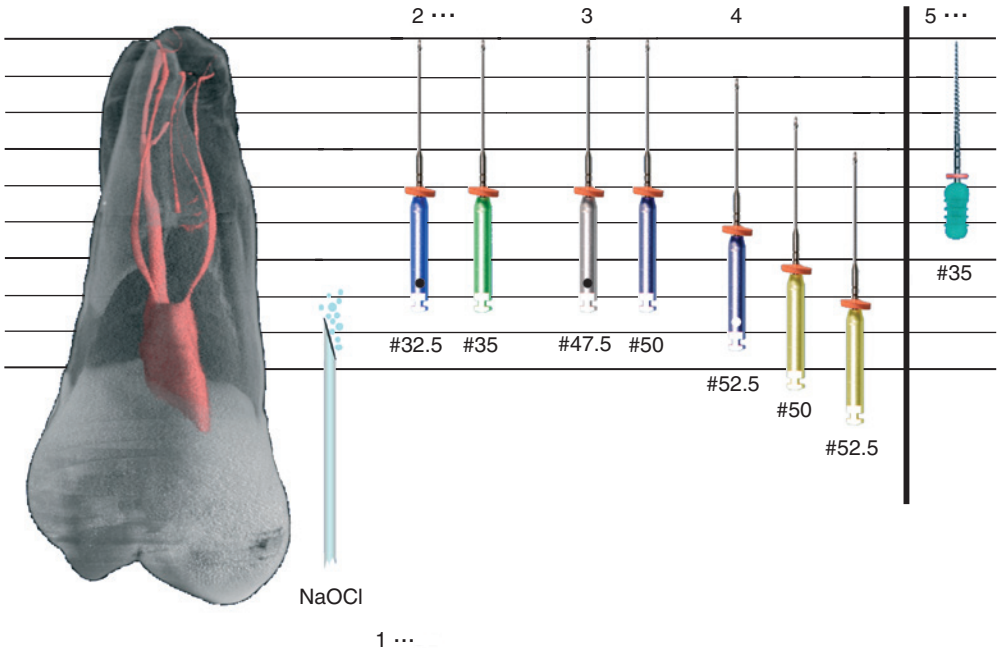


FIG. 6-51 Hybrid technique: Part III. Under irrigation *(1)*, LightSpeed instruments may be used to enlarge substantially (*2* and *3*) and to flare the apical section *(4)*. NiTi hand instruments *(5)* may be used similarly (see text for a more detailed explanation).

DISINFECTION OF THE ROOT CANAL SYSTEM

Hydrodynamics of Irrigation

Irrigation dynamics refers to how irrigants flow, penetrate, and readily exchange within the root canal system as well as the forces they produce. A better understanding of the fluid dynamics of different modes of irrigation will contribute to achieving predictable disinfection of the root canal system. Hence, in endodontic disinfection, the process of delivery is as important as the antibacterial characteristics of the irrigants.[65]

Irrigation is defined as "to wash out a body cavity or wound with water or a medicated fluid" and aspiration as "the process of removing fluids or gases from the body with a suction device." Disinfectant, meanwhile, is defined as "an agent that destroys or inhibits the activity of microorganisms that cause disease."[106]

The objectives of irrigation in endodontics are mechanical, chemical, and biologic. The mechanical and chemical objectives are as follows: (1) flush out debris, (2) lubricate the canal, (3) dissolve organic and inorganic tissue, and (4) prevent the formation of a smear layer during instrumentation or dissolve it once it has formed.[42] The mechanical effectiveness will depend on the ability of irrigation to generate optimum streaming forces within the entire root-canal system. The chemical effectiveness will depend on the concentration of the antimicrobial irrigant, the area of contact, and the duration of interaction between irrigant and infected material.[65] The final efficiency of endodontic disinfection will depend on its chemical and mechanical effectiveness.[190]

The biologic function of irrigants is related to their antimicrobial effects. In principle, irrigants should (1) have a high efficacy against anaerobic and facultative microorganisms in their planktonic state and in biofilms, (2) inactivate endotoxin, and (3) be nontoxic when they come in contact with vital tissues, and (4)b not cause an anaphylactic reaction.[42]

Efficiency of root canal irrigation in terms of debris removal and eradication of bacteria depends on several factors: penetration depth of the needle, diameter of the root canal, inner and outer diameter of the needle, irrigation pressure, viscosity of the irrigant, velocity of the irrigant at the needle tip, and type and orientation of the needle bevel (Fig. 6-52).

Penetration Depth of the Needle

The size and length of the irrigation needle—in relation to root canal dimensions—is of utmost importance for the effectiveness of irrigation.

Diameter of the Root Canal

The apical diameter of the canal has an impact on needle penetration depth (see earlier in this chapter for details regarding apical preparation size (see Fig. 6-7, Table 6-2).[72,578]

Inner and Outer Diameter of the Needle (Fig. 6-53, *A* and *B*)

The external needle diameter is of relevance for the depth of introduction into the root canal and for rigidity of the tip, an important consideration for irrigation of curved canals. Common 27 gauge injection needles have an external diameter of 0.42 mm, but smaller irrigation tips with external diameters of 0.32 mm (30 gauge) are available.[235] The Stropko Flexi-Tip (30 gauge) needle is fabricated from nickel-titanium to improve penetration into curved root canals.[205]

Irrigation Pressure

The internal diameter determines the pressure necessary for moving the syringe plunger. The speed of the plunger

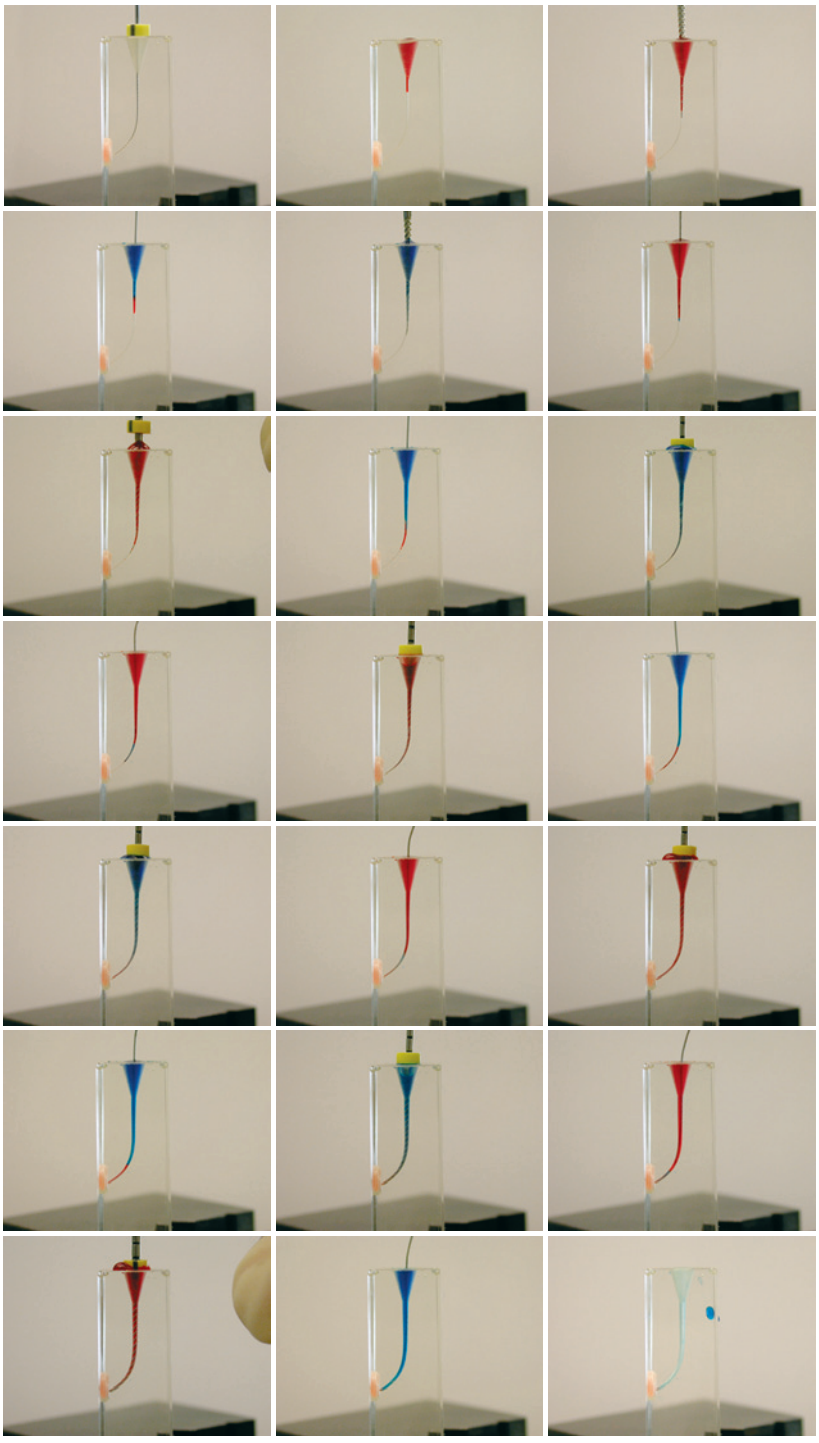


FIG. 6-52 Irrigation and movement of irrigants depends on canal shape. Sequential enlargement of a canal in clear plastic block was performed with a sequence of ProFile instruments in accordance with the manufacturer's recommendations. Alternating irrigation with blue and red fluid was done after each preparation step. Note the apical presence of irrigant after sufficient shape has been provided. Note the distribution of fluid immediately after irrigation with a 30-gauge needle.

determines the velocity with which the irrigant is extruded. Narrow needles require more pressure onto the plunger and extrude the irrigant with higher velocity than large needle sizes, which extrude greater amounts of irrigants but cannot be introduced as deep.

Type and Orientation of the Bevel of the Needle

To improve safety of irrigation and prevent extrusion of the irrigant through the apical foramen, some needles release the solution via lateral openings and have a closed, safe-ended tip. The orientation of the bevel is crucial to produce a turbulence effect on the dentinal wall of the canal. Side-vented and double side-vented needles lead to maximum shear stress concentrated on the wall facing the outlet (the proximal outlet for the double side-vented).[67]

Irrigants

An optimal irrigant would have all of the characteristics considered beneficial in endodontics but none of the negative or harmful properties. Presently, no solution can be regarded

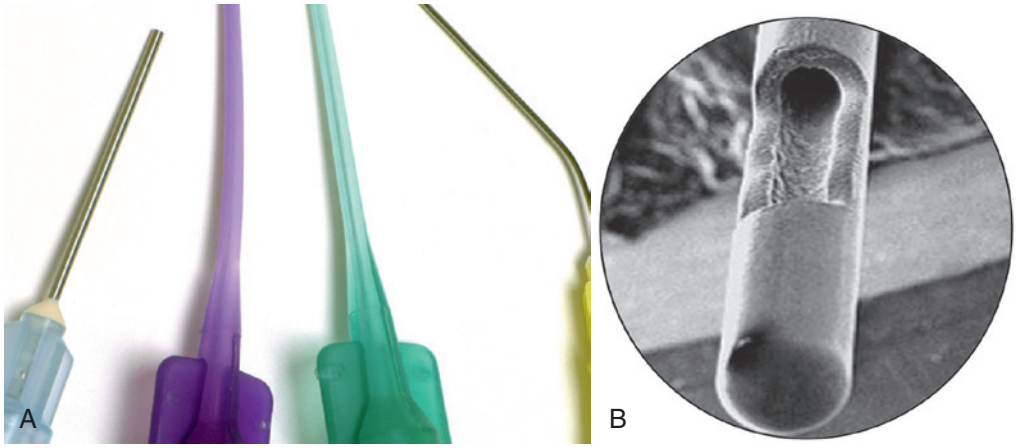


FIG. 6-53. A, Various types of needles for root canal irrigation. Shown are examples with open end and closed end, side vented. These are manufactured from plastic and stainless steel. B, SEM image of 30 gauge safety needle. (Courtesy Dr. F. Tay.)

BOX 6-5

Benefits of Using Irrigants in Root Canal Treatment

- Removal of particulate debris and wetting of the canal walls
- Destruction of microorganisms
- Dissolution of organic debris
- Opening of dentinal tubules by removal of the smear layer
- Disinfection and cleaning of areas inaccessible to endodontic instruments

BOX 6-6

Properties of an Ideal Irrigant for Root Canal Treatment

An ideal irrigant should do the following:

- Be an effective germicide and fungicide
- Be nonirritating to the periapical tissues
- Remain stable in solution
- Have a prolonged antimicrobial effect
- Be active in the presence of blood, serum, and protein derivatives of tissue
- Have low surface tension
- Not interfere with repair of periapical tissues
- Not stain tooth structure
- Be capable of inactivation in a culture medium
- Not induce a cell-mediated immune response
- Be able to completely remove the smear layer, and be able to disinfect the underlying dentin and its tubules
- Be nonantigenic, nontoxic, and noncarcinogenic to tissue cells surrounding the tooth
- Have no adverse effects on the physical properties of exposed dentin
- Have no adverse effects on the sealing ability of filling materials
- Have a convenient application
- Be relatively inexpensive

as optimal. However, combined use of selected irrigation products greatly contribute to successful outcome of treatment (Box 6-5 and Box 6-6).

Sodium Hypochlorite

NaOCl is the most commonly used irrigating solution[332] because of its an antibacterial capacity and the ability to dissolve necrotic tissue, vital pulp tissue, and the organic components of dentin and biofilms in a fast manner.[453]

NaOCl solution is frequently used as a disinfectant or a bleaching agent. It is the irrigant of choice in endodontics, owing to its efficacy against pathogenic organisms and pulp digestion, and satisfies most of the preferred characteristics stated earlier.[332]

History

Hypochlorite was first produced in 1789 in France. Hypochlorite solution was used as a hospital antiseptic that was sold under the trade names Eusol and Dakin's solution. Dakin recommended NaOCl as a buffered 0.5% solution for the irrigation of wounds during World War I.[114] Coolidge[108] later introduced NaOCl to endodontics an intracanal irrigation solution.[575]

Mode of Action

When sodium hypochlorite contacts tissue proteins, nitrogen, formaldehyde, and acetaldehyde are formed. Peptide links are fragmented and proteins disintegrate, permitting hydrogen in the amino groups (-NH-) to be replaced by chlorine (-NCl-) forming chloramines; this plays an important role for the antimicrobial effectiveness. Necrotic tissue and pus are dissolved and the antimicrobial agent can better reach and clean the infected areas.

In 2002 Estrela reported that sodium hypochlorite exhibits a dynamic balance (Fig. 6-54)[142]:

1. Saponification reaction: Sodium hypochlorite acts as an organic and fat solvent that degrades fatty acids and transforms them into fatty acid salts (soap) and glycerol (alcohol), reducing the surface tension of the remaining solution.
2. Neutralization reaction: Sodium hypochlorite neutralizes amino acids by forming water and salt. With the exit of hydroxyl ions, the pH is reduced.
3. Hypochlorous acid formation: When chlorine dissolves in water and it is in contact with organic matter, it forms

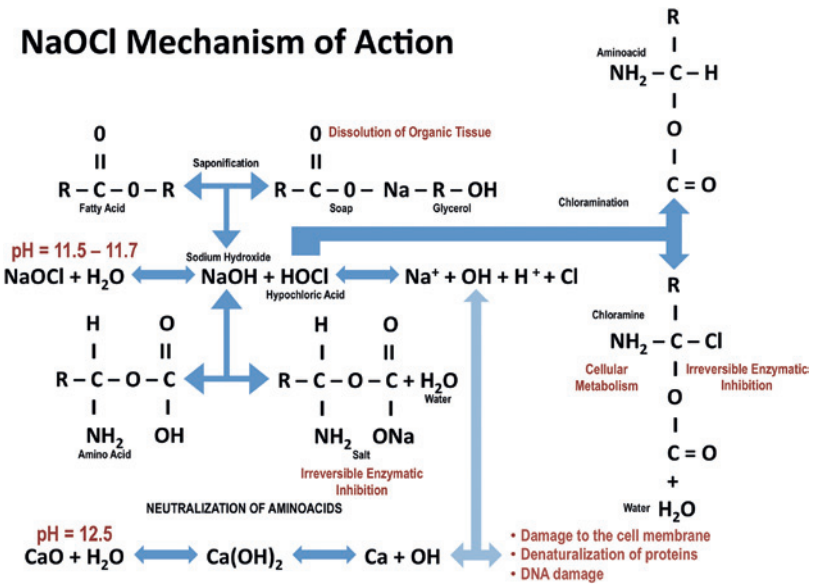


FIG. 6-54 Schematic diagram of the mechanism of action of NaOCl with the main interactions and properties highlighted. (Courtesy Dr. A. Manzur.)

hypochlorous acid. It is a weak acid with the chemical formula HClO that acts as an oxidizer. Hypochlorous acid ($HOCl^-$) and hypochlorite ions (OCl^-) lead to amino acid degradation and hydrolysis.

4. Solvent action: Sodium hypochlorite also acts as a solvent, releasing chlorine that combines with protein amino groups (NH) to form chloramines (chloramination reaction). Chloramines impede cell metabolism; chlorine is a strong oxidant and inhibits essential bacterial enzymes by irreversible oxidation of SH groups (sulfydryl group).[142]
5. High pH: Sodium hypochlorite is a strong base (pH > 11). The antimicrobial effectiveness of sodium hypochlorite, based on its high pH (hydroxyl ions action), is similar to the mechanism of action of calcium hydroxide. The high pH interferes in cytoplasmic membrane integrity due to irreversible enzymatic inhibition, biosynthetic alterations in cellular metabolism, and phospholipid degradation observed in lipidic peroxidation.[142]

Allergic Reactions to Sodium Hypochlorite

Although few reports have been published on allergic reactions to NaOCl,[144,198] real allergies to NaOCl are unlikely to occur, as both Na and Cl are essential elements in the physiology of the human body. It must be remembered that the hypochlorous acid (active component of the sodium hypochlorite) is a chemical substance that is elaborated by neutrophils in the process of phagocytosis; it may create local tissue damage when it is produced in excess (liquefaction necrosis: purulent exudate) but does not cause allergic responses. Nevertheless, hypersensitivity and contact dermatitis may occur in rare situations. A case report describes a serious chemical burn in an endodontist's eye caused by accidental contact with 3.5% NaOCl used as an irrigant during root canal.[403]

When hypersensitivity to NaOCl is suspected or confirmed, chlorhexidine should not be used either because of its chlorine content. For individuals who genetically have more possibilities than the normal population to generate allergies to multiple elements (allergies to food or to latex), a skin test

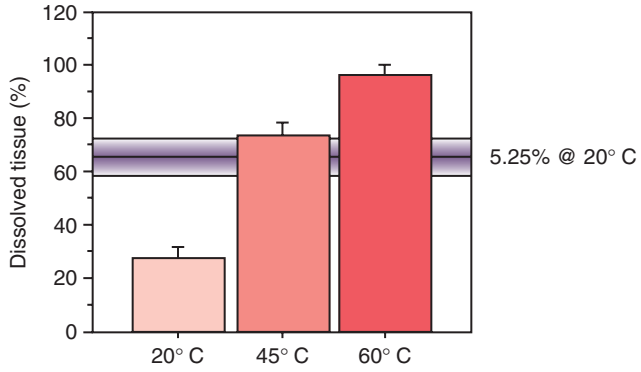


FIG. 6-55 Effect of heating on the ability of 0.5% sodium hypochlorite (NaOCl) to dissolve pulp tissue: NaOCl heated to 113° F (45° C) dissolved pulp tissue as well as the positive control (5.25% NaOCl) did. When the NaOCl was heated to 140° F (60° C), almost complete dissolution of tissue resulted. (Modified from Sirtes G, Waltimo T, Schaetzle M, Zehnder M: The effects of temperature on sodium hypochlorite short-term stability, pulp dissolution capacity, and antimicrobial efficacy, *J Endod* 31:669, 2005.)

for both NaOCl and CHX may be indicated. The use of an alternative irrigant with high antimicrobial efficacy, such as iodine potassium iodide, should be considered, assuming there is no known allergy to that irrigant. Solutions such as alcohol or tap water are less effective against microorganisms and do not dissolve vital or necrotic tissue; however, $Ca(OH)_2$ could be used as a temporary medicament because it dissolves both vital and necrotic tissue.[18,207]

Temperature

Increasing the temperature of low-concentration NaOCl solutions improves their immediate tissue-dissolution capacity (Fig. 6-55).[575] Furthermore, heated hypochlorite solutions remove organic debris from dentin shavings more efficiently. Bactericidal rates for NaOCl solutions, the capacity of human pulp dissolution, and increased efficacy were detailed in several

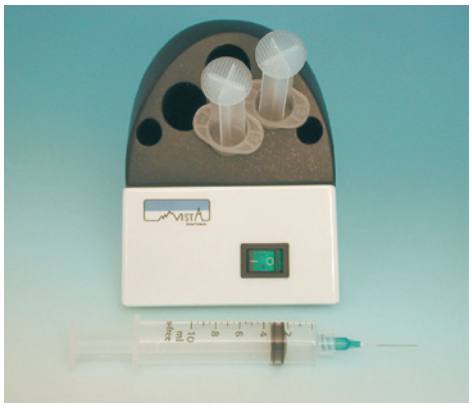


FIG. 6-56 Device for heating syringes filled with irrigation solution (e.g., sodium hypochlorite) before use. (Courtesy Vista Dental Products, Racine, WI.)

studies.[475] There are various devices to preheat NaOCl syringes (Fig. 6-56); however, it was demonstrated that as soon as the irrigant touches the root canal system, the temperature reaches the body temperature.[579] Therefore, some authors recommend in situ heating of NaOCl. This can be done by activating ultrasonic or sonic tips to the NaOCl inside the root canal for a couple of minutes (see irrigation timings, discussed later). Macedo and colleagues stated that the efficacy of NaOCl on dentin is improved by refreshment, ultrasonic activation, and exposure time.[307] In this investigation, a 10° C temperature rise during ultrasonic activation was insufficient to increase the reaction rate. However, no clinical studies are available at this point to support the use of heated NaOCl.[42,104]

Concentrations

NaOCl is used in concentrations between 0.5% and 6% for root canal irrigation. Controversy exists over recommended concentrations of sodium hypochlorite during root canal treatment. Some in vitro studies have shown that NaOCl in higher concentrations is more effective against *Enterococcus faecalis* and *Candida albicans*.[177,395,547] In contrast, clinical studies have indicated both low and high concentrations to be equally effective in reducing bacteria from the root canal system.[87,111] NaOCl in higher concentrations has a better tissue-dissolving ability.[204] However, in lower concentrations when used in high volumes it can be equally effective.[336,470] Higher concentrations of NaOCl are more toxic than lower concentrations.[491] However, due to the confined anatomy of the root canal system, higher concentrations have successfully been used during root canal treatment, with a low incidence of mishaps.

In summary, if lower concentrations are to be used for intracanal irrigation, it is recommended that the solution be used in higher volume and in more frequent intervals to compensate for the limitations in effectiveness.[470]

Instrumentation coupled with an antimicrobial irrigant, such as NaOCl, has been shown to yield more negative cultures than instrumentation alone.[86,323,364,372,463] However, even with the use of NaOCl, removal of bacteria from the root canal systems following instrumentation remains an elusive goal.

Grossman observed pulp tissue dissolution capacity and reported that 5% sodium hypochlorite dissolved this tissue in between 20 minutes and 2 hours.[185] The dissolution of bovine pulp tissue by sodium hypochlorite (0.5, 1.0, 2.5, and 5.0%) was studied in vitro under different conditions.[142] The study yielded the following conclusions:

1. The velocity of dissolution of the bovine pulp fragments was directly proportional to the concentration of the sodium hypochlorite solution and was greater without the surfactant;
2. variations in surface tension, from beginning to end of pulp dissolution, were directly proportional to the concentration of the sodium hypochlorite solution and greater in the solutions without surfactant. Solutions without surfactant presented a decrease in surface tension and those with surfactant an increase;
3. in heated sodium hypochlorite solutions, dissolution of the bovine pulp tissue was more rapid;
4. the greater the initial concentration of the sodium hypochlorite solutions, the smaller was the reduction of its pH.[142]

Time

There is conflicting evidence regarding the time course of the antibacterial effect of NaOCl.[42,195] In some articles hypochlorite is reported to kill the target microorganism in seconds, even at low concentrations, whereas other reports have published considerably longer times for the killing of the same species.[185] Such differences are likely a result of several factors: the presence of organic matter during experiments has a detrimental effect on the antibacterial activity of NaOCl. Haapasalo and colleagues showed that the presence of dentin caused marked delays in the killing of *Enterococus faecalis* by 1% NaOCl.[193] Morgental and colleagues reported similar findings.[337]

Many of the earlier studies were performed in the presence of an unknown amount of organic matter. When such confounding factors are eliminated, it has been shown that NaOCl kills the target microorganisms rapidly even at low concentrations of less than 0.1%.[195,539] However, in vivo the presence of organic matter (inflammatory exudate, tissue remnants, and microbial biomass) consumes NaOCl and weakens its effect. Therefore, continuous replenishing of irrigation solution and allowing sufficient contact time are important factors for the effectiveness of NaOCl.[195]

The chlorine ion, which is responsible for the dissolving and antibacterial capacity of NaOCl, is unstable and consumed rapidly during the first phase of tissue dissolution, probably within 2 minutes,[336] which provides another reason for continuous replenishment. This should especially be considered in view of the fact that rotary root canal preparation techniques have expedited the shaping process. The optimal time that a hypochlorite irrigant at a given concentration needs to remain in the canal system is an issue yet to be resolved.[575]

Toxicity

If inadvertently NaOCl is extruded through the apex, severe accidents may occur (Fig. 6-57, A-C). It is important to recognize the symptoms and act accordingly. After an accident with NaOCl, the following can be expected: severe pain, edema of neighboring soft tissues, possible extension of edema over the injured half of face and upper lip, profuse bleeding from root canal, profuse interstitial bleeding with hemorrhage of skin and mucosa (ecchymosis), chlorine taste and irritation of throat after injection into maxillary sinus, secondary infection possible, and reversible anesthesia or paresthesia is possible. To manage these lesions, the clinician should inform the patient and control pain with local anesthesia and analgesics.

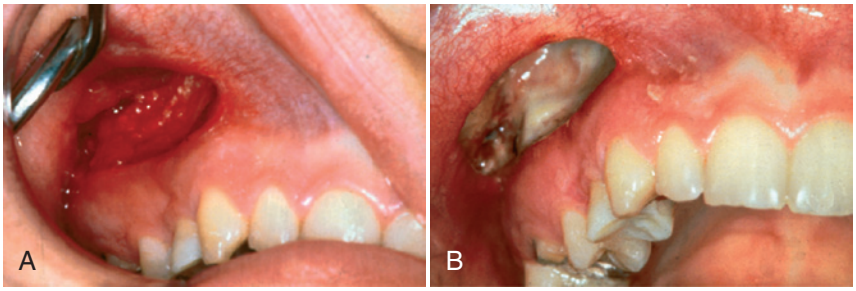


FIG. 6-57 Toxic effect of sodium hypochlorite on periradicular tissues. After root canal treatment of tooth #3, the patient reported pain. A, On a return visit, an abscess was diagnosed and incised. B, Osteonecrosis was evident after 3 weeks.

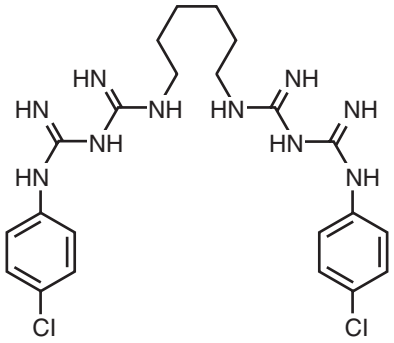


FIG. 6-58 Schematic drawing of the chlorhexidine molecule.

The application of extraoral cold compresses to reduce swelling is also effective. After 1 day, warm compresses and frequent warm mouth rinses for stimulation of local systemic circulation should be used. Patients should be recalled daily to monitor recovery. The use of antibiotics is not obligatory and is recommended only in cases of high risk or evidence of secondary infection. The administration of antihistamine is also not obligatory, and the use of corticosteroids is controversial. Further endodontic therapy with sterile saline or chlorhexidine as root canal irrigants and referral to a hospital in case of worsening symptoms were suggested.[260]

Chlorhexidine

History

Chlorhexidine (CHX) was developed in the UK and first marketed as an antiseptic cream.[148] It has been used for general disinfection purposes and the treatment of skin, eye, and throat infections in both humans and animals.[148,294] It has been used as an irrigant and medicament in endodontics for more than a decade.[309,333,363]

Molecular Structure

CHX is a strongly basic molecule with a pH between 5.5 and 7 that belongs to the polybiguanide group and consists of two symmetric four-chlorophenyl rings and two bisbiguanide groups connected by a central hexamethylene chain. CHX digluconate salt is easily soluble in water and very stable[184] (Fig. 6-58).

Mode of Action

Chlorhexidine, because of its cationic charges, is capable of electrostatically binding to the negatively charged surfaces of bacteria,[117] damaging the outer layers of the cell wall and rendering it permeable.[214,229,228] CHX is a wide-spectrum antimicrobial agent, active against gram-positive, gram-negative bacteria and yeasts.[129]

Depending on its concentration, CHX can have both bacteriostatic and bactericidal effects. At high concentrations, CHX acts as a detergent; and exerts its bactericidal effect by damaging the cell membrane and causes precipitation of the cytoplasm. At low concentrations, CHX is bacteriostatic, causing low molecular-weight substances (i.e., potassium and phosphorus) to leak out without the cell being permanently damaged.[43]

Substantivity

Because of the cationic nature of the CHX molecule, it can be absorbed by anionic substrates such as the oral mucosa and tooth structure.[308,312,414,527] CHX is readily adsorbed onto hydroxyapatite and teeth. Studies have shown that the uptake of CHX onto teeth is reversible.[222] This reversible reaction of uptake and release of CHX leads to substantive antimicrobial activity and is referred to as *substantivity*. This effect depends on the concentration of CHX. At low concentrations of 0.005% to 0.01%, only a constant monolayer of CHX is adsorbed on the tooth surface, but at higher concentrations (>0.02%), a multilayer of CHX is formed on the surface, providing a reservoir of CHX that can rapidly release the excess into the environment as the concentration of CHX in the surrounding environment decreases.[139] Time and concentration of CHX can influence the antibacterial substantivity, and the conclusions are inconsistent. Some studies demonstrated that 4% CHX has greater antibacterial substantivity than 0.2% after a 5-minute application.[333] Other studies stated that CHX should be left for more than 1 hour in the canal to be adsorbed by the dentin.[291] Komorowski and colleagues suggested that a 5-minute application of CHX did not induce substantivity, so dentin should be treated with CHX for 7 days.[265] However, when Paquette and Malkhassian,[364] in their in vivo study, medicated the canals with either liquid or gel forms of CHX for 1 week, neither of them could achieve total disinfection. Therefore, residual antimicrobial efficacy of CHX in vivo still remains to be demonstrated.

Cytotoxicity

CHX is normally used at concentrations between 0.12% and 2%. Löe and colleagues[294] demonstrated that at these concentrations, CHX has a low level of tissue toxicity, both locally and systemically. When 2% CHX was used as a subgingival irrigant, no apparent toxicity was noted on gingival tissues.[295,488] Additionally, CHX rinses have been suggested to promote healing

of periodontal wounds after surgery.[24] These reports were the basis of many studies that assumed that CHX will be tolerated on periapical tissues with a similar response than in the gingival tissues.[245] When compared CHX and NaOCl into subcutaneous tissues of guinea pigs and rats, an inflammatory reaction developed; however, the toxic reaction from CHX was less than that of NaOCl.[352,571] Moreover, a reduced incidence of alveolar osteitis was found when CHX was applied as a rinse in the extraction sites of the third molars on the day of surgery and several days after.[91] Allergic and anaphylactic reactions to CHX were reported in only a few articles.[166,351] It is important to mention that patients that are allergic to NaOCl may be also allergic to CHX.

On the other hand, some controversial results were found. Some studies demonstrated that CHX is cytotoxic to some lines of cultured human skin fibroblasts.[219] It has been reported that CHX has a higher cytotoxicity profile than povidone iodine when studied in osteoblastic human alveolar bone.[88] Also, when CHX was injected in the hind paw of mice, it could induce severe toxic reactions.[149]

Finally, when CHX is mixed with NaOCl, parachloroaniline (PCA) is formed.[46] The toxicity level of CHX on periapical tissues when applied in the root canals, especially with other irrigants, merits further investigation.

Chlorhexidine as an Endodontic Irrigant

CHX has been extensively studied as an endodontic irrigant and intracanal medication, both in vivo[35,292,311,364,564] and in vitro.[41,44,45,141,245,265,273,281,282,469,471,506,540,574]

The antibacterial efficacy of CHX as an irrigant is concentration dependent. It has been demonstrated that 2% CHX has a better antibacterial efficacy than 0.12% CHX in vitro.[45] When comparing with the effectiveness with NaOCl, controversial results can be found. NaOCl has an obvious advantage over CHX with the dissolution capacity of organic matter that CHX lacks; therefore, even though in vitro studies suggest some advantages with the use of CHX, as soon as organic and dental tissue is added, NaOCl is clearly preferable.

The antibacterial effectiveness of CHX in infected root canals has been investigated in several in vivo studies. Investigators reported that 2.5% NaOCl was significantly more effective than 0.2% CHX when the infected root canals were irrigated for 30 minutes with either of the solutions.[409]

In a controlled and randomized clinical trial, the efficacy of 2% CHX liquid was tested against saline using culture technique. All the teeth were initially instrumented and irrigated using 1% NaOCl. Then either 2% CHX liquid or saline was applied as a final rinse. The authors reported a further reduction in the proportion of positive cultures in the CHX group. Their results showed a better disinfection of the root canals using CHX compared to saline as a final rinse.[574]

The antibacterial efficacy of 2% CHX gel was tested against 2.5% NaOCl in teeth with apical periodontitis, with the bacterial load assessed using a real-time quantitative polymerase chain reaction (RTQ-PCR) and colony forming units (CFUs). The bacterial reduction in the NaOCl group was significantly greater than that for the CHX group when measured by RTQ-PCR. Based on culture technique, bacterial growth was detected in 50% of the CHX group cases compared to 25% in the NaOCl group.[540] On the other hand, another study based on this culture technique revealed no significant difference between the antibacterial efficacy of 2.5% NaOCl and 0.12% CHX liquid when used as irrigants during the treatment of infected canals.[469,471]

In a systematic review, Ng and colleagues demonstrated that abstaining from using 2% CHX as an adjunct irrigant to NaOCl was associated with superior periapical healing.[345] Unlike NaOCl, CHX lacks a tissue-dissolving property. Therefore, NaOCl is still considered the primary irrigating solution in endodontics.

Chlorhexidine as an Intracanal Medication

CHX as intracanal medication has been the focus of many in vitro[38,128,265,281,468] CHX used as intracanal medicament has at least as good or even better antimicrobial efficacy than $Ca(OH)_2$.[468] and it was shown to be very effective in eliminating a biofilm of *E. faecalis*.[288]

When in vivo studies are analyzed,[35,115,123,140,292,311,364,580] some controversial results were found. On one hand, CHX, inhibits experimentally induced inflammatory external root resorption when applied for 4 weeks.[292] In infected root canals, it was shown to reduce bacteria as effectively as $Ca(OH)_2$ when applied for 1 week.[34] Because of its substantivity, CHX has the potential to prevent bacterial colonization of root canal walls for prolonged periods of time.[245,265] It was demonstrated that its effect depended on the concentration of CHX but not on its mode of application, which may be as a liquid or gel.[44]

In vivo, results can be different; one of the reasons for this is that researchers[193] developed an experimental model using dentin powder particles to investigate the possible inactivation of some antibacterial medicaments when they come in contact with dentin. They showed that dentin powder had inhibitory effects on all medicaments tested. The effect was dependent on the concentration of the medicament and the duration of contact. The effect of $Ca(OH)_2$ was totally abolished by the presence of dentin powder. The effect of 0.05% CHX and 1% NaOCl was reduced but not totally eliminated by the presence of dentin. No inhibition could be measured when full-strength solutions of CHX and IKI were used.

An in vivo investigation assessed the antibacterial efficacy of three different intracanal medications: camphorated paramonochlorophenol, $Ca(OH)_2$, and 0.12% CHX liquid by applying them for 1 week in single-rooted teeth of patients with apical periodontitis. The proportions of positive cultures were not significantly different among the tested medications, but they were slightly lower in teeth medicated with CHX (0.12%) liquid than those medicated with camphorated paramonochlorophenol or $Ca(OH)_2$.[35] Another in vivo study evaluated antibacterial effectiveness of 2% CHX liquid as an intracanal medication in teeth with apical periodontitis. The authors concluded that a moderate increase in bacterial counts during a medication period of 7 to 14 days that was similar to outcomes seen and reported for $Ca(OH)_2$ by Peters and colleagues.[364, 372] On the other hand, an alternative investigation[311] demonstrated no significant differences among the medication groups. Intracanal medication with $Ca(OH)_2$, 2% CHX gel, or a mixture of $Ca(OH)_2$/CHX applied for 7 days did not reduce the bacterial concentration beyond what was achieved after chemomechanical preparation using 1% NaOCl. Other research showed that a final rinse with MTAD and medication with 2%CHX gel, did not reduce bacterial counts beyond levels achieved by a chemomechanical preparation using NaOCl.[310]

Chlorhexidine Mixed with Calcium Hydroxide

To enhance the properties of both CHX and $Ca(OH)_2$, their combination was analyzed in several in vitro and in vivo studies. The high pH of $Ca(OH)_2$ was unaffected when combined with CHX.[46] However, the results have not been conclusive. Some in vitro studies have reported an improved antibacterial action when both agents were combined,[45,145,575] whereas other studies reported contradictory results.[199]

The combination of CHX and $Ca(OH)_2$ showed good antimicrobial properties when tested in animal studies.[480] When studied in vivo in patients with apical periodontitis, the results showed similar antibacterial efficacy of each medicament alone or in combination.[311] In another clinical study, when 0.12% CHX was used during cleaning and shaping and an intracanal medication with $Ca(OH)_2$/0.12% CHX was left in the canals for 7 days, it was found that using 0.12% CHX solution as an irrigant significantly reduced the number of intracanal bacteria, but it failed to render the canals bacteria free.[465] Therefore, it seems that the usefulness of mixing $Ca(OH)_2$ with CHX remains controversial.

Chlorhexidine and Coronal Penetration of Bacteria

Several studies analyzed the property of antibacterial substantivity and bacterial penetration.[178] It was demonstrated that placement of intracanal medication delays bacterial penetration because of the physical barrier alongside the antibacterial action of the medicament. Gomes and colleagues,[178] in a laboratory study, investigated the time required for recontamination of the root canal system of teeth with coronal restorations medicated with $Ca(OH)_2$, 2% CHX gel, or a combination of both and concluded that if medication is present, retardation of microorganism invasion was seen. Overall, because of its substantivity, CHX as an intracanal medicament/irrigant may delay the coronal recontamination of the root canal system, but more in vivo studies are needed to corroborate these results.

Interaction between CHX, NaOCl, and EDTA

NaOCl and CHX when in contact produce a change of color and a precipitate (Fig. 6-59, *A* and *B*). The reaction is dependent of the concentration of NaOCl. The higher the concentration of NaOCl, the more precipitate is generated in the presence of 2% CHX.[46] Furthermore, concerns have been raised that the color change may have some clinical relevance because of staining, and the resulting precipitate might interfere with the seal of the root obturation. Basrani and colleagues evaluated the chemical nature of this precipitate and reported the formation of 4-chloroaniline (PCA).[46] Furthermore, a study using time-of-flight secondary ion mass spectrometry (TOF-SIMS) analysis shows the penetration of PCA inside dentinal tubules. PCA has been shown to be toxic in humans with short-term exposure, resulting in cyanosis, which is a manifestation of methemoglobin formation. The combination of NaOCl and CHX causes color changes and formation of a possibly toxic insoluble precipitate that may interfere with the seal of the root obturation. Alternatively, the canal can be dried using paper points before the final CHX rinse.[575]

The combination of CHX and EDTA produces a white precipitate, so a group of investigators [399] did a study to determine whether the precipitate involves the chemical degradation of CHX. The precipitate was produced and redissolved in a known amount of dilute trifluoroacetic acid. Based on the results, CHX was found to form a salt with EDTA rather than undergoing a chemical reaction.

Chlorhexidine and Dentin Bonding

Researchers evaluated the effect of CHX on resin-dentin bond stability ex vivo.[90] They concluded that autodegradation of collagen matrices can occur in resin-infiltrated dentin, but this may be prevented by the application of a synthetic protease inhibitor such as CHX.[90] Because of its broad-spectrum matrix metalloproteinase (MMP)-inhibitory effect, CHX may significantly improve resin-dentin bond stability.[90]

Allergic Reactions to Chlorhexidine

Allergic responses to CHX are rare, and there are no reports of reactions following root canal irrigation with CHX.[27,235] Several studies have reported the sensitization rate to be approximately 2%.[268] However, some allergic reactions such as anaphylaxis, contact dermatitis, and urticaria have been reported following direct contact to mucosal tissue or open wounds.[134,388,451,479]

Decalcifying Agents

Debris is defined as dentin chips or residual vital or necrotic pulp tissue attached to the root canal wall. Smear layer was defined by the American Association of Endodontists in 2003 as a surface film of debris retained on dentin or another surface after instrumentation with either rotary instruments or endodontic files; it consists of dentin particles, remnants of vital or necrotic pulp tissue, bacterial components, and retained irrigants. Although it has been viewed as an impediment to irrigant penetration into dentinal tubules (Fig. 6-60), there is

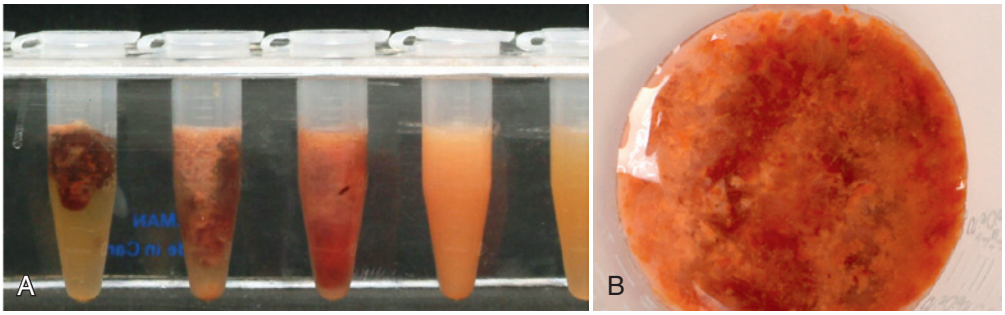


FIG. 6-59 Red precipitate forming after contact between NaOCl and chlorhexidine. **A**, When 2% CHX is mixed with different concentration of NaOCl, a change of color and precipitate occurs. The higher the concentration of NaOCl, the larger is the precipitate formation. **B**, Detail of the interaction between 2% CHX and 5%NaOCl.

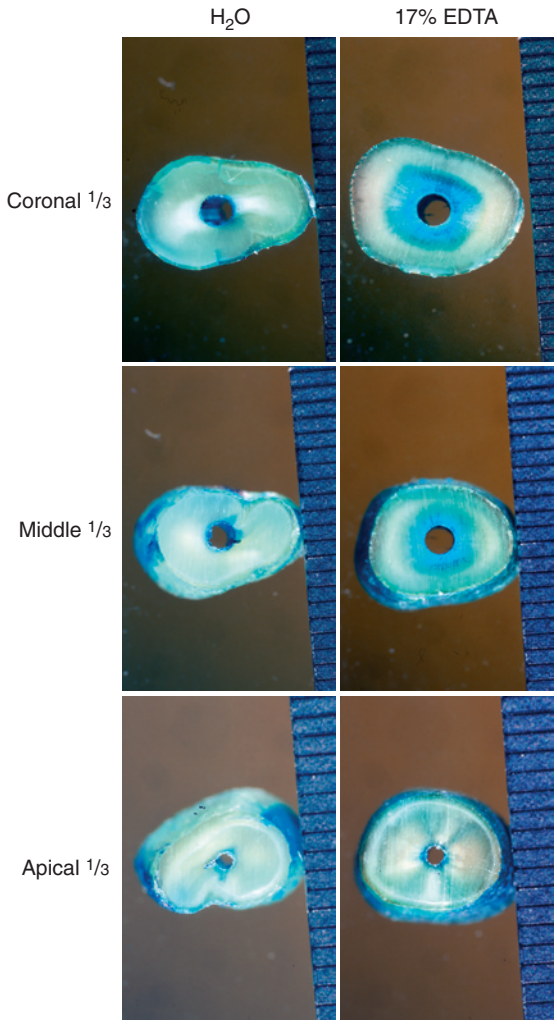


FIG. 6-60 Penetration of irrigants into dentinal tubules after root canal preparation with different dentin pretreatments. *Left column,* Irrigation with tap water and then with blue dye. *Right column,* Smear layer is removed with 17% EDTA, applied in high volume and with a 30-gauge needle, followed by irrigation with blue dye. Note the comparable diffusion of dye in the apical sections, whereas dye penetrated deeper into the dentin in the two coronal sections.

still a controversy about the influence of smear layer on the outcome of endodontic treatment. Some researchers emphasize the importance on removing the smear layer to allow irrigants, medications, and sealers to penetrate dentinal tubules and improve disinfection. On the other hand, other researchers focused on keeping the smear layer as a protection for bacterial invasion, apical and coronal micro leakage, bacterial penetration of the tubules, and the adaptation of root canal materials. The majority of the conclusions on smear layer are based on in vitro studies. A clinical study by Ng and colleagues[345] found that the use of EDTA significantly increased the odds of success of retreatment cases twofold.

Ethylenediamine Tetra-Acetic Acid

Ethylenediamine tetra-acetic acid, widely abbreviated as EDTA, is an aminopolycarboxylic acid and a colorless, water-soluble solid EDTA is often suggested as an irrigation solution because it can chelate and remove the mineralized portion of the smear layer (Fig. 6-61). It is a polyaminocarboxylic acid with the formula $[CH_2N(CH_2CO_2H)_2]_2$. Its prominence as a chelating agent arises from its ability to sequester di- and tricationic metal ions such as Ca^{2+} and Fe^{3+}. After being bound by EDTA, metal ions remain in solution but exhibit diminished reactivity.[494]

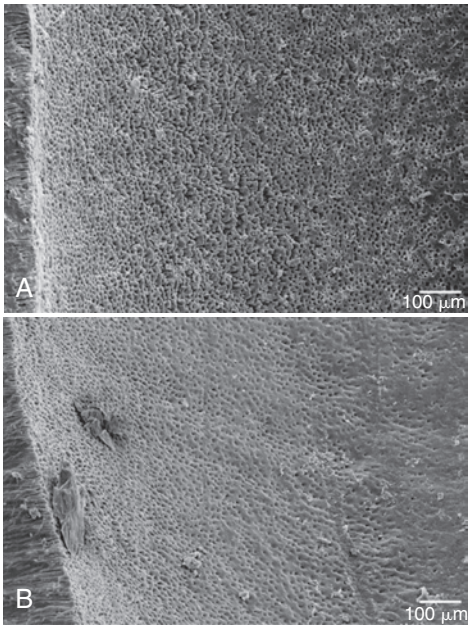


FIG. 6-61 Example of canals with minimal smear layer. **A**, Middle third after irrigation with 17% ethylenediaminetetraacetic acid (EDTA) and 2.5% sodium hypochlorite (NaOCl). **B**, Apical third with some particulate debris.

History

The compound was first described in 1935 by Ferdinand Munz, who prepared the compound from ethylenediamine and chloroacetic acid. Chelating agents were introduced into endodontics as an aid for the preparation of narrow and calcified root canals in 1957 by Nygaard-Østby.[232] Today, EDTA is mainly synthesized from ethylenediamine (1,2-diaminoethane), formaldehyde (methanal), and sodium cyanide.[232,494]

Mode of Action

On direct exposure for extended time, EDTA extracts bacterial surface proteins by combining with metal ions from the cell envelope, which can eventually lead to bacterial death.[232] Chelators such as EDTA form a stable complex with calcium. When all available ions have been bound, equilibrium is formed and no further dissolution takes place; therefore, EDTA is self-limiting.[232]

Applications in Endodontics

EDTA alone normally cannot remove the smear layer effectively; a proteolytic component, such as NaOCl, must be added to remove the organic components of the smear layer.[176] For root canal preparation, EDTA has limited value alone as an irrigation fluid.[176] EDTA is normally used in a concentration of 17% and can remove the smear layers when in direct contact with the root canal wall for less than 1 minute. Even though EDTA has self-limited action, if it is left in the canal for longer or NaOCl is used after EDTA, erosion of dentin has been demonstrated (Fig. 6-62).

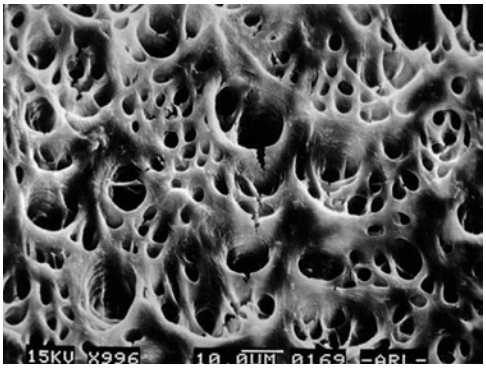


FIG. 6-62 Scanning electron microscope (SEM) image of root canal dentin exposed to 2 minutes of EDTA.

FIG 6-63 Container with BioPure MTAD. (Courtesy Dentsply Tulsa Dental Specialties, Tulsa, OK.)

Although citric acid appears to be slightly more potent at similar concentration than EDTA, both agents show high efficiency in removing the smear layer. In addition to their cleaning ability, chelators may detach biofilms adhering to root canal walls.[188] This may explain why an EDTA irrigant proved to be highly superior to saline in reducing intracanal microbiota despite the fact that its antiseptic capacity is relatively limited.[188] Antiseptics such as quaternary ammonium compounds (EDTAC) or tetracycline antibiotics (MTAD) (see the discussion on the combination of irrigants, presented later) have been added to EDTA and citric acid irrigants, respectively, to increase their antimicrobial capacity. The clinical value of this, however, is questionable. EDTAC shows similar smear-removing efficacy as EDTA, but it is more caustic.

The effect of chelators in negotiating narrow, tortuous, calcified canals to establish patency depends on both canal width and the amount of active substance available, since the demineralization process continues until all chelators have formed complexes with calcium.[232,452,577] Therefore, studies should be read with cautious because one study can show demineralization up to a depth of 50 μ m into dentin,236 but other reports demonstrated significant erosion after irrigation with EDTA.[519]

A comparison of bacterial growth inhibition showed that the antibacterial effects of EDTA were stronger than citric acid and 0.5% NaOCl but weaker than 2.5% NaOCl and 0.2% CHX.[467] EDTA had a significantly better antimicrobial effect than saline solution. It exerts it strongest effect when used synergistically with NaOCl, although no disinfecting effect on colonized dentin could be demonstrated.[212]

Interaction of EDTA and NaOCl

Investigators studied the interactions of EDTA with NaOCl.[183] They concluded that EDTA retained its calcium-complex ability when mixed with NaOCl, but EDTA caused NaOCl to lose its tissue-dissolving capacity, with virtually no free chlorine detected in the combinations. Clinically, this suggests that EDTA and NaOCl should be used separately. In an alternating irrigating regimen, copious amounts of NaOCl should be administered to wash out remnants of the EDTA. In modern endodontics, EDTA is used once the cleaning and shaping is completed for around 1 minute. It can be activated with ultrasonic activation for better penetration in dentinal tubules. It should be taken into consideration that an increase in the temperature of EDTA is not desirable. Chelators have a temperature range within which they work best. When EDTA is heated from 20° to 90°, the calcium binding capacity decreases.[576]

HBPT

HEBP (1-hydroxyethylidene-1, 1-bisphosphonate; also called etidronic acid) is a weak chelator.[575] It is a potential alternative to EDTA because it has no short-term reactivity with NaOCl. It can be used in combination with NaOCl without affecting its proteolytic or antimicrobial properties.[575] It is nontoxic,[26,125] and it is used in medicine to treat bone diseases.[362]

Combination of Irrigants and Added Detergent

Surface Tension

Irrigants with low surface tension have better wettabilty, and it is assumed that they may penetrate better in dentinal tubules and anatomic irregularities. Detergents (e.g., Tween 80) are added to irrigants to lower their surface tension. However, Boutsioukis and Kishen did not find support for this rationale,[65] perhaps because the effect of surface tension is important only at the interface between two immiscible fluids (e.g., between irrigant and an air bubble, but not between irrigant and dentinal fluid). Studies have also confirmed that surfactants do not enhance the ability of NaOCl to dissolve pulp tissue[251] or the efficacy of common chelators to remove calcium or smear layer.[65,125,300,577]

BioPure MTAD and Tetraclean

Two new irrigants based on a mixture of antibiotics, citric acid, and a detergent have been developed. These irrigants are capable of removing both the smear layer and organic tissue from the infected the root canal system.[520] MTAD (Fig. 6-63), introduced by Torabinejad and Johnson at Loma Linda University in 2003,[520] is an aqueous solution of 3% doxycycline, a broad-spectrum antibiotic; 4.25% citric acid, a demineralizing agent; and 0.5% polysorbate 80 detergent (Tween 80).[520] It is mixed as a liquid and powder prior to use. MTAD has been recommended in clinical practice as a final rinse after completion of conventional chemomechanical preparation.[48,248,457,458,520,521]

Tetraclean (Ogna Laboratori Farmaceutici, Muggio, Italy) is a combination product similar to MTAD. The two irrigants differ in the concentration of antibiotics (doxycycline 150 mg/5 ml for MTAD and 50 mg/5 ml for Tetraclean) and the kind of detergent (Tween 80 for MTAD, polypropylene glycol for Tetraclean).

Mode of Action

All tetracyclines are derivatives of four ringed nuclei that differ structurally in regard to the chemical groups at the 2, 5, 6, and

7 positions. These derivatives exhibit different characteristics such as absorption, protein binding, metabolism, excretion, and degree of activity against susceptible organism.[210] Tetracyclines inhibit protein synthesis by reversibly binding to the 30S subunit of bacterial ribosome in susceptible bacteria. It is effective against *Aa. capnocytophaga*, *P. gingivalis*, and *P. intermedia* and affects both gram-positive and gram-negative (more gram-negative effect) types. Tetracycline is a bacteriostatic antibiotic, but in high concentrations, tetracycline may also have a bactericidal effect. Doxycycline, citric acid, and Tween 80 together may have a synergistic effect on the disruption of the bacterial cell wall and on the cytoplasmic membrane.

Smear Layer Removal

In two studies, the efficacy of MTAD or EDTA in the removal of the smear layer was confirmed, but no significant difference between these two solutions was reported.[507,508]

Antibacterial Efficacy

Earlier in vitro research on MTAD showed its antimicrobial efficacy over conventional irrigants.[118,267,507,508,521] Torabinejad and colleagues found that MTAD was effective in killing *E. faecalis* up to 200× dilution.[117] Shabahang and Torabinejad showed that the combination of 1.3% NaOCl as a root canal irrigant and MTAD as a final rinse was significantly more effective against *E. faecalis* than the other regimens.[458]A study using extracted human teeth contaminated with saliva showed that MTAD was more effective than 5.25% NaOCl in disinfection of the teeth. In contrast to the previously mentioned studies, later research suggested less than optimal antimicrobial activity of MTAD.[170,248,258] Krause and colleagues,[267] using bovine tooth sections, showed that 5.25% NaOCl was more effective than MTAD in disinfection of dentin disks inoculated with *E. faecalis*.[458,468]

Clinical Trials

Malkhassain and colleagues, in a clinical controlled trial of 30 patients, reported that the final rinse with MTAD did not reduce the bacterial counts in infected canals beyond levels achieved by a chemomechanical preparation using NaOCl alone.[310]

Protocol for Use

MTAD was developed as a final rinse to disinfect the root canal system and remove the smear layer. The effectiveness of MTAD to completely remove the smear layer is enhanced when a low concentration of NaOCl (1.3%) is used as an intracanal irrigant before placing 1 ml of MTAD in a canal for 5 minutes and rinsing it with an additional 4 ml of MTAD as the final rinse.[457]

QMiX

QMiX was introduced in 2011; it is one of the new combination products introduced for root canal irrigation (Fig. 6-64). It is recommended to be used at the end of instrumentation, after NaOCl irrigation. According to the patent[194] QMix contains a CHX-analog, Triclosan (N-cetyl-N,N,N-trimethylammonium bromide), and EDTA as a decalcifying agent; it is intended as antimicrobial irrigant as well as an agent to remove canal wall smear layers and debris.

Protocol

QMiX is suggested as a final rinse. If sodium hypochlorite was used throughout the cleaning and shaping, saline can rinse out NaOCl to prevent the formation of PCA.

FIG 6-64 QMiX 2in1. Combination of C, EDTA and detergent. QMiX irrigating solution is a single solution used as a final rinse after bleach for one-step smear layer removal and disinfection. (Courtesy Dentsply Tulsa Dental Specialties, Tulsa, OK.)

Smear Layer Removal

Stojic and colleagues investigated the effectiveness of smear layer removal by QMiX using scanning electron microscopy.[500] QMiX removed smear layer equally well as EDTA. Dai and colleagues examined the ability of two pH versions of QMiX to remove canal wall smear layers and debris using an open canal design.[113] Within the limitations of an open-canal design, the two experimental QMiX versions are as effective as 17% EDTA in removing canal wall smear layers after the use of 5.25% NaOCl as the initial rinse.

Antibacterial Efficacy and Effect on Biofilms

Stojic and colleagues assessed,[500] in a laboratory experimental model, the efficacy of QMiX against *Enterococcus faecalis* and mixed plaque bacteria in planktonic phase and biofilms. QMiX and 1% NaOCl killed all planktonic *E. faecalis* and plaque bacteria in 5 seconds. QMiX and 2% NaOCl killed up to 12 times more biofilm bacteria than 1% NaOCl ($P < .01$) or 2% CHX ($P < .05$; $P < .001$). Wang and colleagues compared the antibacterial effects of different disinfecting solutions on young and old *E. faecalis* biofilms in dentin canals using a novel dentin infection model and confocal laser scanning microscopy.[548] Six percent NaOCl and QMiX were the most effective disinfecting solutions against the young biofilm, whereas against the 3-week-old biofilm, 6% NaOCl was the most effective followed by QMiX. Both were more effective than 2% NaOCl and 2% CHX. Morgental and colleagues showed that QMiX was less effective than 6% NaOCl and similar to 1%NaOCl in bactericidal action.[337] According to their in vitro study, it appears that the presence of dentin slurry has the potential to inhibit most current antimicrobials in the root canals system,

Moreover, Ordinola and colleagues found that several endodontic irrigants containing antimicrobial compounds such as chlorhexidine (QMiX), cetrimide, maleic acid, iodine compounds, or antibiotics (MTAD) lacked an effective antibiofilm activity when the dentin was infected intraorally.[353] The irrigant solutions 4% peracetic acid and 2.5% to 5.25% sodium hypochlorite decreased significantly the number of live bacteria in biofilms, providing also cleaner dentin surfaces ($P < .05$). They concluded that several chelating agents containing antimicrobials could not remove or kill significantly biofilms developed on intraorally infected dentin, with the exception of

sodium hypochlorite and 4% peracetic acid. Dissolution ability is mandatory for an appropriate eradication of biofilms attached to dentin.

Clinical Trials

The efficacy and biocompatibility of QMiX was demonstrated via nonclinical in vitro and ex vivo studies. Further clinical research from independent investigators is needed to corroborate the findings.

Iodine Potassium Iodide

Iodine potassium iodide (IKI) is a root canal disinfectant that is used in concentrations ranging from 2% to 5%. IKI kills a wide spectrum of microorganisms found in root canals but shows relatively low toxicity in experiments using tissue cultures.[491] Iodine acts as an oxidizing agent by reacting with free sulfhydryl groups of bacterial enzymes, cleaving disulfide bonds. *E. faecalis* often is associated with therapy-resistant periapical infections (see Chapter 15), and combinations of IKI and CHX may be able to kill $Ca(OH)_2$-resistant bacteria more efficiently. One study evaluated the antibacterial activity of a combination of $Ca(OH)_2$ with IKI or CHX in infected bovine dentin blocks.[473] Although $Ca(OH)_2$ alone was unable to destroy *E. faecalis* inside dentinal tubules, $Ca(OH)_2$ mixed with either IKI or CHX effectively disinfected dentin. Others demonstrated that IKI was able to eliminate *E. faecalis* from bovine root dentin when used with a 15-minute contact time.[32] An obvious disadvantage of iodine is a possible allergic reaction in some patients. Although iodine is not generally considered an allergen, some patients are hypersensitive to this compound and may be considered to have an iodine "allergy."

Intracanal Medication

When treatment cannot be completed in one appointment (see Chapters 3 and 14), the surviving intracanal bacteria proliferate between appointments.[85,546] Therefore, an intracanal medication that will restrict bacterial regrowth, supply continued disinfection, and create a physical barrier can be advantageous.

Calcium Hydroxide

Calcium hydroxide is the most popular intracanal medication in use. Hermann introduced it in 1920.[215,216] Although its use was well documented for its time, evidence of its efficacy in clinical endodontics is controversial. A series of articles promoted the antibacterial efficacy of $Ca(OH)_2$ in human root canals.[85,87] Subsequent studies substantiated these reports,[354,477] and the routine use of $Ca(OH)_2$ as an interappointment intracanal medicament became widespread.[429]

However, newer clinical studies and systematic reviews failed to show a clear benefit of $Ca(OH)_2$ to further eliminate bacterial from the root canal.[372,429] $Ca(OH)_2$ mostly is used as slurry of $Ca(OH)_2$ in a water base; at body temperature, less than 0.2% of the $Ca(OH)_2$ is dissolved into Ca^{++} and OH^- ions. Because $Ca(OH)_2$ needs water to dissolve, water should be used as the vehicle for the $Ca(OH)_2$ paste. In contact with air, $Ca(OH)_2$ forms calcium carbonate ($CaCO_3$). However, this is an extremely slow process and of little clinical significance. $Ca(OH)_2$ paste, with a significant amount of calcium carbonate, feels granular because the carbonate has a very low solubility.[422,477]

$Ca(OH)_2$ is a slow-acting antiseptic; direct-contact experiments in vitro show that a 24-hour contact period is required for complete killing of enterococci.[422,477] Another study of 42 patients found that NaOCl canal irrigation reduced the bacteria level by only 61.9%, but use of $Ca(OH)_2$ in the canals for 1 week resulted in a 92.5% reduction.[463] These researchers concluded that $Ca(OH)_2$ should be used in infected cases to more predictably obtain disinfection.

In addition to killing bacteria, $Ca(OH)_2$ has the beneficial ability to hydrolyze the lipid moiety of bacterial lipopolysaccharides (LPS), thereby inactivating the biologic activity of the lipopolysaccharide and reducing its effect.[424,423] This is a desirable effect because dead cell wall material remains after the bacteria have been killed and can continue to stimulate inflammatory responses in the periradicular tissue.

The main characteristics of $Ca(OH)_2$ include limited solubility, high pH, use as a broad-spectrum antimicrobial agent, and the ability to sustain antimicrobial action for long periods.

Other Uses of $Ca(OH)_2$

Long-term calcium hydroxide treatment can be used to induce apexification of the immature tooth with pulpal necrosis before placing an obturation material such as gutta-percha in the root canal system.[155] Also, in cases where revascularization is desired, $Ca(OH)_2$ can be used instead of antibiotic pastes (for more details, see the discussion presented later in the chapter and Chapter 10).

Clinical Protocol

Calcium hydroxide should be in contact with the tissue to act. $Ca(OH)_2$ powder may be mixed with sterile water or saline and placed into the canal with using a lentulo paste filler.[371] Alternatively, the mix may be applied from sterile, single-dose packages (e.g., Calasept [J.S. Dental, Ridgefield, CT], Calcijet [Centrix, Shelton, CT], and DT Temporary Dressing [Global Dental Products, North Bellmore, NY]) (Fig. 6-65). The mixture should be thick to carry as many $Ca(OH)_2$ particles as possible; however, it should not be overdried to retain enough moisture and to promote continued dissociation with a resulting high pH. For maximum effectiveness, the root canal should be filled homogeneously to the working length.

Limitations of Calcium Hydroxide

There are some concerns regarding the use of $Ca(OH)_2$. The handling and proper placement of $Ca(OH)_2$ present a challenge to the average clinician.[296,474] Moreover, the removal of $Ca(OH)_2$ is frequently incomplete,[306] resulting in a residue covering 20% to 45% of the canal wall surfaces, even after copious irrigation with saline, NaOCl, or EDTA.[276] Residual $Ca(OH)_2$ can shorten the setting time of zinc oxide eugenol–based endodontic sealers.[315] Most notably, it may interfere with the seal of the root filling and compromise the quality of treatment. An additional concern is that $Ca(OH)_2$ is not totally effective against several endodontic pathogens, including *E. faecalis* and *Candida albicans*.[547] The ability of $Ca(OH)_2$ to completely eradicate bacteria from the root canal has been questioned. For example, in vitro studies have shown that dentin can inactivate the antibacterial activity of $Ca(OH)_2$,[193,393] and one clinical study has shown that the number of bacteria-positive canals actually increased after $Ca(OH)_2$ medication.[372] Other studies have also indicated that $Ca(OH)_2$ could not predictably eliminate bacteria or that cultures changed from negative to positive after $Ca(OH)_2$ placement.[372,546]

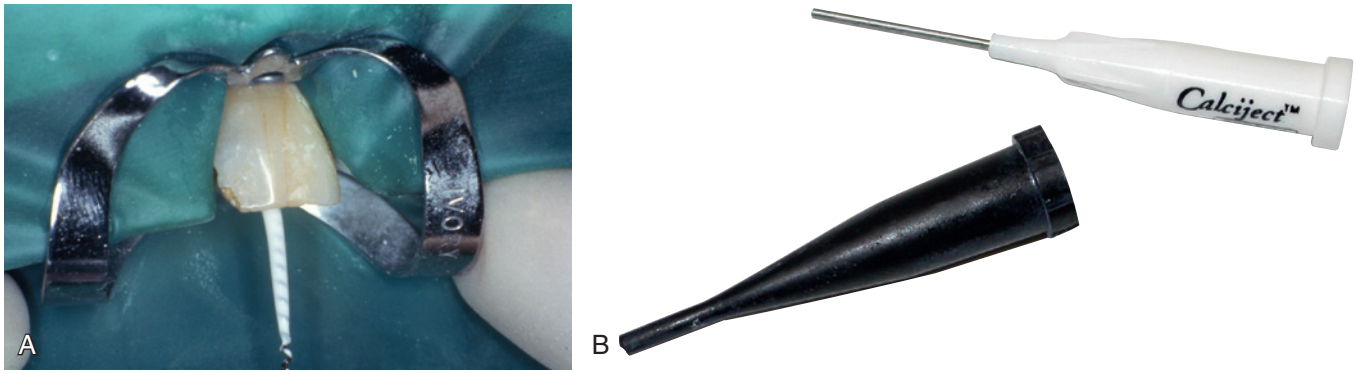


FIG. 6-65 A, Application of calcium hydroxide paste in the canal with a lentulo spiral. B, Calciject is a calcium hydroxide prefilled, easy-to-use, single-dose syringe system. Centrix NeedleTube cartridges can be used for direct syringe injection into the root canal. (*A,* Courtesy Dr. S. Friedman. *B,* Courtesy Centrix, Shelton, CT.)

When different studies report inconsistent results, a systematic review and meta-analysis technique can clarify conflicting research data and the current state of knowledge regarding specific issues. Therefore, based on the current best available evidence, $Ca(OH)_2$ has limited effectiveness in eliminating bacteria from human root canals when assessed by culture techniques. The quest for better antibacterial protocols and sampling techniques must continue to ensure that bacteria can be been reliably eradicated prior to obturation.

Phenolic Preparations

Phenol (C_6H_5OH) and phenolic preparation used to be commonly used intracanal medicament in endodontics. It was thought that because of their volatile properties, they could penetrate dentinal tubules and anatomic irregularities. However, later it was demonstrated that these compounds have a short life span, and their volatility can diffuse through the temporary fillings and also through the periapical tissue causing toxicity. Despite the severe toxicity of phenolic preparations, derivatives of phenol, such as paramonochlorophenol (C_6H_4OHCl), thymol ($C_6H_3OHCH_3C_3H_7$), and cresol ($C_6H_4OHCH_3$), remain available. Currently, $Ca(OH)_2$ or no medication is preferred.[167] Phenol is a nonspecific protoplasm poison that has an optimal antibacterial effect at 1% to 2%. Many dental preparations use much too high a concentration of phenol (e.g., in the range of 30%).[167] At such a concentration, the antimicrobial effect in vivo is lower than optimal and of very short duration.[326] Derivatives of phenol are stronger antiseptics and more toxic than phenol. Phenolic compounds are available as camphorated solutions.[491,492] Camphorated solutions result in a less toxic phenolic compound because they slow the release of toxins to the surrounding tissues. Studies in vitro have shown that phenol and phenol derivatives are highly toxic to mammalian cells, and their antimicrobial effectiveness does not sufficiently balance their toxicity.[491,492] Phenols are ineffective antiseptics under clinical conditions.[85,137]

Formaldehyde

Formaldehyde, used as formocresol, is highly toxic, mutagenic, and carcinogenic; however, it has been used extensively in endodontic therapy.[284] These formulations are still being recommended for use in pediatric dentistry when treating deciduous teeth. The formaldehyde component of formocresol may vary substantially between 19% and 37%. Tricresol formalin, another formaldehyde preparation, contains 10% tricresol and 90% formaldehyde.[284] All of these preparations have a formaldehyde content well above the 10% normally used for fixation of pathologic specimens. Formaldehyde is volatile and releases antimicrobial vapors when applied to a cotton pellet for pulp chamber disinfection. All formaldehyde preparations are potent toxins with an antimicrobial effectiveness much lower than their toxicity.[492,493] There is no clinical reason to use formocresol as an antimicrobial agent for endodontic treatment, based on what is known at this time. The alternatives are better antiseptics with significantly lower toxicity.[492,493]

Halogens

Chlorinated solutions have been used for many years to irrigate root canals. They are also used as intracanal dressings in the form of chloramine-T, an *N*-chloro-tosylamide sodium salt. Iodine, in the form of IKI, is a very effective antiseptic solution with low tissue toxicity. IKI is an effective disinfectant for infected dentin and can kill bacteria in infected dentin in 5 minutes in vitro.[422] IKI releases vapors with a strong antimicrobial effect. The solution can be prepared by mixing 2 g of iodine in 4 g of potassium iodide; this mixture then is dissolved in 94 ml of distilled water. Tincture of iodine (5%) has proved to be one of the few reliable agents for disinfecting rubber dam and tooth surfaces during the preparation of an aseptic endodontic workfield.[334]

Chlorhexidine

CHX is also used as an intracanal medication and was discussed extensively earlier in this chapter.

Steroids

Steroids have been used locally, within the root canal system, to reduce pain and inflammation. Ledermix (Riemser Arzneimittel AG, Insel Riems, Germany) is a commercially available product that was developed about 1960 by Prof. André Schroeder.[450] The active ingredients are the potent anti-inflammatory corticoid triamcinolone acetonide in combination with the broad-spectrum antibiotic demeclocycline. It is an intracanal medicament paste popularly used in some countries. Ledermix paste has been advocated as an initial dressing, particularly if the patient presents with endodontic symptoms.[450] Ledermix paste contains triamcinolone acetonide as an anti-inflammatory agent, at a concentration of 1%.[261] The

clinical effect is a rapid relief of pain associated with acute inflammatory conditions of the pulp and periodontium.

Ledermix paste is a nonsetting, water-soluble paste material for use as root canal medicament or as a direct or indirect pulp-capping agent. The mechanism of action of this substance is based on inhibition of the ribosomal protein synthesis in the bacteria. The release and dentin diffusion characteristics of triamcinolone from Ledermix paste when used as a root canal medicament have been investigated under different conditions.[1-3] Collectively, these studies show that triamcinolone is released from Ledermix paste in the root canal and can reach the systemic circulation via diffusion through dentinal tubules, lateral canals, and the apical foramen. Also, because of its root resorption inhibition property, it was tested for replanted teeth in dogs. The results showed that the groups treated with Ledermix, triamcinolone, and demeclocycline had significantly more favorable healing and more remaining root structure than the group filled with gutta-percha and sealer (positive control).[95]

Triple-Antibiotic Paste

The triple-antibiotics regimen, composed of metronidazole, ciprofloxacin, and minocycline, was first tested[430] for its effectiveness against *Escherichia coli*–infected dentin in vitro. The same research group also tested its bactericidal efficacy against microbes from carious dentin and infected pulp. They found that the mixture of antibiotics is sufficiently potent to eradicate the bacteria.[511] The clinical effectiveness of the triple-antibiotic paste in the disinfection of immature teeth with apical periodontitis has been reported.[557] One potential concern of using an intracanal antibiotic paste is that it may cause bacterial resistance. Additionally, intracanal use of minocycline can cause tooth discoloration, creating potential cosmetic complications. For this reason, a dual paste (metronidazole, ciprofloxacin) and, alternatively, abandonment of this protocol in favor of $Ca(OH)_2$ have been considered.[279] Another reason for such a change could be the reported high toxicity to stem cells of paste prepared from antibiotic powder.[418]

Bioactive Glass

Research is under way in the use of bioactive glass as an intracanal medicament. In one study,[578] the glass used was composed of 53% SiO_2 (w/w), 23% Na_2O, 20% CaO, and 4% P_2O_5 and was prepared from reagent-grade Na_2CO_3, $CaHPO_4$, $2H_2O$, $CaCO_3$, and Belgian sand. When used in root canals, bioactive glass was found to kill bacteria, but the mechanism of action was not pH related, and dentin did not seem to alter its effect.[578] Some newer obturating materials (e.g., Resilon; Pentron Clinical Technologies, Wallingford, CT) contain bioactive glass.

Lubricants

In root canal treatment, lubricants are mostly used to emulsify and keep in suspension debris produced by mechanical instrumentation. Although irrigation solutions serve as lubricants for hand instrumentation, special gel-type substances are also marketed. Two of these are wax-based RC-Prep, which contains EDTA and urea peroxide, and glycol-based Glyde. Another purported function of lubricants is to facilitate the mechanical action of endodontic hand or rotary files. A study evaluating the effects of lubrication on cutting efficiency found that tap water and 2.5% NaOCl solutions increased cutting efficiency compared with dry conditions.[572] The authors of this study cited the ability of an irrigant to remove debris as the factor for the increased efficiency. Similarly, a reduction of torque scores was found when canals in normed dentin disks were prepared with ProFile and ProTaper instruments under irrigation, but the use of a gel-type lubricant resulted in similar torques as in dry, nonlubricated canals.[61,376]

In summary, irrigation is an indispensable step in root canal treatment to ensure disinfection. NaOCl is the irrigant of choice because of its tissue-dissolving and disinfecting properties. EDTA or other chelators should be used at the end of a procedure to remove the smear layer, followed by a final flush with NaOCl for 1 minute for maximum cleaning efficiency and to minimize dentin erosion. This strategy also minimizes inactivation of NaOCl by chemical interactions.

Disinfection Devices and Techniques

Syringe Delivery

Application of an irrigant into a canal by means of a syringe and needle allows exact placement, replenishing of existing fluid, rinsing out of larger debris particles, as well as allowing direct contact to microorganisms in areas close to the needle tip. In passive syringe irrigation, the actual exchange of irrigant is restricted to 1 to 1.5 mm apical to the needle tip, with fluid dynamics taking place near the needle outlet.[66,575] Volume and speed of fluid flow are proportional to the cleansing efficiency inside a root canal.[66] Therefore, both the diameter and position of the needle outlet determine successful chemomechanical debridement; placement close to working length is required to guarantee fluid exchange at the apical portion of the canal, but close control is required to avoid extrusion.[66,224,322] Therefore, the choice of an appropriate irrigating needle is important. Although larger-gauge needles allows a quicker and larger amount of fluid exchange, the wider diameter does not allow cleaning of the apical and narrower areas of the root canal system (see irrigation dynamics earlier in this chapter) (see Fig. 6-7). Excess pressure or binding of needles into canals during irrigation with no possibility of backflow of the irrigant should be avoided under all circumstances[231] to prevent extrusion into periapical spaces. In immature teeth with wide apical foramina or when the apical constriction no longer exists, special care must be taken to prevent irrigation extrusion and potential accidents.[111]

There are different sizes and types of irrigation needles. The size of the irrigation needle[101] should be chosen depending on the canal size and taper.[89,328,347] Most root canals that have not been instrumented are too narrow to be reached effectively by disinfectants, even when fine irrigation needles are used (see Figs. 6-7 and 6-52). Therefore, effective cleaning of the root canal must include intermittent agitation of the canal content with a small instrument[320,535] to prevent debris from accumulating at the apical portion of the root canal (see Fig 6-47).

Preparation size[328] and taper[105] ultimately determine how close a needle can be placed to the final apical millimeters of a root canal. Open-ended needles are recommended over the end open needles to prevent extrusion of the irrigant. Some needles and suction tips may be attached to the air/water syringe to increase both the speed of irrigant flow and the volume of irrigant. Examples include the Stropko Irrigator (Vista Dental Products), which is an adapter that connects to the air/water syringe and accepts standard Luer-lock needle tips for irrigant removal and application as well as air-drying.

Manually Activated Irrigation

Liquid placed inside the root canal more effectively reaches crevices and mechanically untouched areas if it is agitated inside the root canal. Corono-apical movements of the irrigation needle,[231] stirring movements with small endodontic instruments,[320,535] and manual push-pull movements using a fitted master gutta-percha cone have been recommended.[225]

Other than conventional irrigation, additional techniques for endodontic disinfection have been proposed and tested, including laser systems and gaseous ozone. Several new devices for endodontic irrigation or disinfection have been introduced, among which are the EndoActivator System (Dentsply Tulsa Dental Specialties), passive ultrasonic irrigation, EndoVac (Discus Dental Inc., Culver City, CA), the Safety-Irrigator (Vista Dental Products, Racine, WI), the Self-adjusting File (discussed earlier), photoactivation disinfection, and ozone. These new devices use pressure, vacuum, oscillation, or a combination with suction.

Sonically Activated Irrigation

The EndoActivator System uses safe, noncutting polymer tips in an easy-to-use subsonic hand piece to quickly and vigorously agitate irrigant solutions during endodontic therapy (Fig. 6-66).

In one study,[130] the safety of various intracanal irrigation systems was analyzed by measuring the apical extrusion of irrigant. The authors concluded that EndoActivator had a minimal statistically insignificant amount of irrigant extruded out of the apex in comparison with manual, ultrasonic, and Rinsendo (Dürr Dental, Bietigheim-Bissingen, Germany) groups.[130]

When cleanliness of the root canal walls was analyzed, [421] investigators suggested that both passive sonic or ultrasonic irrigation rendered root canals significantly cleaner than manual preparation in comparison with manual syringe irrigation.[82,533] When comparing sonic with ultrasonic irrigation, the results can be controversial. The majority of the studies benefit ultrasonic irrigation.[246,421] The difference lies in the oscillating movements: sonic devices range between 1500 Hz and 6000 Hz, and ultrasonic equipment requires vibrations greater than 20,000 Hz.[246,302,497] If sonic devices are left in the canal for longer periods of time, better cleaning effects can be found. Sonic or ultrasonic irrigation may be carried out with activated smooth wires or plastic inserts, endodontic instruments, or activated irrigation needles. Examples include EndoSonor (Dentsply Maillefer) and EndoSoft ESI (EMS Electro Medical Systems, Nyon, Switzerland) inserts, IrriSafe (Acteon Satelec), the EndoActivator System (Dentsply Tulsa Dental Specialties), and the Vibringe sonic syringe (Vibringe B.V., Amsterdam, Netherlands). Inadvertent cavitation of root canal walls has not been observed with sonic activation of instruments.[320]

Passive Ultrasonic Irrigation

Richman was the first to introduce ultrasonic devices in endodontics.[405] The files are driven to oscillate at ultrasonic frequencies of 25 to 30 kHz to mechanically prepare the root canal walls.[543,544] (See the discussion presented earlier in this chapter for more details.) It has been shown that ultrasonically driven files are effective to activate the irrigation liquids inside the root canal system by inducing acoustic streaming and cavitation.

Two types of ultrasonic irrigation have been described in the literature: one where irrigation is combined with simultaneous ultrasonic instrumentation (UI) and another without simultaneous instrumentation, called *passive ultrasonic irrigation* (PUI).[8,554] During UI, the file is intentionally brought into contact with the root canal wall. But because of the complex canal anatomy, the UI will never contact the entire wall and it may result in uncontrolled cutting of the root canal walls without effective disinfection.[8,373,560] In a study by Macedo,[307] instrument oscillation frequency, ultrasonic power, and file taper determined the occurrence and extent of cavitation. Some degree of cavitation occurred between the file and canal surface and reached lateral canals and isthmuses.

Passive ultrasonic irrigation (PUI) was first described by Weller and colleagues[554] The term *passive* is related to the noncutting action of the ultrasonically activated file.[8] PUI relies on the transmission of acoustic energy from an oscillating file or smooth wire to an irrigant in the root canal.

PUI should be introduced in the canal once that the root canal system has a final apical size and taper. A fresh solution of irrigant should be introduced and a small file or smooth wire (for example, size #15) is ultrasonically activated. Because the root canal has already been shaped, the file or wire can move more freely,[416,534,535] and the irrigant can penetrate into the apical part of the root canal system,[269] with the cleaning effect being more significant.[301] Using this noncutting methodology, the potential to create aberrant shapes within the root canal are reduced to a minimum. Obviously, a file larger than a #15 or #20 will require a wide root canal to reduce oscillation dampening by wall contact.

Ultrasonic activation of the irrigant seems to improve debridement of the root canal system in vitro, and the results in vivo present some controversy. Therefore, objective guidelines regarding their risks and benefits have not been ascertained.[286,575] Ultrasonic activation of the irrigant can be intermittent or continue. The ProUltra PiezoFlow (Dentsply Tulsa Dental Specialties) has been introduced to irrigate and activate the liquids at the same time. The device consists mainly of an ultrasonically energized needle connected to a reservoir of sodium hypochlorite (NaOCl). This continuous ultrasonic irrigation (CUI) system allows simultaneous continuous irrigant delivery and ultrasonic activation; unlike PUI,

FIG. 6-66 The EndoActivator, a sonic frequency system. (Courtesy Dentsply Tulsa Dental Specialties, Tulsa, OK.)

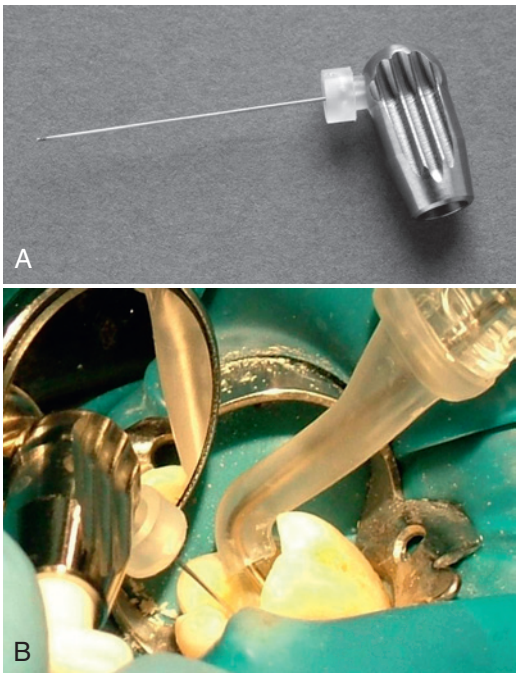


FIG. 6-67 The EndoVac system: magnification of the closed-ended microcannula (**A**), and clinical view of the EndoVac system combined with the Safety-Irrigator (**B**). (*A,* Courtesy Discus Dental Inc., Culver City, CA. *B,* Courtesy Dr. A. Azarpazhooh.)

it does not require the intermittent replenishment of irrigant between ultrasonic file activations. Research shows better elimination of debris and better penetration of irrigant into dentinal tubules.[6,93,247]

Negative Apical Pressure

Another approach to afford better access of irrigation solution to the apical portion of the canal is so-called negative-pressure irrigation (Fig. 6-67). Here, irrigant is delivered into the access chamber, and a very fine needle connected to the dental unit's suction device is placed into the root canal. Excess irrigant from the access cavity is then transported apically and ultimately removed via suction. First, a macrocannula, equivalent to an ISO size #55, .02 taper instrument, removes coronal debris. Subsequently, a microcannula, equivalent to a size #32, .02 taper, removes particles lodged close to working length. Such a system is commercially available (EndoVac, Discus Dental) and may prove a valuable adjunct in canal disinfection.[348] One of the main characteristics of the system is the safety. Many studies proved that EndoVac will not extrude irrigation solution through the apex. On the other hand, because the irrigation is deposited in the coronal area, the irrigant flow in the apical last millimeters of the canal is very passive, and some concerns were expressed that flow is laminar and passive in the apical region. In one study, the apical negative pressure mode of irrigation generated the lowest wall shear stress.[96]

Another device that makes use of pressure-suction technology is the RinsEndo system (Dürr Dental, Bietigheim-Bissingen, Germany). It aspirates the delivered rinsing solution into an irrigation needle that is placed close to working length and at the same time activates the needle with oscillations of 1.6 Hz amplitude.[69,322]

FIG. 6-68 The Safety-Irrigator. (Courtesy Vista Dental Products, Racine, WI.)

FIG. 6-69 The GentleWave system. The system uses multisonic energy to develop a broad section of waves within the irrigation solution to clean inside the roots canal system. It has two main components: a handpiece and a console (shown in figure). (Courtesy Sonendo, Inc, Laguna Hills, CA.)

Safety-Irrigator

The Safety-Irrigator (Vista Dental Products) is an irrigation/evacuation system that apically delivers the irrigant under positive pressure through a thin needle containing a lateral opening and evacuates the solution through a large needle at the root canal orifice (Fig. 6-68). The Safety-Irrigator features a large coronal evacuation tube, enabling the user to safely irrigate and evacuate simultaneously. It fits any standard Luer-lock syringe. Designed to limit risk of NaOCl accidents, this "negative-pressure" irrigation device comes fully assembled and fitted with a side-vented irrigating needle for added safety. This system was tested in vitro to evaluate the removal of dentin debris from artificially made grooves in standardized root canals and showed that there was no significant difference among the manual dynamic activation (MDA) with a nontapered gutta-percha cone, the Safety Irrigator, and the apical negative pressure irrigation. These techniques produced better cleaning efficacy than syringe irrigation ($P < .005$) but significantly worse than MDA with a tapered cone ($P < .05$). Continuous ultrasonic irrigation was significantly better than all the other techniques tested in this study ($P < .001$).[247]

Gentle Wave System

Sonendo Inc. develops a so-called multisonic cleaning technology (Gentle Wave, Fig. 6-69) that only requires pulp chamber access. This system is noninstrumentational and being tested

clinically with, according to the company, promising results. It appears to have the potential to reach inaccessible canal areas with significantly more cleaned surface than other systems. A first in vitro study shows the potential of the system to debride better than conventional needle irrigation or ultrasonically activated irrigation.[197]

Laser-Activated Irrigation

Lasers are widely used in dentistry and include diode, Nd:YAG, erbium, and CO_2, which produces radiation in both the near- and far-infrared electromagnetic spectrum.[186] Laser devices have been proposed to improve the efficacy of irrigants.[186] Lasers have been studied for their ability to clean and effectively disinfect root canals. The Er:YAG laser wavelength (2940 nm) has the highest absorption in water and a high affinity to hydroxyapatite, which makes it suitable for use in root canal treatment.[107]

Laser energy may be used to activate irrigant solutions in different ways—for example, at a molecular level, as in photoactivated disinfection (PAD), or at a bulk flow level, as in laser-activated irrigation (LAI). Several studies in vivo and ex vivo have indicated that laser activated irrigation is promising for removing smear layer[168] and dentin debris[121,122] in less time than PUI. The mechanism of action[56] is based on the generation of a secondary cavitation effect with expansion and successive implosion of fluids.[56]

These results are in agreement with data related to a new erbium laser technique that used a photon-induced photoacoustic streaming (PIPS) of irrigants. In that technique, the laser tip is placed into the coronal access opening of the pulp chamber only and is kept stationary without advancing into the orifice of the canal.[132] The use of a newly designed tapered and stripped tip with specific minimally ablative laser settings is required, resulting in low energy (20 mJ), a pulse repetition rate of 15 Hz, and a very short pulse duration (50 µs). The difference in laser penetration and bacterial killing is attributed to the difference in the degree of absorption of different wavelengths of light within the dentin. Bergmans and colleagues concluded in their in vivo study that the Nd:YAG laser irradiation is not an alternative but a possible supplement to existing protocols for canal disinfection, as the properties of laser light may allow a bactericidal effect beyond 1 mm of dentine.[49] Endodontic pathogens that grow as biofilms, however, are difficult to eradicate even upon direct laser exposure.[65]

Photoactivation Disinfection

Photodynamic therapy (PDT) or light-activated therapy (LAT) may have endodontic applications because of its antimicrobial effectiveness.[203] In principle, antimicrobial photodynamic therapy (APDT) is a two-step procedure that involves the introduction of a photosensitizer (step 1: photosensitization of the infected tissue) followed by light illumination (step 2: irradiation of the photosensitized tissue) of the sensitized tissue, which would generate a toxic photochemistry on the target cell, leading to cell lysis. Each of these elements used independently will not have any action, but together they have a synergism effect to produce antibacterial action. Indeed, in vitro experiments showed promising results when used as an adjunct disinfected device. Shresta and Kishen concluded that the tissue inhibitors existing within the root canal affected the antibacterial activity of PDT at varying degrees,[462] and further research is required to enhance their antimicrobial efficacy in an endodontic environment.

Antibacterial Nanoparticles

Nanoparticles are microscopic particles with one or more dimensions in the range of 1 to 100 nm. Nanoparticles are recognized to have properties that are unique from their bulk or powder counterparts. Antibacterial nanoparticles have been found to have a broad spectrum of antimicrobial activity and a far lower propensity to induce microbial resistance than antibiotics. Such nanoparticles in endodontics are being studied in different ways, such as mixed with irrigants, photosensitizer, and sealers.[259] Currently, the consensus is that the successful application of nanoparticles in endodontics will depend on both the effectiveness of antimicrobial nanoparticles and the delivery method used to disperse these particles into the anatomic complexities of the root canal system.

Superoxidized Water

Superoxidized water,[191] also called electrochemically activated water[313,481] or oxidative potential water,[208,455] is effectively saline that has been electrolyzed to form superoxidized water, hypochlorous acid, and free chlorine radicals. It is commercially available as Sterilox (Sterilox Technologies, Radnor, PA). This solution is nontoxic to biologic tissues yet able to kill microorganisms. The solution is generated by electrolyzing saline solution, a process no different than that used in the commercial production of NaOCl.[154] The difference, however, is that the solution accumulating at the anode is harvested as the anolyte and that at the cathode as the catholyte. These solutions display properties that are dependent on the strength of the initial saline solution, the applied potential difference, and the rate of generation. The technology that allows harvesting of the respective solutions resides in the design of the anode and the cathode and originates either in Russia (electrochemically activated water) or in Japan (oxidative potential water).[312,313] Although the solutions bear different names, the principles in the manufacturing process appear to be similar.

The use of superoxidized water is sparsely described in the endodontic literature but shows early promise. The solutions from both technologies have been tested for their ability to debride root canals,[208,313] remove smear layer,[455,481] and kill bacteria[223] and bacterial spores.[297] Results are favorable and show biocompatibility with vital systems.[237]

Anolyte and catholyte solutions generated from one such technology (Radical Waters Halfway House 1685, South Africa) have shown promise as antibacterial agents against laboratory-grown, single-species biofilm models.[169] Such solutions have been recommended as suitable for removing biofilms in dental unit water lines[312] and have even been marketed for this purpose. Cautious clinicians may prefer to wait for more studies to demonstrate safety and efficacy under ordinary clinical setting conditions before adopting newer, less tested irrigating solutions.

CRITERIA TO EVALUATE CLEANING AND SHAPING

Well-Shaped Canals

The main aims of canal shaping are to directly remove tissues and microbial irritants and to provide sufficient

geometrical space for subsequent obturation (see Table 6-2). To achieve these goals, the prepared canal should include the original canal (see the red areas in Fig. 6-4); there should be an apical narrowing and the canal should be tapered. These concepts were popularized by Schilder[445] and are still maintained today.[17,258]

Therefore, a well-shaped canal is defined more specifically by the absence of procedural errors (discussed later) and the achievement of disinfection; more recently another element was added to this equation, the retention of as much tooth structure as feasible.[172]

A clinician can often determine whether a canal is adequately shaped by examining radiographs and relying on clinical experience—for example, when fitting a cone. At this time the feel would be of tug-back, a slight resistance to pull; a radiograph should show a symmetrical canal-shape lateral of the cone, the presence of an intact apical narrowing, and no thinned-out radicular wall sections.

Using magnification, clinicians should inspect the canal orifice and the coronal third of each shaped canal for clean canal walls.[367] Immediately after irrigation with sodium hypochlorite, an absence of visible turbidity and effervescence should be noted. If present, these phenomena, along with visible deposits on the canal walls, are indicative of organic matter still in suspension or adherent to the radicular walls.

Signs of Mishaps

Instrument Fracture

Most reports suggest that manual endodontic file fracture or rotary instrument fracture occur at a rate of approximately 1% to 6% and 0.4% to 5%, respectively.[365,495,531] Such fractures are untoward events and perceived as such by clinicians.[55] Evidently, retained instrument fragments limit access of disinfecting irrigants to the root canal system, possibly impeding sufficient elimination of microorganisms.[196] However, the current clinical evidence does not suggest that the presence of a retained instrument must result in a significantly higher rate of failing root canal treatments when done by specialists.[495]

In general, instruments used in rotary motion break into two distinct modes: torsional and flexural.[374,431,530] Torsional fracture occurs when an instrument tip is locked in a canal while the shank continues to rotate, thereby exerting enough torque to fracture the tip. This also may occur when instrument rotation is sufficiently slowed in relation to the cross-sectional diameter. In contrast, flexural fracture occurs when the cyclic loading leads to metal fatigue. This problem precludes the manufacture of continuously rotating stainless steel endodontic instruments, because steel develops fatal fatigue after only a few cycles.[454] NiTi instruments can withstand several hundred flexural cycles before they fracture,[201,285,394,530,567] but they still can fracture in the endodontic setting after a low (i.e., below 10,000) number of cycles.[97]

Repeated loading and cyclic fatigue tests for endodontic instruments are not described in pertinent norms. Initially, rotary instruments such as Gates-Glidden burs and Peeso reamers were tested with a superimposed bending deflection.[68] In GG burs, a 2-mm deflection of the instrument tip resulted in fatigue life spans ranging from 21,000 revolutions (size #1 burs) to 400 revolutions (size #6 burs).[68] In another study, stainless steel and NiTi hand files were rotated to failure in steel tubes with an acute 90-degree bend and an unspecified radius.[454] Under these conditions, size #40 stainless steel instruments fractured after fewer than 20 rotations, whereas various NiTi files of the same size withstood up to 450 rotations.

Cyclic fatigue was also evaluated for ProFile .06 taper instruments using a similar device.[567,568] The number of rotations to failure for unused control instruments ranged from 1260 (size #15 files) to 900 (size #40 files). These scores did not change when the instruments were tested under simulated clinical conditions such as repeated sterilization and contact with 2.5% NaOCl. Subsequently, control instruments were compared with a group of instruments used in the clinical setting in five molar cases[568]; again, no significant differences were found in resistance to cyclic fatigue.

One study used a different testing method involving tempered metal cylinders with radii of 5 mm and 10 mm that produced a 90-degree curve.[201] The researchers reported fatigue fractures for size #15, .04 taper ProFile instruments after about 2800 cycles with the 10 mm cylinders. In size #40, .04 taper ProFile instruments, fractures occurred after about 500 cycles with the 5-mm cylinders. In comparison, size #15, .06 taper ProFile instruments also failed after about 2800 revolutions with the 10-mm cylinders, but failure occurred in size #40, .06 taper ProFile specimens after only 223 cycles with the 5-mm cylinders.

Rotary NiTi instruments with larger tapers and sizes consistently fractured after fewer rotations,[390] and although the radius of the curves was halved, fatigue life was reduced by 400%. Another investigation reported similar results for selected HERO instruments,[201] and the findings were confirmed by other tests on GT rotary instruments. Size #20, .06 taper GT files failed after 530 rotations in a 90-degree curve with a 5-mm radius; size #20, .12 taper GT files failed after 56 rotations under the same conditions.[378]

Reuse of rotary instruments depends on safety, specifically on assessment of fatigue and also the potential to properly clean NiTi surfaces.[36,63,350,357,460,486,489,525] Specific instruments perform differently in this regard, as fatigue depends more on the amount of metal in cross section at the point of stress concentration[182,523] than on the specifics of instrument design.[98]

On the other hand, manufacturers constantly claim that their instrument has been equipped with design elements that render it more fatigue resistant. For example, LightSpeed LSX is manufactured without a milling process. However, no data have been published regarding its fatigue resistance. GTX is manufactured from a novel NiTi alloy, M-Wire, to increase its fatigue resistance.[249] However, investigators could not confirm these findings.[266] Similarly, another study did not find the Twisted File,[277] which is not milled and hence thought to be fatigue resistant,[163] to perform better than conventionally manufactured ProFile rotaries. Another feature, electropolishing (discussed earlier), does not appear to confer a significantly increased fatigue resistance to EndoSequence[277,401] and RaCe.[523,525,565] One possible reason for these variable outcomes is the different testing environments used in vitro[100]; clinically, even greater variability is to be expected.

Attempts have been made to use tests according to norms and specifications described for stainless steel hand instruments such as K-files and Hedström files,[239] as no comparable norms exist for instruments used in continuous rotary motion. Consequently, a number of models have been devised to assess specific properties of NiTi rotary instruments, including torque at failure, resistance against cyclic fatigue, and others

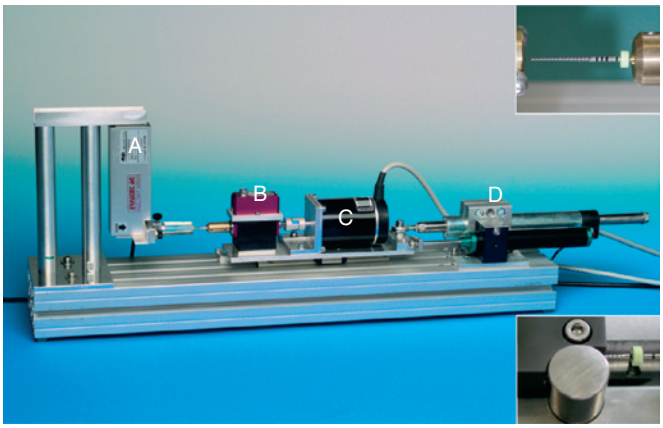


FIG. 6-70 Testing platform for analysis of various factors during simulated canal preparation with rotary endodontic instruments. Labeled components are a force transducer **(A)**, a torque sensor **(B)**, a direct-drive motor **(C)**, and an automated feed device **(D)**. For specific tests, a cyclic fatigue phantom or a brass mount compliant with ISO No. 3630-1 *(insets)* may be attached.

(Fig. 6-70). These systems may simultaneously assess torque at failure, working torque axial force, and cyclic fatigue.

According to the norms mentioned previously, torque at failure is recorded with the apical 3 mm of the instrument firmly held in the testing device while the instrument's handle is rotated. A wide variety of rotary NiTi endodontic instruments have been tested in this way—for example, ProFile NiTi rotary files in ISO sizes #25, #30, and #35 (.04 taper) fractured at 0.78, 1.06, and 1.47 Ncm, respectively.[503]

Investigators reported similar scores when instruments were forced to fracture in plastic blocks with simulated curved canals.[504] In a different setup, GT rotary instruments (size #20, .06 taper to size #20, .12 taper) fractured at 0.51 and 1.2 Ncm, respectively.[378] These values are somewhat lower than data obtained from the same but slightly modified torque bench,[254] pointing to the importance of experimental conditions for torque and fatigue measurements.

Compared with NiTi instruments with tapered flutes, LightSpeed instruments had lower torque to fracture (0.23 to 2 Ncm).[318] No such data are currently available for LightSpeed LSX.

When analyzing clinical factors involved in instrument fracture, one must consider both torsional load and cyclic fatigue.[431] However, these are not separate entities, especially in curved canals.[64] Working an instrument with high torque may lower resistance to cyclic fatigue.[161] Conversely, cyclic prestressing has been shown to reduce the torsional resistance of ProTaper finishing files,[530] as well as K3[31] and MTwo.[390] Also, cyclic fatigue occurs not only in the lateral aspect when an instrument rotates in a curved canal but also axially when an instrument is bound and released by canal irregularities.[54]

The torque generated during canal preparation depends on a variety of factors, and an important one is the contact area.[57] The size of the surface area contacted by an endodontic instrument is influenced by the instrumentation sequence or by the use of instruments with different tapers.[448] A crown-down approach is recommended to reduce torsional loads (and thus the risk of fracture) by preventing a large portion of the tapered rotating instrument from engaging root dentin (known as *taper lock*).[57,569]

The clinician can further modify torque by varying axial pressure, because these two factors are related448 (see Fig. 6-20). In fact, a light touch is recommended for all current NiTi instruments to avoid forcing the instrument into taper lock. The same effect might occur in certain anatomic situations, such as when canals merge, dilacerate, and divide.

The torsional behavior of NiTi rotary endodontic instruments cannot be described properly without advanced measurement systems and a new set of norms. However, the clinician must be able to interpret correctly the stress-strain curves for all rotary NiTi instruments used in the clinical setting to be able to choose an appropriate working torque and axial force.

Therefore, a careful evaluation should be performed before the attempt is made to remove any retained fragment (see Fig. 6-25). In fact, Ward and colleagues suggested that any attempt of fragment removal be made only when the fragment is located coronal of a significant root canal curve and thus visible with the aid of magnification.[549]

There are sophisticated means and strategies to remove retained fragments, which are described in detail in Chapter 19.

Of note, assessment of physical parameters governing rotary root canal preparation was considered crucial because NiTi rotary in vitro had increased risk of fracture compared with K-files. Some clinicians also describe instrument fracture as a main issue for concern.[55]

In a study using plastic blocks, as many as 52 ProFile Series 29 instruments became permanently deformed.[515] Three fractures were reported in a subsequent study on ISO-norm ProFile taper .04 instruments, and three other instruments were distorted.[75] An even higher fracture incidence was shown in a study on rotary instruments used in plastic blocks in a specially designed testing machine.[509] These findings were supported by two studies in which high fracture incidences were reported for LightSpeed and Quantec rotary instruments used in a clinical setting.[33,431]

On the other hand, as stated previously, a retrospective clinical study suggested similar outcomes with and without retained instrument fragments[495]; moreover, others' experience suggests that the number of rotary instrument fractures is lower than previously estimated.[126,262,558] Removal of such fragments is possible in many situations, but there is also the potential for further damage (e.g., perforation) rather than successful removal.[502,558] Consequently, a benefit-versus-risk analysis should be carried out prior to attempts to remove NiTi instrument fragments to address the reasons and the clinical consequences of instrument fracture.

Canal Transportation

Perhaps the most frequent adverse outcomes during canal shaping are aberrations from the original canal path. Much has been written on the appearance of such aberrations using labels as zip and elbow formation, ledging, perforation, stripping, and others.[553]

Canal transportation is at the root of all these clinical problems and may be defined as "the removal of canal wall structure on the outside curve in the apical half of the canal due to the tendency of files to restore themselves to their original linear shape during canal preparation."[17]

As files tend to straighten in the canal, transportation typically occurs toward the inner (or convex) radicular wall at

midroot. Such a shift of the canal axis during shaping results in excessive loss of dentin and may ultimately result in perforation, whereas apical transportation may lead to zipping or apical perforation (see Fig. 6-40).

Conceptually, any canal preparation will shift the canal axis, which is often determined as the center of gravity in cross sections. It has been held that a transportation of about 100 to 150 µm may be clinically acceptable.[233]

If canal transportation has led to ledge formation, subsequent instruments will bypass the area of the ledge only when adequately precurved. In case of rotary instrumentation, hand-operated instruments of comparable size are then recommended (Fig. 6-71).

Perforation

As indicated earlier, perforation may be the ultimate result of canal transportation when it occurs within the root canal system. Other perforations are those in the access cavity; a discussion of access cavity preparation may be found in Chapter 5. Obviously, the preparation of mineralized canals requires advanced operator skills and is facilitated by magnification (Fig. 6-72).

Three types of perforations can be defined: strip perforations that occur toward the furcation in multirooted teeth (also known as *"danger zones"*[15]), perforations associated with canal curvatures, and perforations though the apical foramen.

Blockage

A canal may become impassable during the process of cleaning and shaping due to two distinct, but often connected, occurrences. A ledge is a dentinal shelf that is created by shaping instruments that straighten and dig into the convex side of the canal wall. In less severe cases, ledges can be corrected and smoothed out with precurved instruments. This condition may lead to false paths and impede optimal obturation when working length cannot be reached with master cones.

A blockage refers mainly to a root canal area that is filled with densely compacted debris or collagenous pulp remnants (see Fig. 6-39). It may also be caused by other obstacles such as a fractured files or remnants of a preexisting coronal or radicular filling materials. Clinically the presence of ledge or block is signaled by the inability of a straight flexible instrument to penetrate deeper into the root canal; however, this needs to be differentiated from a small or mineralized canal, which causes longer portions of the instrument's cutting flutes to bind.

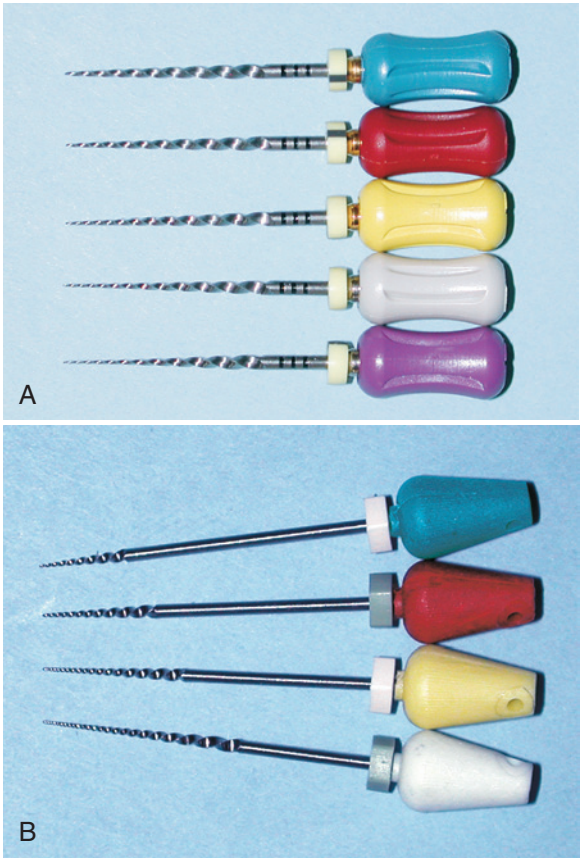


FIG. 6-71 Instruments with increased taper that can be used by hand. **A**, ProTaper instruments with special handles attached to rotary instrument shanks. **B**, GT hand instruments.

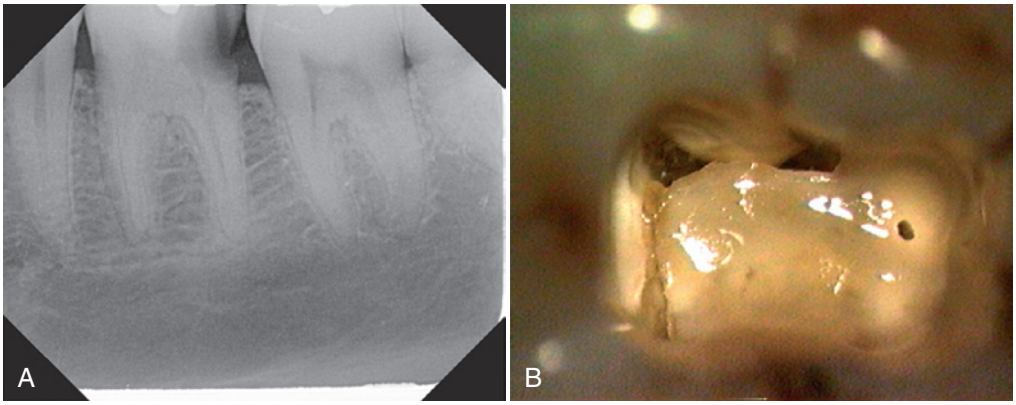


FIG. 6-72 Evidence of coronal hard-tissue deposition. **A**, Periapical radiograph of tooth #19 shows evidence of reduced coronal and radicular pulp space. **B**, Intraoral photograph, taken through an operating microscope (×25), of access cavity of the tooth shown in **A**; note the calcific metamorphosis.

Obviously, such a blockage prevents the apical canal portion from being disinfected. For details of strategies to deal with ledge and blockage, please refer to Chapter 19.

Another reason for a perceived blockage may be an abrupt canal curvature.

SAMPLE PROTOCOL FOR CONTEMPORARY CLEANING AND SHAPING PROCEDURES

- Using well-angulated preoperative radiographs, analyze case difficulty under well-established parameters.
- Place rubber dam and estimate working length.
- Prepare a conservative access cavity sufficient to reveal all root canal orifices.
- Scout canals with a #10 K-file in the presence of a lubricant.
- If the selected series of rotary instruments advances easily to the estimated working length (WL), confirm patency and determine WL using an electronic apex locator.
- If the instruments meet resistance and the file does not progress gently to WL, use a dedicated NiTi instrument; it is prudent to modify the orifice to create a coronal receptacle for the subsequent rotaries. Negotiate, confirm patency, and determine WL.
- Create a reproducible glide path to WL with appropriate instruments.
- Irrigate with sodium hypochlorite throughout the shaping procedure.
- Advance the selected series of rotary instruments (based on canal anatomy) passively in the presence of sodium hypochlorite to shape the middle third. When shaping canals that have a larger buccal-lingual dimension, consider shaping as two canals.
- Clean cutting flutes routinely upon removal, and remove debris with an alcohol-moistened gauze. If the selected rotary does not progress easily, remove irrigant, recapitulate with a #10 K-file, and choose a different, often smaller, instrument.
- Use copious irrigation, and reverify canal patency and working length throughout and upon completion of shaping. Gauge the size of the foramen with an appropriate hand file.
- Protocol for irrigation:
  - Irrigate using copious amounts of sodium hypochlorite.
  - Activate the irrigant.
  - Select irrigation solution for smear layer management.
  - Perform the final irrigation.
- Dry the canal thoroughly and obturate with a technique that promotes a three-dimensional fill.
- Restore the endodontically treated tooth in a timely manner.

SUMMARY

Cleaning and shaping are important, interdependent steps in root canal treatment. Cleaning, as demonstrated by an intracanal surface free of smear layer, can be done only after root canals have been sufficiently enlarged to accommodate adequate irrigation needles. Canal preparation is optimized when mechanical aims are fulfilled and enlargement is acceptable; such aims include avoiding both significant preparation errors and weakening of the radicular structure, which can result in fractures.

Taken together and performed to a high standard, the procedures described in this chapter lay the foundation for biologic success in both straightforward (Fig. 6-73) and more

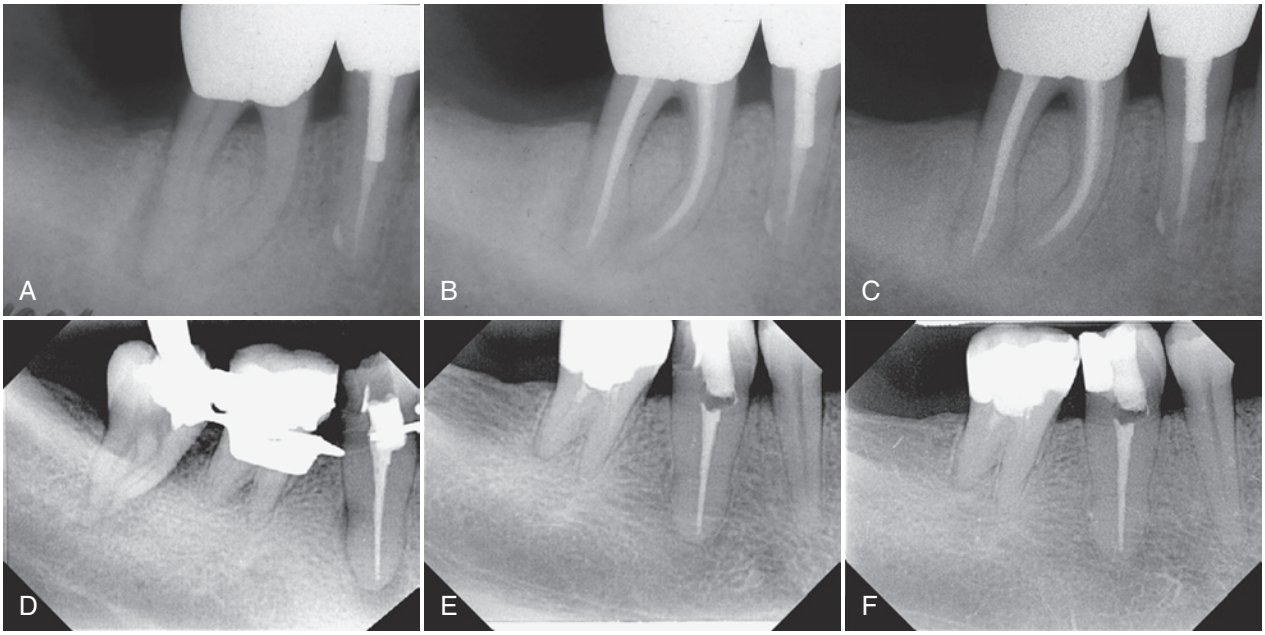


FIG. 6-73 Clinical cases treated according to the principles detailed in this chapter. **A**, Pretreatment radiograph of tooth #30 with a periradicular lesion. **B**, Postobturation radiograph. **C**, Two-year follow-up radiograph shows osseous healing. **D**, Immediate postobturation radiograph of tooth #29 shows both a periapical and a lateral osseous lesion. **E-F**, One-year and three-year follow-up radiographs show progressing osseous healing. Note the imperfect obturation of tooth #30.

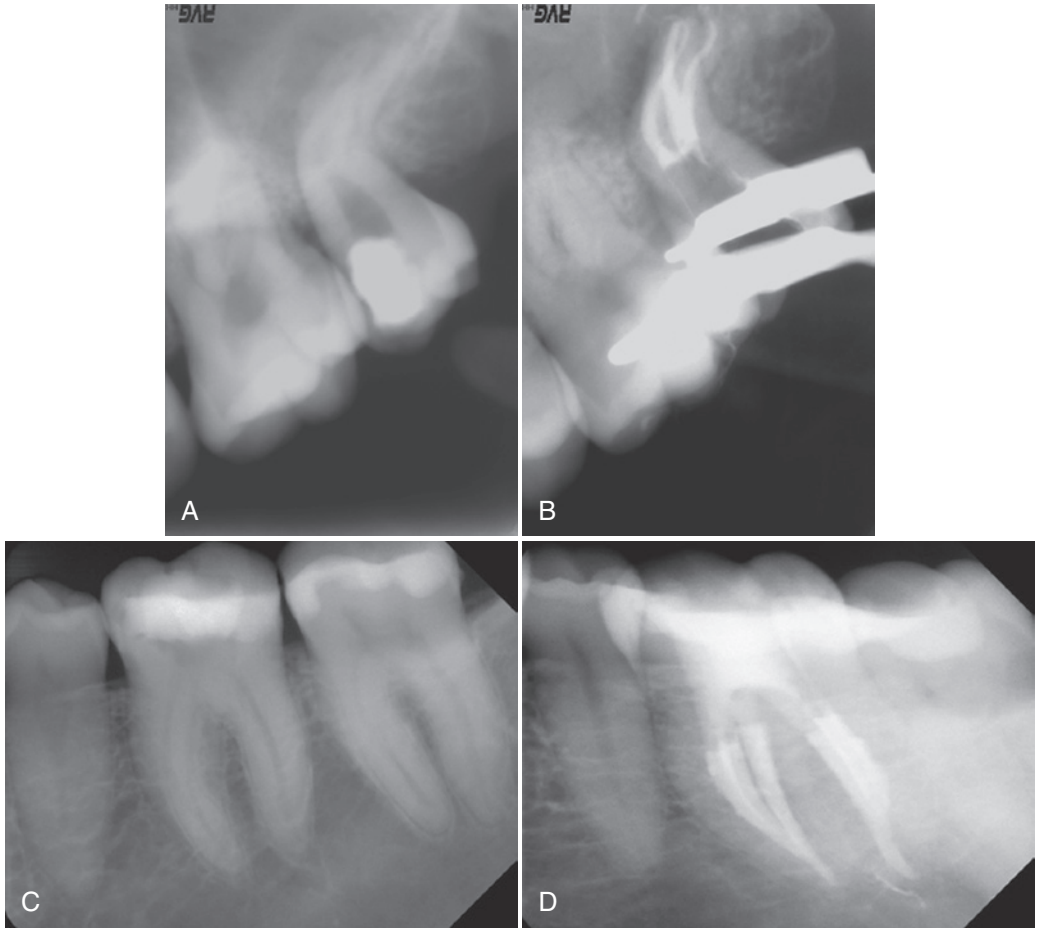


FIG. 6-74 Complicated clinical cases treated with hybrid techniques. **A,** Pretreatment radiograph of tooth #16 indicates laceration and significant curvature of all roots. **B**, Posttreatment radiograph shows multiple planes of curvature. **C,** Pretreatment radiograph of tooth #19, which was diagnosed with irreversible pulpitis. **D,** Angulated posttreatment radiograph shows three canals in the mesiobuccal root canal system, all of which were prepared to apical size #50. (*A-B,* Courtesy T. Clauder. *C-D,* Courtesy Dr. H. Walsch.)

complicated (Fig. 6-74) clinical cases. Recall radiographs confirm favorable outcomes or biologic success (i.e., prevention or healing of periradicular periodontitis) over the years. Similarly, adherence to the principles discussed leads to predictable outcomes for root canal treatments.

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CHAPTER 7

Obturation of the Cleaned and Shaped Root Canal System

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CHAPTER OUTLINE

Importance of Effectively Sealing the Root Canal System
Historical Perspectives
Timing of Obturation
Vital Pulp Tissue
Necrotic Pulp Tissue
Length of Obturation
Preparation for Obturation
The Ideal Root Canal Filling
Types of Sealers
Zinc Oxide and Eugenol
Calcium Hydroxide Sealers
Noneugenol Sealers
Glass Ionomer Sealers
Resin Sealers
Silicone Sealers
Calcium Silicate Sealers
Medicated Sealers
Sealer Placement
Core Materials
Silver Cones
Gutta-Percha
Activ GP
Resilon
Custom Cones
Methods of Obturation
Lateral Compaction
Warm Vertical Compaction
Continuous Wave Compaction Technique
Warm Lateral Compaction
Thermoplastic Injection Techniques
Carrier-Based Gutta-Percha
Thermomechanical Compaction
Solvent Techniques
Pastes
Immediate Obturation
Coronal Orifice Seal

IMPORTANCE OF EFFECTIVELY SEALING THE ROOT CANAL SYSTEM

Success in endodontic treatment was originally based on the triad of debridement, thorough disinfection, and obturation, with all aspects equally important. At present, successful root canal treatment is based on broader principles. These include diagnosis and treatment planning; knowledge of anatomy and morphology; the traditional concepts of debridement, thorough disinfection, and obturation; the coronal restoration, and three-dimensional imaging of the healing of teeth with preexisting periapical pathosis. A meta-analysis of factors influencing the efficacy of primary root canal treatment found that the following four factors influenced success: the absence of a pretreatment periapical lesion, root canal fillings with no voids, obturation to within 2.0 mm of the apex, and an adequate coronal restoration.[265]

In an early radiographic study of success and failure, Ingle and colleagues[179] indicated that 58% of treatment failures were due to incomplete obturation. Unfortunately, teeth that are poorly obturated are often poorly prepared. Procedural errors such as loss of length, canal transportation, perforations, loss of coronal seal, and vertical root fracture may have occurred. These procedural errors have been shown to adversely affect the apical seal.[420]

Since the classic study by Ingle and colleagues, great emphasis has been placed on developing materials and techniques for obturating the radicular space. Various experimental methods have been used to assess microleakage after obturation, including radioisotopes,[102] dyes,[188] bacteria,[68] proteins,[248] endotoxins,[68] glucose penetration,[276] and computerized fluid filtration.[385] These methodologies have employed a variety of in vitro conditions, experimental periods that often produce conflicting results with unclear clinical implications.[113,426] There is general consensus that treatment outcomes cannot be predicted from the results of in vitro dye leakage models.[268,296,373] Even the results of microbial leakage studies have recently been challenged, due to flaws in experimental designs.[306,307] Fortunately, tooth retention rates after root canal treatment are high despite the varied conditions, materials, and techniques employed.[74,218,322] Circumstantial evidence indicates that the cleaning and shaping procedures provide an aseptic environment. With this elimination of the etiology for pathosis, the method of obturation becomes less critical.

A primate study of infected teeth with apical periodontitis demonstrated non-healing in 28% of the teeth with no bacteria after cleaning and shaping, whereas the presence of bacteria after cleaning and shaping resulted in 79% being classified as not healed.[119] When no bacteria were present, healing occurred regardless of the quality of the obturation. When bacteria were present at the time of obturation, there was a correlation between the quality of obturation and nonhealing. These results emphasized the role of bacteria in apical pathosis and the importance of cleaning and shaping procedures.

In a controlled animal study,[318] periapical lesions were created by removing the pulp and leaving the teeth open to the oral cavity. In the control group, the canals were cleaned and shaped before obturation with gutta-percha and a resin sealer. The teeth of the experimental group were cleaned and shaped as in the control group but left unobturated. At 190 days, the animals were killed and histological evaluations were performed. There was no difference in the healing between the instrumented and obturated teeth and the instrumented and unobturated teeth. The results emphasized the importance of cleaning and shaping in eliminating bacteria. Although obturation may not influence the short-term success rates, results may be different in long-term studies if coronal leakage were to occur.[314]

To date, there is no effective method for determining whether cleaning and shaping procedures have been effective. The criteria of clean dentinal filings and/or enlargement beyond the first file to bind at working length proved to be unreliable.[402] Although the length of preparation has been emphasized, the irregular canal diameter (the forgotten dimension) may be a more significant factor in success and failure.[181] Evidence indicates canals are often underprepared in the apical one-third.[67] Historically, culturing has been employed and obturation delayed until a "negative" culture was obtained. In contemporary endodontic treatment, culturing has been abandoned during routine care.[329] With vital pulp tissues, bacteria are not a major concern. In necrotic cases, the organisms involved in the disease process are primarily facultative or obligate anaerobes that may not grow in culture. Molecular microbiologic techniques (polymerase chain reaction) have demonstrated that a variety of organisms are present that do not grow in culture.[10,315,362] The role these organisms play in the disease process is not well understood.[257] The reader is referred to Chapters 14 and 15 for a fuller discussion.

The process of cleaning and shaping determines both the degree of disinfection and the ability to obturate the radicular space. Obturation is therefore a reflection of the cleaning and shaping and is evaluated on the basis of length, taper, density, level of gutta-percha removal, and the coronal seal (i.e., an adequate provisional restoration) (Fig. 7-1). It is not possible to assess the quality of the seal established during obturation with a radiograph, and it is important to remember that no material or technique prevents leakage.[2,153] Indeed, obtaining an impervious seal may not be feasible because of the porous tubular structure of dentin[2] and canal irregularities.

The primary etiology of pulpal and periradicular pathosis is, as discussed in Chapter 15, bacterial.[191,253] Pulpal remnants, necrotic tissue, bacteria, and bacterial by-products remaining in the inaccessible areas of a cleaned and shaped canal system could initiate and/or perpetuate a lesion because the host defense mechanisms are unable to remove them. Studies indicate that root canal systems cannot be completely cleaned and disinfected.[165,359,425] Obturation of the radicular space is necessary to eliminate leakage. Obturation reduces coronal leakage and bacterial contamination, seals the apex from the periapical tissue fluids, and entombs the remaining irritants in the canal.[412]

Coronal leakage has also been proposed to contribute to treatment failure based on in vitro leakage studies.[389,392] The clinical implication is that retreatment has to be performed in those teeth that are not restored permanently after 3 months of root canal treatment. This controversial issue has been recently challenged.[33,310,311] Although a coronal seal may be produced by a well-filled root filling, a coronal restoration with margins that prevent bacteria penetration, or both, data derived from a retrospective clinical study[305] suggest that a favorable treatment outcome may be achieved even in poorly filled root canals when the quality of the coronal restoration is adequate. Data produced by this retrospective study overtly challenged the rationale of endodontics. The data stimulated intense research and discussions for almost two decades on whether the quality of the coronal restoration is more significant than the quality of root canal treatment in eliminating apical periodontitis. A recent systematic review and meta-analysis of the results derived from nine similar studies indicate that poor quality root canal treatment and poor quality coronal restorations have similar odds (i.e., likelihood in non-statistical terms) in adversely affecting the healing of apical periodontitis.[137] Based on the best available evidence currently available, the odds for healing of apical periodontitis increase with both adequate root canal treatment and adequate restorative treatment.[137] It must be emphasized that these two factors were examined in isolation in those studies. While these postoperative prognostic factors are certainly important, as exemplified by the data derived from a prospective clinical study,[262,263] other preoperative and intraoperative prognostic factors also contributed significantly to periapical healing in primary and secondary root canal treatment. Nevertheless, maintaining an effective coronal seal through optimal filling of the instrumented radicular space and placing an appropriate restoration, as discussed in Chapter 22, should be considered essential components of successful root canal treatment.[168,180]

Clinicians are concerned with whether it is more appropriate to place a permanent restorative material instead of a temporary material to prevent leakage.[234,393] This is a controversial issue, as previous studies failed to provide definitive evidence to demonstrate that the presence of a permanent restoration will contribute to the long-term success of root canal treatment when those canals are optimally filled.[76,321] These results were supported by a more recent prospective clinical study that examined the factors affecting the outcomes of nonsurgical root canal treatment.[262] In this study, the authors found that the type of coronal restoration had no significant influence on periapical healing, as long as those restorations are of good quality. Paradoxically, the same group of authors reported in a separate study that cast restorations, when compared with temporary restorations, significantly improved the survival of teeth that had undergone primary or secondary root canal treatment.[263] This may be due to protection of remaining, weakened tooth structure by full-coverage cast restorations that enabled those teeth to function longer. Regarding the necessity for retreatment of root-filled teeth without permanent restorations, there is no clear evidence to justify that retreatment has to be performed in teeth in which a temporary

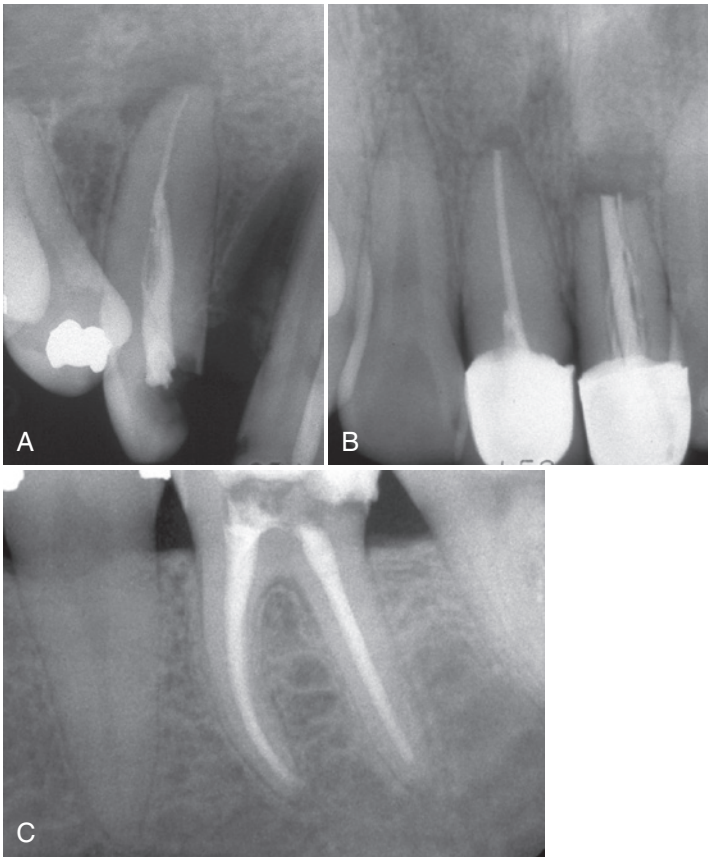

FIG. 7-1 Examples of inadequate obturation. **A,** Maxillary right canine with adequate length but lacking density and no coronal seal. Central incisor is filled to adequate length, but obturation exhibits voids. **B,** Maxillary central incisors. Maxillary right central incisor exhibits a lack of density and taper. Maxillary left central incisor has voids and unfilled canal space. **C,** Mandibular left first molar with adequate obturation; provisional restoration shows poor adaptation on the distal because of the failure to remove caries.

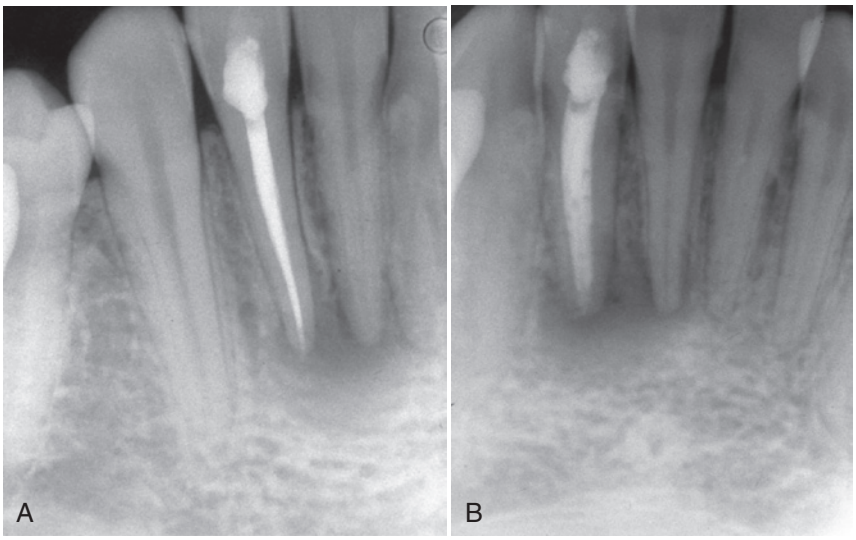

FIG. 7-2 **A,** Posttreatment radiograph of a mandibular left lateral incisor with ostensibly adequate obturation. **B,** Angled view reveals voids.

restoration has been inserted for more than three months, solely because of the suspicion of microleakage.[195] One may consider replacing the temporary restoration with a new coronal restoration immediately and to observe the tooth for at least three months before placing a permanent crown.[195]

Three-dimensional obturation of the radicular space is essential to long-term success. The canal system should be sealed apically, coronally, and laterally. Various methods have been advocated for obturation. Unfortunately, all materials and techniques result in some degree of leakage.[426] Although a poorly obturated canal and leakage are correlated, radiographic evaluation of obturation does not correlate well with leakage.[154,199] An adequate two-dimensional radiographic appearance of the obturation may not correlate with an adequate seal (Fig. 7-2).[110] Variation in radiographic interpretation by the clinician, the overlying osseous structures, and the lack of uniformity in the obturation materials are significant variables.[36,106,107,199,388]

The diagnostic outcome of root canal treatment is based on clinical and radiographic findings. In a series of prospective

studies, the Toronto group evaluated success and failure of root canal treatment at 4 to 6 years after completion of treatment. For primary root canal treatment,[91,121,127,241] teeth with pre-existing apical periodontitis were found to have a lower healing rate (82%) compared with teeth without periapical radiolucency (93%). Better outcomes were associated with teeth without periapical radiolucency, with single roots, and without mid-treatment complications such as root perforation. Teeth were treated by using flared preparation and vertical compaction of warm gutta-percha or step-back preparation and lateral compaction. Differences were noted with the adequacy of the fill and the treatment technique. Adequate length had a higher success rate (87%) when compared with inadequate length (77%). The flared preparation and vertical compaction had a higher success rate (90%) when compared with step-back preparation and lateral compaction (80%). For secondary non-surgical root canal treatment,[92,120] teeth with pre-existing apical periodontitis were also found to have a lower healing rate (80%) compared with teeth without periapical radiolucency (93%). Better outcomes were achieved in teeth with inadequate previous root filling, without perforation and radiolucency. Similar results were reported in a more recent prospective clinical study that examined the effects of primary or secondary root canal treatment on periapical healing.[262] The percentage of roots with complete periapical healing after primary (83%) or secondary (80%) root canal treatment was similar. Absence of preoperative apical periodontitis was identified as one of the prognostic factors affecting primary or secondary root canal treatment. In the presence of apical periodontitis, treatment prognosis was significantly improved by the presence of a smaller lesion. These studies are in agreement with earlier work[364,374] indicating preexisting apical pathosis as a major factor reducing a favorable prognosis and highlighted obturation technique as a factor influencing success and failure.[364]

Although periapical radiographs have been used to examine healing of post-treatment apical periodontitis since 1922,[264] their suitability for evaluating the outcomes of root canal treatment has recently been challenged, in that previously reported outcomes based on this assessment modality may have overestimated the healing rates of apical periodontitis.[97,118,423] Imaging with periapical radiographs produces a two-dimensional superimposition of a three-dimensional structure; partially healed periapical lesions that are confined within the cancellous bone alone are not usually detected using periapical radiographs.[32] With the advent of cone-beam computed tomography (CBCT), a better diagnosis of periapical lesions within the cancellous bone can be made. This is because CBCT softwares create reconstructed images from slices of data in any plane and location of the volume of interest, thus eliminating the lack of three-dimensional assessment and anatomical noise which hampers the accuracy of periapical radiography. This results in a higher signal-to-noise ratio and image contrast, and improves the detection of periapical radiolucencies.[283,335] Although the use of CBCT may not be practical for every clinical case, the significance in adopting this novel imaging technology for assessing the healing of apical periodontitis after primary root canal treatment can be demonstrated in a recent one-year follow-up study.[284] Diagnosis using CBCT revealed a lower healed and healing rate for primary root canal treatment compared with the use of digital radiography. Complete resolution of periapical radiolucency was found to be 93% using periapical radiography and 74% for CBCT. For those teeth with preexisting periapical radiolucency, reconstructed CBCT images also showed more failures (14%) when compared with the use of periapical radiographs (10%). In another follow-up study, the technical quality of root canal fillings and the associated treatment outcome were evaluated two years post-treatment, using both periapical radiography and CBCT.[226] Complete absence of post-treatment periapical radiolucency was observed in CBCT scans in 81% and 49% of adequate and inadequate root fillings, respectively, as compared to 87% and 61% revealed by periapical radiographs. Preoperative apical periodontitis and the quality of root filling were identified by both periapical radiographs and CBCT as significant prognosis predictors. The combined use of periapical radiography and CBCT imaging confirmed that satisfactory root fillings were associated with a favorable outcome. The results of this work emphasized that developing good technical skills for obturating root canals should not be underscored, and that suitable canal obturation methods should be developed to achieve more compacted, void-free filling materials and at the correct length.

HISTORICAL PERSPECTIVES

The achievement of a "hermetic seal" is often cited as a major goal of root canal treatment. According to accepted dictionary definitions, the word *hermetic* means sealed against the escape or entry of *air*—or made airtight by fusion or sealing. However, root canal seals are commonly evaluated for *fluid* leakage—a parameter used to praise or condemn obturation materials and techniques. This occurs both apically and coronally. Somehow, the term *hermetic* has crept into endodontic nomenclature in a manner probably quite similar to the invention of an airtight seal. A god of wisdom, learning, and magic in ancient Egypt, Thoth, better known as Hermes Trismegistus (Hermes thrice greatest), is credited with this invention.[343] His significant contribution to civilization allowed the preservation of oils, spices, aromatics, grains, and other necessities in previously porous, earthenware vessels. A simple wax seal of the vessel walls helped to create the "hermetic seal." Endodontically speaking, the term *hermetic* is inappropriate; instead, terms such as *fluid-tight, fluid-impervious,* or *bacteria-tight* seals are more contemporary.

In 1924, Hatton indicated: "Perhaps there is no technical operation in dentistry or surgery where so much depends on the conscientious adherence to high ideals as that of pulp canal filling."[162] The essence of this statement had been significantly influenced by years of trial and error in both the techniques and materials used to obturate the prepared root canal system. Much of the frustration and challenge that emanated from this concern, however, was due to the lack of development in root canal preparation techniques coupled with indictments of the "focal infection" craze of that era.[178]

Before 1800, root canal filling, when done, was limited to gold. Subsequent obturations with various metals, oxychloride of zinc, paraffin, and amalgam resulted in various degrees of success and satisfaction.[203] In 1847, Hill developed the first gutta-percha root canal filling material known as "Hill's stopping."[203] The preparation, which consisted principally of bleached gutta-percha and carbonate of lime and quartz, was patented in 1848 and introduced to the dental profession. In 1867, Bowman made claim (before the St. Louis Dental Society) of the first use of gutta-percha for canal filling in an extracted first molar.[12]

References to the use of gutta-percha for root canal obturation before the turn of the twentieth century were few and vague. In 1883, Perry claimed that he had been using a pointed gold wire wrapped with some soft gutta-percha (the origin of the present-day core carrier technique?).[289] He also began using gutta-percha rolled into points and packed into the canal. The points were prepared by cutting base plate gutta-percha into slender strips, warming them with a lamp, laying them on his operating case, and rolling them with another flat surface (a contemporary technique still used by a few to custom roll a large cone?). Perry then used shellac warmed over a lamp and rolled the cones into a point of desired size, based on canal shape and length. Before placing the final gutta-percha point, he saturated the tooth cavity with alcohol; capillary attraction let the alcohol run into the canal, softening the shellac so that the gutta-percha could be packed (the forerunner of a chemical-softening technique?).

In 1887, the S.S. White Company began to manufacture gutta-percha points.[193] In 1893, Rollins introduced a new type of gutta-percha to which he added vermilion.[405] Because vermilion is pure oxide of mercury and therefore dangerous in quantity, many people justifiably criticized this technique.

With the introduction of radiographs for the assessment of root canal obturations, it became painfully obvious that the canal was not cylindrical, as earlier imagined, and that additional filling material was necessary to fill the observed voids. At first, hard-setting dental cements were used, but these proved unsatisfactory. It was also thought that the cement used should possess strong antiseptic action, hence the development of many phenolic or formalin-type paste cements. The softening and dissolution of the gutta-percha to serve as the cementing agent, through the use of resins was introduced by Callahan in 1914.[60] Subsequently a multitude of various pastes, sealers, and cements were created in an attempt to discover the best possible sealing agent for use with gutta-percha.

Over the past 70 to 80 years, the dental community has seen attempts to improve on the nature of root canal obturation with these cements and with variations in the delivery of gutta-percha to the prepared canal system. During this era, the impetus for these developments was based heavily on the continued belief in the concept of focal infection, elective localization, the hollow-tube theory, and the concept that the primary cause for failure of root canal treatment was the apical percolation of fluids, and microorganisms, into a poorly obturated root canal system.[102,317] From this chronological perspective of technical and scientific thought, this chapter clarifies and codifies contemporary concepts in the obturation of the cleaned and shaped root canal system.

TIMING OF OBTURATION

Factors influencing the appropriate time to obturate a tooth include the patient's signs and symptoms, status of the pulp and periradicular tissue, the degree of difficulty, and patient management.

Vital Pulp Tissue

At present, the consensus is that one-step treatment procedures are acceptable when the patient exhibits a completely or partially vital pulp.[123,328,372] Removal of the normal or inflamed pulp tissue and performance of the procedure under aseptic conditions should result in a successful outcome because of the relative absence of bacterial contamination. Obturation at the initial visit also precludes contamination as a result of leakage during the period between patient visits.

Elective root canal treatment for restorative reasons can be completed in one visit provided the pulp is vital, to some degree, and time permits. Obturation of root canals in patients whose condition is urgent depends on the pretreatment diagnosis. When pain occurs as the result of irreversible pulpitis, obturation can occur at the initial visit because removal of the vital tissue will generally resolve the patient's pain.

Necrotic Pulp Tissue

Patients who present with pulp necrosis with or without asymptomatic periradicular pathosis (asymptomatic apical periodontitis, chronic apical abscess, condensing osteitis) may be treated in one visit, based on the best available information. When patients present with acute symptoms caused by pulp necrosis and acute periradicular abscess, obturation is generally delayed until the patient is asymptomatic. However, more than 20 years ago, investigators demonstrated that cases with soft-tissue swelling could be completed in one visit with appropriate endodontic treatment, incision for drainage, and a regimen of antibiotics.[366] Management of these patients, however, may be more difficult should problems persist or become worse after the completion of treatment.

During the 1970s, there was concern about the timing of obturation. Performing endodontic treatment in one visit was controversial. Conventional wisdom suggested that patients would have a higher incidence of posttreatment pain. However, recent clinical studies[252,285,292] and systematic reviews[123,328,372] indicate that there is no significant difference in the healing rates of apical periodontitis between single-visit and multiple-visit root canal treatment. Patients experience less frequency of short-term post-obturation pain after single-visit than those having multiple-visit root canal treatment.[372]

In contrast to teeth with vital pulp tissue, teeth exhibiting pulp necrosis frequently exhibit bacterial contamination and may require a different approach to treatment. Sjögren and colleagues raised questions regarding the long-term prognosis of teeth exhibiting necrotic pulp tissue and apical periodontitis treated in a single-visit.[363] In their clinical study, the authors thoroughly instrumented 55 infected teeth with apical pathosis, using only 0.5% sodium hypochlorite (NaOCl) [*Editor's note:* Today, stronger concentrations of NaOCl are more commonly used. The reader is referred to Chapter 14 for a fuller discussion of this issue]. Before obturation, cultures were obtained, using anaerobic bacteriologic techniques. After cleaning and shaping, bacteria could be detected in 22 teeth. Complete healing occurred in 94% of cases that yielded a negative culture, whereas the rate of successful treatment of teeth with positive cultures before obturation was 68%, a statistically significant difference.

With the introduction of the concept of biofilms, other investigators examined the intracanal microbial status of sixteen mesial roots of human mandibular first molars with primary apical periodontitis immediately after one-visit root canal treatment.[258] In that study, the instrumented canals were irrigated with 5.25% NaOCl and 17% ethylenediaminetetraacetic acid (EDTA), and obturated with gutta-percha and zinc oxide–eugenol cement. The apical portion of the root of each tooth was removed by flap-surgery and prepared for correlative light and transmission electron microscopy

examination. Fourteen of the 16 root-treated teeth revealed residual intracanal infection after instrumentation, antimicrobial irrigation, and obturation. The microbes existed mostly as biofilms in inaccessible recesses of instrumented main canals, the intercanal isthmus, and accessory canals.

In a more recent histobacteriological study, the in vivo microbiological status of the mesial roots of mandibular molars with primary apical periodontitis was examined after single-visit or two-visit root canal treatment.[397] Those roots were instrumented and irrigated with 5% NaOCl, 17% EDTA, and 2% chlorhexidine. In the single-visit group, the canals were immediately obturated, whereas in the two-visit group, calcium hydroxide dressing was placed in the canals for one week prior to obturation. In the single-visit group, no canal, out of the six canals examined, was completely free of bacteria. Residual biofilms were identified in the main canal, isthmus, apical ramifications, and dentinal tubules. In the two-visit group, two out of seven roots were rendered bacteria-free. Residual biofilms were found predominantly in the isthmus and ramifications, intermixed with necrotic tissues and debris. Taken together, these 3 studies illustrate that microbes are extremely difficult to eliminate within the complex root canal system, and that the use of an inter-appointment antimicrobial dressing resulted in improved microbiological status of the root canal system when compared with the single-visit protocol.

Calcium hydroxide has been advocated as an antimicrobial and temporary dressing in necrotic cases that cannot be treated in one visit[363] because investigators noted that bacteria in instrumented, unfilled canals can multiply and reach their pretreatment numbers in 2 to 4 days.[57] Nevertheless, the ability of calcium hydroxide to completely eradicate microbial species from the root canal system has recently been questioned. In vitro studies demonstrated that the antibacterial activity of calcium hydroxide can be inactivated by dentin.[156,297] Other clinical studies showed that the number of bacteria-positive canals did not decrease after the use of calcium hydroxide as an inter-appointment dressing.[291,401] A systematic review and meta-analysis of 8 clinical studies concluded that calcium hydroxide is useful but has limited effectiveness in completely eliminating bacteria from human root canals when assessed by culture techniques.[330] Although calcium hydroxide has a wide range of antimicrobial activity against common endodontic pathogens and is an effective anti-endotoxin agent, it is less effective against *Entercoccus faecalis* and *Candida albicans*.[251] Alternative inter-appointment dressings such as the use of chlorhexidine gel, calcium hydroxide in chlorhexidine gel, or triantibiotic paste (metronidazole, minocycline, and ciprofloxacin) has been proposed, with dichotomous results.[95,271,361,403] While there are clinical trials to support the use of calcium hydroxide or alternative inter-appointment dressings, they do not reduce bacteria load beyond levels already achieved by canal preparation with NaOCl.[238,240,360] It is fair to say inter-appointment antimicrobial dressings are fairly well accepted by clinicians in cases that cannot be treated in a single visit.

In general, obturation can be performed after cleaning and shaping procedures when the canal can be dried and the patient is not experiencing swelling. An exception is the presence or persistence of exudation from the canal. Obturation of a canal that cannot be dried is contraindicated. Procedural concerns also dictate the time of obturation. Difficult cases may require more time for preparation and can be managed more uneventfully in multiple appointments. Patients may require multiple short appointments because of medical conditions, their psychological state of mind, and fatigue.

LENGTH OF OBTURATION

One of the controversies in endodontics that remains unresolved is the apical limit of root canal treatment and obturation.[33,309] Early studies identified the dentinocemental junction as the apical limit for obturation. However, this histologic landmark cannot be determined clinically, and it has been found to be irregular within the canal. The dentinocemental junction may be several millimeters higher on the mesial canal wall when compared with the distal wall. In addition, the dentinocemental junction does not coincide with the narrowest portion of the canal or apical constriction. The reader is referred to Chapter 5 for more information about this anatomy.

Traditionally, the apical point of termination has been approximately 1 mm from the radiographic apices as determined by radiographs. Kuttler noted that the apical anatomy consists of the major diameter of the foramen and the minor diameter of the constriction (Fig. 7-3),[212] with the apical constriction identified as the narrowest portion of the canal. The average distance from the foramen to the constriction was found to be 0.5 mm, with the foramen varying in distance from the apex up to 2.5 mm. Kuttler also noted that the foramen-to-constriction distance increases with age because of cementum deposition. Supporting this finding, other investigators found that the location of the foramen was not at the apex. Deviations occurred in 92% of the roots and averaged 0.6 mm.[56] Another study noted the average apex-to-constriction distance was 0.9 mm and that 95% of the constrictions were between 0.5 and 1 mm in diameter[108]; this study also noted that the classic apical anatomy described by Kuttler was present in only 46% of the teeth. Other variations identified were the tapering constriction, the multiconstriction, and the parallel constriction. Other investigators examined 230 roots of permanent teeth stereomicroscopically and with radiographs.[37] Results of this study indicated a deviation of the foramen from the apex in 76% of the roots with microscopy and 57% with radiography; the mean distance was 1 mm.

A later study found that no foramina coincided with the longitudinal axis of the root, with the distance ranging from 0.2 to 3.8 mm (Fig. 7-4).[152] Root resorption is an additional factor in length determination. Resorption is more common

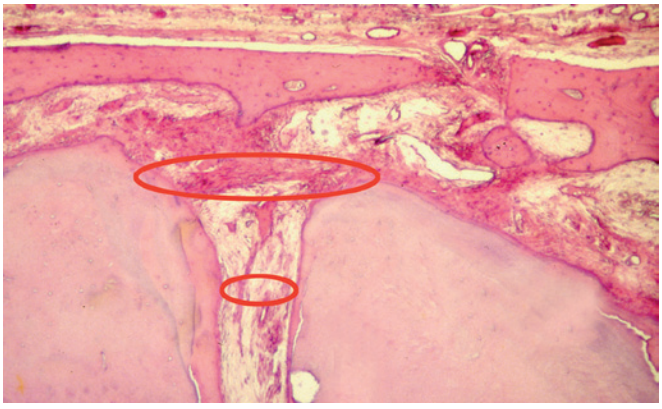

FIG. 7-3 Histologic section of a root apex, demonstrating anatomy of the classic foramen and constriction.

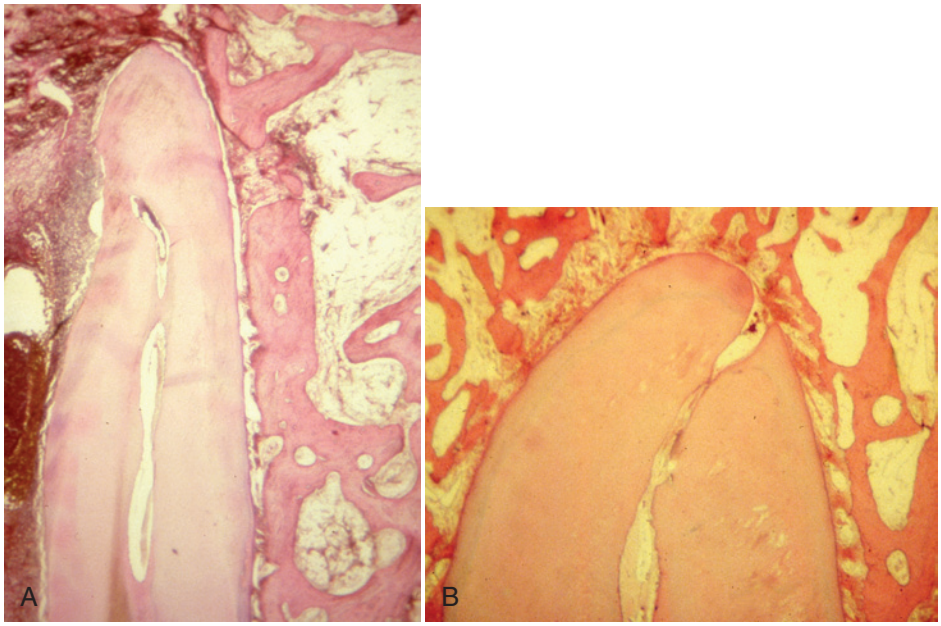

FIG. 7-4 Histologic sections demonstrating the foramen exiting short of the root apex.

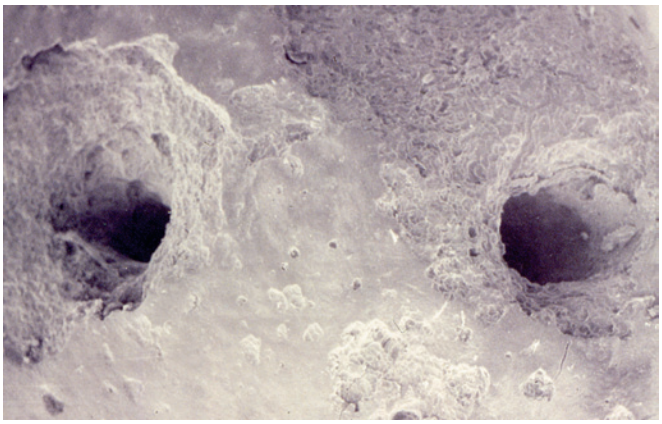

FIG. 7-5 Scanning electron microscopy of a tooth exhibiting a necrotic pulp and apical pathosis and resorption.

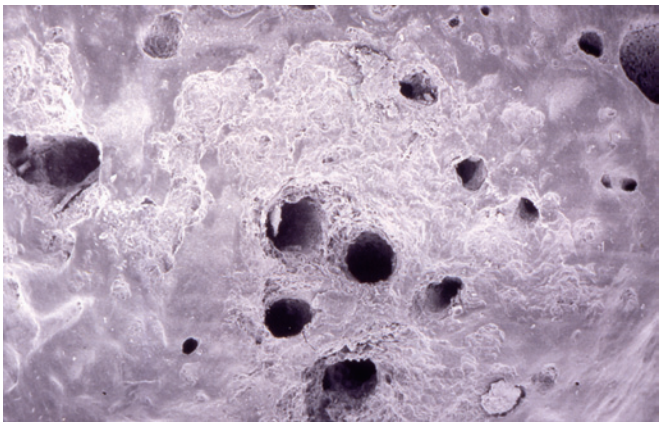

FIG. 7-6 Scanning electron microscopy of the apex of an extracted tooth that was removed because of pulp necrosis. Note the multiple accessory foramina and resorption.

with necrosis and apical bone resorption, and this can result in loss of the constriction (Fig. 7-5).[122,239] On the basis of these findings it appears that canals filled to the radiographic apex reflect an overextension of the obturating material. If overextension occurs that cannot be retrieved, and there appears to be resultant nerve damage, the practitioner is obligated to refer the patient to a qualified practitioner skilled in cases of this type.[139]

A study by the Toronto group on the prognosis of retreatment identified perforation,[92] pretreatment periradicular disease, and adequate length of the root canal filling as factors significantly influencing success and failure. The authors speculated that canals filled more than 2 mm short harbored necrotic tissue, bacteria, and irritants that when re-treated could be cleaned and sealed. The success rate for negotiating the apical unfilled canal was 74%.

Controversy also exists regarding the role accessory canals play in success and failure (Fig. 7-6). A scanning electron microscopy (SEM) study of the apical anatomy of each tooth group except the third molars noted no pattern for foraminal openings[151]; the number of accessory canals ranged from 1 to 16. Although lateral canals can be associated with pathosis, one study that examined root-treated teeth from human cadavers reported no relationship between unfilled lateral canals and periradicular pathosis.[20]Accessory/lateral canals are often obturated by chance and only serendipitously identified on the posttreatment radiograph (Fig. 7-7).

A later histobacteriological study also found no evidence to support that lateral canals must be filled to achieve a successful long-term treatment outcome (Fig. 7-8).[313] In cases with vital pulps, forcing obturation materials into lateral canals resulted in unnecessary damage to the periradicular tissues with consequential inflammation. In cases with nonvital, infected pulps in which lateral canals appeared radiographically filled, they were actually not sealed or disinfected, and the remaining tissue in the ramification was inflamed and enmeshed with the filling material. This, however, does not mean that lateral canals should not be optimally debrided and disinfected by

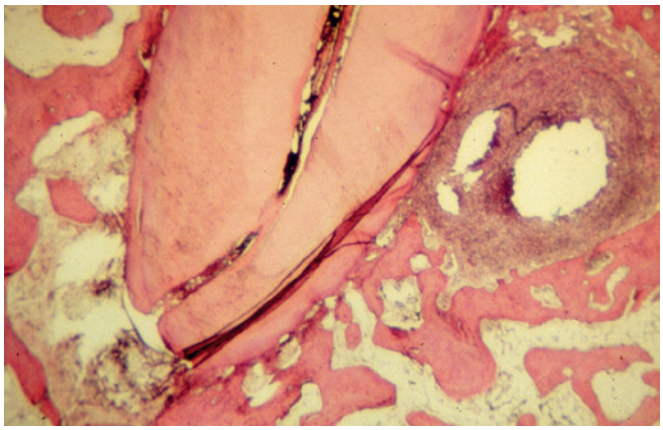

FIG. 7-7 Histologic section of a mesial root of a mandibular molar with a lateral canal present and associated lesion. Will the lesion resolve after the removal of the main canal contents, or will the lesion persist because of necrotic pulpal remnants in the lateral canal? The question remains unanswered.

FIG. 7-8 Posttreatment radiograph of a mandibular right first molar with a lateral canal associated with the distal root.

contemporary irrigant delivery and agitation techniques to reduce microbes and/or biofilms that reside within those spaces, prior to obturation of the canal space with root-filling materials and sealers.

The importance of length control in obturation relates to extrusion of root-filling materials. In a quantitative systematic review of the literature,[338] the authors critically analyzed 12 outcome studies that fulfilled the following criteria: i) a minimum follow-up period of two years; ii) data available on termination of obturation/instrumentation; iii) adequate definition of treatment failure; iv) data available on success/failure of root canal therapy in relation to the obturation/instrumentation length; and v) presence or absence of periapical radiolucency. The 12 studies were classified into three categories based on the obturation length from the radiographic apex: A) 0-1 mm; B) >1 mm but <3 mm; and C) obturated past the radiographic apex, including sealer. From the 12 studies, only four studies that included data that could be placed into the three length categories were further subjected to meta-analysis. Results of the meta-analysis indicated that the success rate in group A (obturated 0-1 mm from apex) was marginally better than group C (obturated past apex) by 29%. Although group A had better success than group B (obturated >1 mm short), the difference was not statistically significant. The authors concluded that a better success rate was achieved when treatment involved obturation short of the apex.

The aforementioned results derived from the sophisticated meta-analysis of multiple publications highlighted the data presented in the classic work performed by Sjögren and coworkers on healing of root-treated teeth with necrotic pulps and periapical lesions 8 to 10 years after treatment.[364] When those teeth were filled within 2 mm of the apex, 94% revealed normal periapical conditions with the use of periapical radiography at the follow-up examination. Conversely, roots with excess root fillings and those with fillings more than 2 mm short of the apex had significantly lower success rates of 76% and 68%, respectively.

Similar results were also reported in a prospective clinical study on the healing[262] and survival[263] of teeth that had undergone primary or secondary root canal treatment for 2 to 4 years. Absence of root-filling extrusion was found to be a highly significant postoperative prognostic factor affecting the success of both primary and secondary root canal treatment.[262] With respective to tooth survival, extrusion of gutta-percha root filling did not have any effect on tooth survival within the first 22 months, but significantly increased the risk of tooth loss beyond 22 months.[263] This prognostic factor was common to both primary and secondary root canal treatment. The authors attributed the delayed effect of extrusion of gutta-percha root filling on tooth survival to the possibility of subcritical cracks created by excessive forces during compaction of gutta-percha, which eventually propagated during function and ultimately resulted in catastrophic failure.

On the basis of biologic and clinical principles, the conclusions derived from studies performed for more than two decades apart are all agreeable, in that instrumentation and obturation should not extend beyond the apical foramen.[259] This was also demonstrated in a histologic study which evaluated 41 human root-filled teeth from 36 patients.[312] In six cases exhibiting overfills, histologic examination revealed severe inflammation.

Whereas the guideline of 1 mm from the radiographic apex remains rational when using radiographs, the point of apical termination of the preparation and obturation remains empirical. The use of an apex locator in conjunction with radiographs and sound clinical judgment makes this decision more logical. The need to compact the gutta-percha and sealer against the apical dentin matrix (constriction of the canal) is necessary to prevent extrusion of materials into the periapical tissues. Deciding where the apical constriction of the canal lies is based on the clinician's basic knowledge of apical anatomy, tactile sensation, radiographic interpretation, apex locators, apical bleeding, and (if not anesthetized) the patient's response.

PREPARATION FOR OBTURATION

During the cleaning and shaping process, organic pulpal materials and inorganic dentinal debris accumulate on the canal wall, producing an amorphous irregular smear layer (Fig. 7-9).[25,244,281] As shown in one of these studies, the smear layer is superficial with a thickness of 1 to 5 μm.[244] This superficial debris can be packed into the dentinal tubules to various distances.[1]

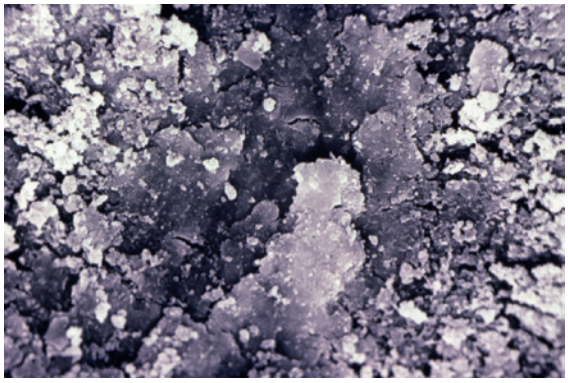

FIG. 7-9 Scanning electron microscopy of a prepared canal wall. The tubules are covered with a smear layer of organic and inorganic material.

In cases of necrosis this layer may also be contaminated with bacteria and their by-products. For example, one study found that bacteria can extend 10 to 150 μm into the dentinal tubules of necrotic teeth.[346] Another study noted that capillary action and fluid dynamics play a role in packing debris into the tubules.[3] Another investigation noted a mean penetration of 479 μm after a 28-day incubation period.[287]

The smear layer is not a complete barrier to bacteria but may act as a physical barrier, decreasing bacterial penetration into tubules. This was illustrated by a study demonstrating that removal of the smear layer permitted colonization of the dentinal tubules at a significantly higher rate when compared with leaving the smear layer in place.[104]

The smear layer may also interfere with adhesion and penetration of sealers into dentinal tubules.[410] Evidence indicates that sealer penetration into dentinal tubules does not occur when the smear layer is present.[132,277] For example, one study found that removal of the smear layer permitted Roth 811 (Roth International, Ltd., Chicago, IL), Calciobiotic root canal sealer (CRCS; Coltène/Whaledent, Cuyahoga Falls, Ohio), and Sealapex (SybronEndo, Orange, California) to penetrate to between 35 and 80 μm, whereas the presence of the smear layer obstructed tubular penetration of all sealers.[208] Other studies found that smear layer removal increased bond strength and reduced microleakage in teeth obturated with AH-26 (DENTSPLY Maillefer, Ballaigues, Switzerland).[111,133] Another investigation found that a combination of smear layer removal, AH-26 as the sealer, and vertical compaction of gutta-percha had a cumulative effect in reducing leakage.[386]

There does not appear to be a consensus on removing the smear layer before obturation.[70,347,353] The advantages and disadvantages of the smear layer remain controversial; however, growing evidence supports removal of the smear layer before obturation.[177,353] The organic debris present in the smear layer might constitute a substrate for bacterial growth.[281] It has been suggested that the smear layer prohibits sealer contact with the canal wall and permits leakage.[29] Bacterial penetration in the presence of a smear layer in canals obturated with thermoplasticized gutta-percha and sealer has been shown to be significantly higher than with smear layer removal before obturation.[281] An additional consideration is the presence of viable bacteria that remain in the dentinal tubules and use the smear layer for sustained growth and activity.[49] Removal of the smear layer introduces the possibility of reinfecting the dentinal tubules if leakage occurs.[347] However, one study demonstrated that bacteria present before obturation are not viable after obturation.[99]

The smear layer may also interfere with the action of irrigants used as disinfectants.[272] When the smear layer is not removed, it may slowly disintegrate and dissolve around leaking obturation materials, or it may be removed by bacterial by-products such as acids and enzymes.[347]

The smear layer may interfere with the adhesion and penetration of root canal sealers. It also may prevent gutta-percha penetration during thermoplastic techniques.[155] Significant tubular penetrations of gutta-percha and sealers have been reported with thermoplasticized obturations[155] and with dentin-bonded composite resins.[221] Removal of the smear layer also enhances the adhesion of sealers to dentin and tubular penetration.[221,267,347,409] Root canal filling materials adapt better to the canal walls after smear layer removal.[101,267,409,410,411]

One investigation examined the penetration depth of three different root canal sealers into the dentinal tubules with and without the smear layer. Scanning electron microscopy of extracted single-rooted human teeth obturated by lateral compaction of gutta-percha, using AH Plus (DENTSPLY Maillefer), Apexit (Ivoclar Vivadent, Schaan, Liechtenstein), and Roth 811, demonstrated that the smear layer prohibited the sealers from penetrating dentinal tubules. Smear layer removal allowed the penetration of all sealers to occur to various depths.[205] Another study found that removal of the smear layer reduced both coronal and apical leakage regardless of the sealer tested.[79]

Another study examined the smear layer and the passage of bacteria through and around obturating materials,[77] using human maxillary incisors obturated with gutta-percha and AH-26. The teeth were exposed to standardized bacterial suspensions containing *Fusobacterium nucleatum, Campylobacter rectus,* and *Peptostreptococcus micros* for a period of 60 days, using a leakage model employing upper and lower chambers. Results indicated that 60% of the samples in which the smear layer was not removed demonstrated bacterial leakage. There was no leakage in specimens from which the smear layer was removed.

An additional method for removing the smear layer involves sonic and ultrasonic instruments. In early studies of ultrasonic instrumentation, investigators noted the technique was effective in removing the smear layer.[84] Another investigator also demonstrated smear layer removal with ultrasonication and NaOCl.[63] One study compared the cleaning efficacy of short-term sonic and ultrasonic passive irrigation with 5.25% NaOCl after hand instrumentation in the apical 3 to 6 mm of maxillary molar root canals.[319] Passive sonic or ultrasonic irrigation for 30 seconds resulted in significantly cleaner canals than hand filing alone, and ultrasonic irrigation produced significantly cleaner canals than irrigation. However, other studies found ultrasonication and NaOCl to be ineffective in removing the smear layer.[24,400] In a more recent study, a group of investigators reported that the use of the Vibringe, EndoActivator, or needle irrigation did not significantly improve sealer penetration when compared with conventional irrigation.[43]

After the completion of cleaning and shaping procedures, removal of the smear layer is generally accomplished by irrigating the canal with 17% disodium ethylenediaminetetraacetic acid (EDTA) and 5.25% NaOCl (Fig. 7-10).[25] Chelators remove the inorganic components, leaving the organic tissue elements intact. Sodium hypochlorite is necessary for removal of the

FIG. 7-10 Scanning electron microscopy of the canal wall after removal of the smear layer with 17% EDTA and 5.25% sodium hypochlorite.

remaining organic components. Citric acid has also been shown to be an effective method for removing the smear layer,[16,23,170,337] as has tetracycline.[19,164]

Chelating agents were introduced to endodontic treatment by Nygaard-Østby in 1957 for treatment of calcified narrow root canals.[266] Ethylenediaminetetraacetic acid is the chelating solution customarily used in endodontic treatment. It is available in both liquid and paste forms with common concentrations between 15% and 17%.[177]A detergent is frequently added to the liquid to decrease surface tension, to increase the cleaning ability, and to enhance the bactericidal action of the solution.[399] The effectiveness of EDTA is related to time of application, the pH, and the concentration.[254,266]

Demineralization results in increased dentin permeability[150] because of the removal of the smear layer and plugs and enlargement of the tubules. It appears that the tubular enlargement is due to selective removal of the peritubular dentin.[175] The action of chelators and acids appears to be more effective in the coronal and middle thirds of the root and is reduced apically.[177,228] This reduced activity may be a reflection of canal size.[210] This is a clinical concern because of the more irregular structure of dentin in the apical third. Another investigation demonstrated marked variations in the apical portion of the root,[250] including accessory root canals, areas of resorption and repaired resorptions, pulp stones, irregular or absent primary tubules, irregular secondary dentin, and cementum-like tissue lining the apical root canal wall. The variable structure of the apical region of human teeth presents challenges to the use of endodontic obturation techniques requiring adhesives, because this may influence the dentin bonding ability in the apical region.[250]

Ethylenediaminetetraacetic acid appears to be biocompatible when used clinically[266]; however, irreversible decalcification of periapical bone and neuroimmunologic disturbances have been noted.[342] Extrusion of both NaOCl and EDTA in clinical treatment should be avoided.[163,282,380]

The recommended time for removal of the smear layer is 1 to 5 minutes.[62,177,337] The small particles of the smear layer are primarily inorganic with a high surface-to-mass ratio that facilitates removal by acids and chelators. Investigators have found that a 1-minute exposure to 10 mL of EDTA was adequate to remove the smear layer and that a 10-minute exposure caused excessive removal of both peritubular and intratubular dentin.[62]

The use of EDTA in combination with NaOCl is recommended[371,381] and may enhance the cleaning[228] and antimicrobial effects of these solutions when compared with using them alone.[58] Although the use of EDTA alone does not cause erosion of the canal wall dentin, it should be noted the adjunctive use of NaOCl with EDTA may result in erosion of the intraradicular dentin, depending on the timing and the concentrations of irrigants employed.[236,300,345,433]

New smear layer–removing irrigants are commercially available that combine calcium chelation of the inorganic component of the smear layer with antimicrobial activities. Examples of these irrigants include BioPure MTAD (DENSPLY Tulsa Dental Specialties)[349] and QMix 2in1 Irrigating Solution (DENSPLY Tulsa Dental Specialties).[86]

THE IDEAL ROOT CANAL FILLING

Various endodontic materials have been advocated for obturation of the radicular space. Most techniques employ a core material and sealer. Regardless of the core material a sealer is essential to every technique and helps achieve a fluid-tight seal.

The American Association of Endodontists' *Guide to Clinical Endodontics* outlines contemporary endodontic treatment.[8] Nonsurgical root canal treatment of permanent teeth "involves the use of biologically acceptable chemical and mechanical treatment of the root canal system to promote healing and repair of the periradicular tissues." The process is accomplished under aseptic conditions with rubber dam isolation. Regarding obturation, the guide states, "Root canal sealers are used in conjunction with a biologically acceptable semi-solid or solid obturating material to establish an adequate seal of the root canal system." In this area the guidelines indicate that "Paraformaldehyde-containing paste or obturating materials have been shown to be unsafe. Root canal obturation with paraformaldehyde-containing materials is below the standard of care for endodontic treatment" (Fig. 7-11). Chapter 29 gives further information about this issue.

Assessment of nonsurgical treatment is based primarily on the posttreatment radiographic examination. The radiographic criteria for evaluating obturation include the following categories: length, taper, density, gutta-percha and sealer removal to the facial cementoenamel junction in anterior teeth and to the canal orifice in posterior teeth, and an adequate provisional or definitive restoration (Fig. 7-12).

Quality assurance is accomplished through a careful evaluation of treatment procedures. Only by this approach can deficiencies be identified and corrected. Although the anatomy and morphology of the radicular space vary tremendously, the obturated root canal should reflect the original canal shape. Procedural errors in preparation, such as loss of length, ledging, apical transportation, apical perforation, stripping perforation, and separated instruments, may not be correctable. Errors in obturation, such as length, voids, inadequate removal of obturation materials, and temporization, may be correctable.

Radiographic interpretation may vary among clinicians because of differences in radiopacity in root canal sealer/cements, constituents in specific brands of gutta-percha, interpretation of voids in vivo versus in vitro,[431] the overlying bony anatomy, radiographic angulation, and the limited two-dimensional view of the obturated root canal or canals.

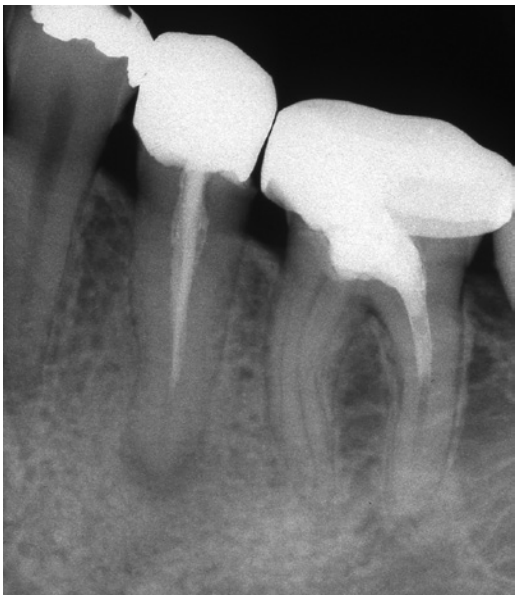

FIG. 7-11 A periapical radiograph of a mandibular left second premolar and first molar, demonstrating Sargenti paste root canal treatment. In addition to the toxic material, the technique often accompanies inadequate cleaning and shaping procedures.

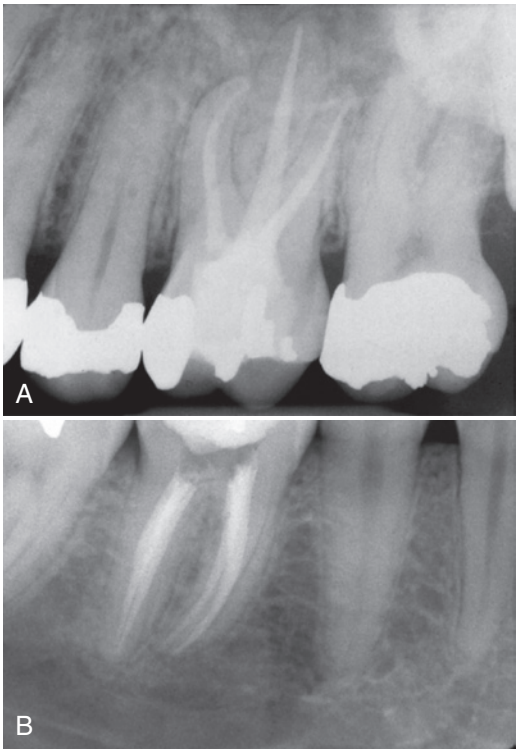

FIG. 7-12 A, Posttreatment radiograph of a maxillary right first molar, demonstrating adequate length, density, and taper. B, Posttreatment radiograph of a mandibular right first molar with an adequate obturation.

An often overlooked aspect in the assessment of root canal obturation is the density of the apical portion of the fill.[153] The apical third of the canal may be filled with a sea of root canal cement and a single master cone or poorly compacted mass of previously softened gutta-percha. Radiographically, the apical third of the canal appears less radiodense. An ill-defined outline to the canal wall is evident, along with obvious gaps or voids in the filling material or its adaptation to the confines of the canal. Because of the use of highly radiopaque root canal sealers/cements, the apical portion may be filled only with sealer, giving the clinician the false impression of a dense, three-dimensional obturation with gutta-percha.

Root canal sealers vary in radiopacity.[304,379] Some contain silver particles or significant amounts of barium sulfate to enhance their radiopacity. Although these components may enhance visualization of anatomic structures such as lateral canals, it is important to realize they do not increase the sealing ability of the sealer and the quality of the obturation. They may also give the impression that a canal is well obturated when voids are masked by the density of the sealer. It is erroneous to claim that obturations with highly radiopaque sealers are better than those made with less radiopaque materials. This type of comparison and claim to superiority are both unfounded and unwarranted. The radiographic appearance or aesthetic appearance of the obturated canal system should be secondary to meticulous cleaning and shaping. Although assessment of the root canal obturation is based on radiographic findings, root canal sealers do not have to be highly radiopaque to be effective.

BOX 7-1

Properties of an Ideal Sealer

- Exhibits tackiness when mixed to provide good adhesion between it and the canal wall when set
- Establishes a hermetic seal
- Radiopaque, so that it can be seen on a radiograph
- Very fine powder, so that it can mix easily with liquid
- No shrinkage on setting
- No staining of tooth structure
- Bacteriostatic, or at least does not encourage bacterial growth
- Exhibits a slow set
- Insoluble in tissue fluids
- Tissue tolerant; that is, nonirritating to periradicular tissue
- Soluble in a common solvent if it is necessary to remove the root canal filling

TYPES OF SEALERS

Root canal sealers are necessary to seal the space between the dentinal wall and the obturating core interface. Sealers also fill voids and irregularities in the root canal, lateral and accessory canals, and spaces between gutta-percha points used in lateral condensation. Sealers also serve as lubricants during the obturation process. Grossman outlined the properties of an ideal sealer (Box 7-1).[149] At present no sealer satisfies all the criteria.

Sealers should be biocompatible and well tolerated by the periradicular tissues.[367] All sealers exhibit toxicity when freshly mixed; however, their toxicity is greatly reduced on setting.[214] Sealers are resorbable when exposed to tissues and tissue fluids.[14] Tissue healing and repair generally appear unaffected by most sealers, provided there are no adverse breakdown products of the sealer over time.[42,50-52,54] Breakdown products from the

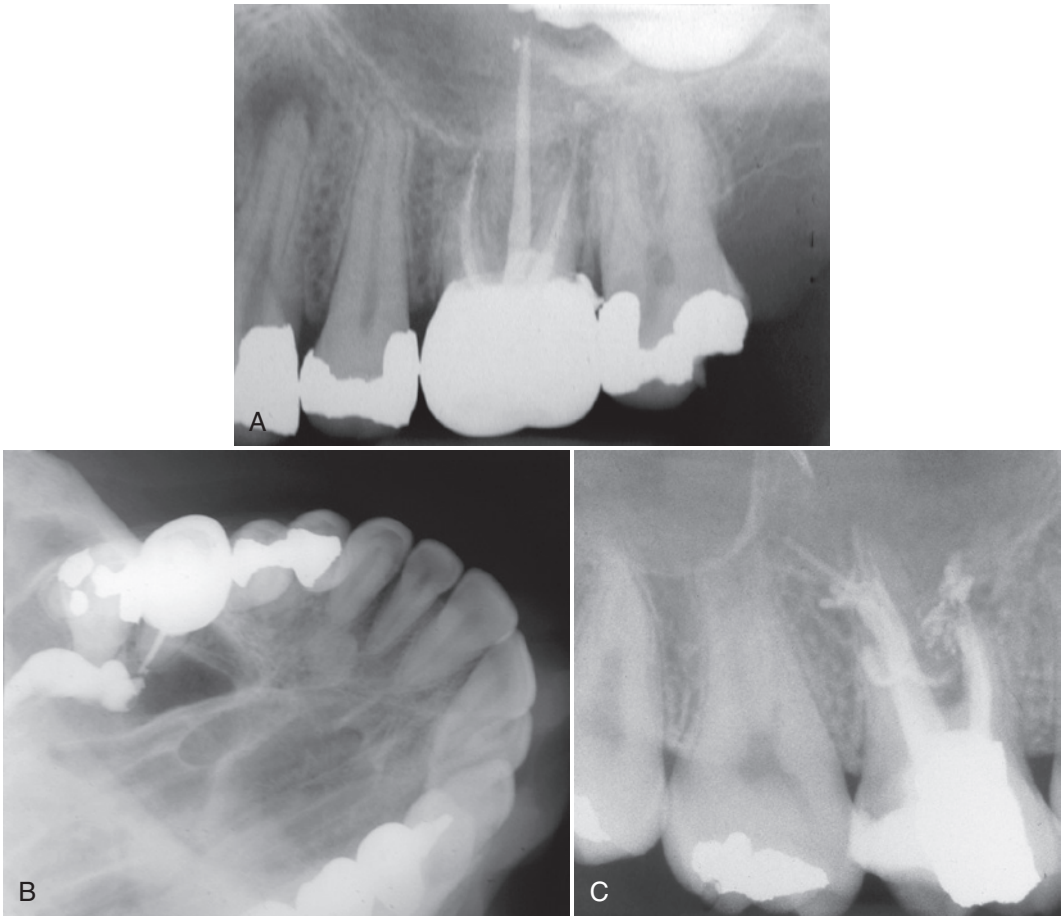

FIG. 7-13 **A,** Extrusion of sealer evident on this posttreatment radiograph of a maxillary first molar. The separated lentulo spiral in the mesiobuccal root indicates a possible method of sealer placement. **B,** Maxillary occlusal film demonstrates that the sealer is located in the maxillary sinus. Correction by nonsurgical techniques is not possible. **C,** Maxillary right first molar with extrusion of the sealer and gutta-percha.

sealers may have an adverse effect on the proliferative capability of periradicular cell populations.[146] As a result, sealers should not be placed routinely in the periradicular tissues as part of an obturation technique.[214] Although an osteogenic response has been observed with calcium hydroxide–based root canal sealers,[172,365,376,390] the ability of these sealers to sustain a high pH over time has been questioned.[207]

The most popular sealers are zinc oxide–eugenol formulations, calcium hydroxide sealers, glass ionomer sealers, resin-based (epoxy resin or methacrylate resin) sealers, and the recently introduced calcium silicate–based sealers. Despite claims by the manufacturers on the advantages of each class of sealers, there are no evidence-based data, based on randomized clinical trials, demonstrating the superiority of one class of sealer over another. Regardless of the sealer selected, all exhibit toxicity until they have set. For this reason, extrusion of sealers into the periradicular tissues should be avoided (Fig. 7-13).

Zinc Oxide and Eugenol

Zinc oxide–eugenol sealers have a history of successful use over an extended period of time. Zinc oxide–eugenol sealers will resorb if extruded into the periradicular tissues.[14] They exhibit a slow setting time,[6] shrinkage on setting,[192] solubility,[290] and they can stain tooth structure.[90,209,394] An advantage to this sealer group is antimicrobial activity.[4,18,167,249]

An early zinc oxide–eugenol sealer was introduced by Rickert and Dixon.[308] This powder/liquid sealer contained silver particles for radiopacity. Although it was possible to demonstrate the presence of lateral and accessory canals the sealer had the distinct disadvantage of staining tooth structure if not completely removed. Marketed as Pulp Canal Sealer (SybronEndo) and Pulp Canal Sealer EWT (extended working time), this sealer is popular with clinicians using thermoplastic techniques. Procosol (Procosol, Inc., Philadelphia, Pennsylvania) is a modification of Rickert's formula in which the silver particles have been removed (zinc oxide, hydrogenated resin, bismuth subcarbonate and barium sulfate; liquid eugenol).

Grossman modified the formulation and introduced a non-staining formula in 1958 (Table 7-1).[148] This is the formulation in Roth's Sealer (Roth International) Tubli-Seal (SybronEndo) is a catalyst/base zinc oxide–eugenol sealer that is convenient to mix but has a faster setting time when compared with the liquid/powder sealers. Tubli-Seal EWT provides an extended working time. Wach's Sealer (Balas Dental, Chicago, Illinois) contains Canada balsam, which gives the material a sticky or tacky property that softens the gutta-percha into a more homogeneous mass when used with lateral compaction.

Although zinc oxide–eugenol sealers possess marked cytotoxic and tissue-irrigating potencies in ex vivo cell culture

TABLE 7-1
Formula for Zinc Oxide–Eugenol Root Canal Sealer

Powder	Liquid
Zinc oxide	42 parts
Staybelite resin	27 parts
Bismuth subcarbonate	15 parts
Barium sulfate	15 parts
Sodium borate, anhydrous	1 part

studies,[134] their clinical usefulness has been well demonstrated in an in vivo animal model,[129] as well as in retrospective human clinical studies.[83] This discrepancy between the results of in vitro and in vivo cytotoxicity testing may be explained by the fact that most cell culture systems are represented by only one cell type (i.e. no cell-cell interactions) which is often monoclonal in origin. Another important issue to consider is the fact that culture conditions are not homeostatic and there is no elimination of toxic substances as there would be in vivo. By contrast, the human body possesses a lymphatic system and periapical defenses such as polymorphonuclear leukocytes, plasma cells, and macrophages to help eliminate toxic substances.[61] These mechanisms do not exist in a culture plate and must be taken into account for interpretations of the results of cell culture–based cytotoxicity studies reported in the endodontic literature.

Calcium Hydroxide Sealers

Calcium hydroxide sealers were developed for therapeutic activity. It was thought that these sealers would exhibit antimicrobial activity and have osteogenic–cementogenic potential. Unfortunately, these actions have not been demonstrated.[100,251] Solubility is required for release of calcium hydroxide and sustained activity. This is inconsistent with the purpose of a sealer. Calciobiotic root canal sealer (CRCS) is a zinc oxide–eugenol sealer with calcium hydroxide as one ingredient. Sealapex (SybronEndo) is a catalyst/base system. The base contains zinc oxide, calcium hydroxide, butyl benzene, sulfonamide, and zinc stearate. The catalyst contains barium sulfate and titanium dioxide as radiopacifiers in addition to resin, isobutyl salicylate, and aerosol R972. Apexit and Apexit Plus (Ivoclar Vivadent) consist of an activator (disalicylate, bismuth hydroxide/bismuth carbonate, and fillers) and a base (calcium hydroxide, hydrated colophonium [i.e., pine resin], and fillers).

Noneugenol Sealers

Developed from a periodontal dressing, Nogenol (GC America, Alsip, Illinois) is a root canal sealer without the irritating effects of eugenol. The base contains zinc oxide, barium sulfate, and bismuth oxychloride.

Glass Ionomer Sealers

Glass ionomers have been advocated for use in obturation because of their dentin-bonding properties. Ketac-Endo (3M ESPE, St. Paul, Minnesota) enables adhesion between the material and the canal wall.[128] It is also difficult to properly treat the dentinal walls in the apical and middle thirds with conditioning agents to receive the glass ionomer sealer. A disadvantage of glass ionomers is that they must be removed if retreatment is required.[229] This sealer has minimal antimicrobial activity.[167]

FIG. 7-14 Activ GP (Brasseler USA) glass ionomer–coated gutta-percha points and sealer.

Activ GP (Brasseler USA, Savannah Georgia) consists of a glass ionomer–impregnated gutta-percha cone with a glass ionomer external coating and a glass ionomer sealer (Fig. 7-14). Available in 0.04 and 0.06 tapered cones, the sizes are laser verified to ensure a more precise fit. This single cone technique is designed to provide a bond between the dentinal canal wall and the master cone (monoblock). A bacterial leakage study comparing Activ GP/glass ionomer sealer, Resilon/Epiphany, and gutta-percha (GP)/AH Plus demonstrated no statistically significant differences at 65 days.[125]

Resin Sealers

Resin sealers have a long history of use, provide adhesion, and do not contain eugenol. There are two major categories: epoxy resin–based and methacrylate resin–based sealers.

Epoxy Resin Sealers

AH-26 (DENTSPLY DeTrey, Konstanz, Germany) is a slow-setting epoxy resin that was found to release formaldehyde when setting.[204,368] AH Plus (DENTSPLY DeTrey) is a modified formulation of AH-26 in which formaldehyde is not released (Fig. 7-15).[222] The sealing abilities of AH-26 and AH Plus appear comparable.[96] AH Plus is an epoxy resin–amine based system that comes in two tubes. The epoxide paste tube contains a diepoxide (bisphenol A diglycidyl ether) and fillers as the major ingredients, while the amine paste tube contains a primary monoamine, a secondary diamine, a disecondary diamine, silicone oil, and fillers as the major ingredients. It exhibits a working time of approximately 4 hours.

Methacrylate Resin Sealers

Four generations of methacrylate resin–based root canal sealers have been marketed for commercial use.[200,278] The first generation of hydrophilic methacrylate resin–based material (Hydron; Hydron Technologies, Inc., Boca Raton, Florida) was designed for en masse root filling and appeared in the mid 1970s when

FIG. 7-15 AH Plus sealer is a resin formulation. (Courtesy DENTSPLY DeTrey, Konstanz, Germany.)

scientific foundations behind dentin bonding were at their infancy stage of development. The major component of Hydron was poly[2-hydroxyethyl methacrylate] (poly[HEMA]), which was injected into a root canal and polymerized in situ within the canal space without the adjunctive use of a root-filling material. Hydron became obsolete in the 1980s as subsequent clinical findings were unacceptable.[215]

Prior to the advent of contemporary methacrylate resin–based sealers that are specifically designed for endodontic application, there had been sporadic attempts on the use of low viscosity resin composites (i.e., resin cements) and dentin bonding agents as sealers for root-filling materials. Leonard and coworkers were the first to demonstrate, with the use of an etch-and-rinse technique (citric acid–ferric chloride etchant known as 10:3 solution), the formation of a hybrid layer in radicular dentin with C&B-Metabond (Parkell Inc., Edgewood, New York), an adhesive resin cement for cementation of indirect restorations and prostheses.[221] Following the marketing of self-priming, self-etching and self-adhesive resin cement technologies in restorative dentistry, functionally analogous, low viscosity methacrylate resin–based root canal sealers have since been available for use in endodontics. This type of bondable root canal sealers has been aggressively promoted with the highly desirable property of creating monoblocks within the root canal space.[383] The term monoblock refers to the idealized scenario in which the canal space becomes perfectly filled with a gap-free, solid mass that consists of different materials and interfaces, with the purported advantages of simultaneously improving the seal and fracture resistance of the filled canals.

The second generation of bondable sealer is nonetching and hydrophilic in nature and does not require the adjunctive use of a dentin adhesive. It is designed to flow into accessory canals and dentinal tubules to facilitate resin tag formation for retention and seal after smear layer removal with NaOCl and EDTA. EndoREZ (Ultradent Products Inc., South Jordan, Utah) is a dual-cured radiopaque hydrophilic methacrylate sealer that contains non-acidic diurethane dimethacrylate. The addition of triethyleneglycol dimethacrylate to the sealer composition renders it hydrophilic, so that it may be used in the wet environment of the root canal system and be very effective in penetrating dentinal tubules and forming long resin tags.[34,382] The sealer was found to seal best when applied to slightly moist intraradicular dentin.[440] EndoREZ is recommended for use with either a conventional gutta-percha cone or with specific EndoREZ points (resin-coated gutta-percha). A retrospective clinical and radiographic study evaluating the 10-year treatment outcome of one-visit root canal treatment using gutta-percha and the EndoREZ sealer reported accumulative probability of success of 92.1% after 10 years.[439] The authors concluded that EndoREZ may be recommended as an alternative to other commonly used root canal sealers. Unfortunately, no additional root canal sealer was used in that study for comparison.

New generations of self-etching (third generation) and self-adhesive (fourth generation) resin cements have been introduced to restorative dentists to simplify bonding procedures. They became commercially available shortly after the introduction of those resin cement systems. The third generation self-etching sealers contain a self-etching primer and a dual-cured resin composite root canal sealer. The use of self-etching primers reintroduced the concept of incorporating smear layers created by hand/rotary instruments along the sealer-dentin interface. An acidic primer is applied to the dentin surface that penetrates through the smear layer and demineralizes the superficial dentin. The acidic primer is air-dried to remove the volatile carrier and then a dual-cured moderately filled flowable resin composite sealer is applied and polymerized. Provided that these materials are sufficiently aggressive to etch through thick smear layers, the technique sensitivity of bonding to root canals may be reduced when smear layers are inadvertently retained in the apical third of instrumented canal walls.

Third-generation methacrylate resin–based sealers that incorporate the use of self-etching primers became popularized following the introduction of Resilon (Resilon Research LLC, Madison, Connecticut), a dimethacrylate-containing polycaprolactone-based thermoplastic root-filling material.[355] In RealSeal (SybronEndo), the self-etching primers are supplied as a single-bottle system and contain 2-acrylamido-2-methyl-propanesulfonic acid (AMPS) as the functional acidic monomer. The functional acidic monomer, solvent, water that is necessary for ionization of the acidic monomers, and self-cured catalysts are incorporated into "one-component" (i.e., incorporated inside a single bottle). This is similar to the so-called "one-component" type all-in-one adhesives that are currently available in restorative dentistry. The sealer that is used after application of the self-etching primer consists of bisphenol-A-glycidyldimethacrylate (BisGMA), ethoxylated BisGMA, urethane dimethacrylate (UDMA), and hydrophilic methacrylate with calcium hydroxide, barium sulfate, barium glass, bismuth oxychloride, and silica.[186] An ethoxylated bisphenol-A-dimethacrylate (EBPADMA)–based resinous solvent (e.g., RealSeal Thinning Resin, SybronEndo) is also available for adjusting the sealer viscosity.[301]

The fourth-generation methacrylate resin–based sealers (e.g., MetaSEAL, Parkell Inc.; RealSeal SE, SybronEndo) is functionally analogous to a similar class of recently introduced self-adhesive resin luting cements in that they have further eliminated the separate etching/bonding step.[302] Acidic resin monomers that are originally present in dentin adhesive primers are now incorporated into the resin-based sealer/cement to render them self-adhesive to dentin substrates. The combination of an etchant, a primer, and a sealer into an all-in-one self-etching, self-adhesive sealer is advantageous in that it reduces the application time as well as errors that may occur during each bonding step. MetaSEAL is the first commercially available fourth-generation self-adhesive dual-curable sealer.[217]

The liquid component of MetaSEAL comprises 4-META, HEMA and difunctional methacrylate monomers. The powder contains zirconium oxide as spherical radiopaque fillers, silica nanofillers, and a hydrophilic initiator. The inclusion of an acidic resin monomer, 4-methacryloyloxyethyl trimellitate anhydride (4-META), makes the sealer self-etching and hydrophilic in nature and promotes monomer diffusion into the underlying intact dentin to produce a hybrid layer after polymerization. According to the manufacturer, MetaSEAL is recommended exclusively for cold compaction and single-cone techniques and supports the use of either Resilon or gutta-percha as a root-filling material. The sealer purportedly bonds to thermoplastic root-filling materials as well as radicular dentin via the creation of hybrid layers in both substrates. MetaSEAL is also marketed as Hybrid Bond SEAL (Sun Medical Co. Ltd., Shiga, Japan) in Japan and had been reported to produce equivalent or slightly inferior sealing properties as conventional non-bonding epoxy resin-based sealers.[30,269]

RealSeal SE is the simplified dual-cured version of RealSeal and uses a polymerizable methacrylate carboxylic acid anhydride (i.e., 4-META) as the acidic resin monomer.[15,201,237] It contains EBPADMA, HEMA, BisGMA, benzoyl peroxide, tertiary amine, photoinitiators, silane-treated barium borosilicate glass, silica, bismuth oxychloride, Ca-Al-F silicate, tricalcium phosphate as additional components. It may be used with Resilon cones or pellets using cold lateral or warm vertical techniques, or with RealSeal 1, a carrier-based Resilon obturator system.[166]

Silicone Sealers

RoekoSeal (Coltène/Whaledent) is a polydimethylsiloxane that has been reported to expand slightly on setting.[273]

GuttaFlow and GuttaFlow2 (Coltène/Whaledent) are cold flowable matrices that are triturated. They consist of gutta-percha in particulate form (less than 30 μm) added to RoekoSeal (Fig. 7-16). The material is provided in capsules for trituration. The technique involves injection of the material into the canal, followed by placement of a single master cone. The material provides a working time of 15 minutes and it cures in 25 to 30 minutes. Evidence suggests that the material fills canal irregularities with consistency[435] and is biocompatible,[45,116] but the setting time is inconsistent and may be delayed by final irrigation with sodium hypochlorite.[45] The sealing ability appears comparable to other techniques in some studies and inferior in others.[45,47,206,276,396]

FIG. 7-16 GuttaFlow trituration capsule and injection syringe (Coltène/Whaledent).

Calcium Silicate Sealers

A new category of root canal sealers based on mineral trioxide aggregate (MTA) has recently been commercially available. These sealers are an outgrowth of the popularity of MTA materials, which are based on tricalcium silicate, a hydraulic (water-setting) powder used for various surgical and vital pulp therapy treatments.[279,280] This type of root canal sealer is attractive because of the bioactivity that has been reported for MTA-type materials,[131,384] which are also known for being hydrophilic.[65]

Calcium silicate sealers include some of the same hydraulic compounds found in Portland cement, primarily tricalcium silicate and dicalcium silicate powder.[65] The first use of hydraulic calcium silicate materials in dentistry dates to 1878, when a German, Dr. Witte, published a case report on using Portland cement to fill root canals.[103] Tricalcium silicate–based materials did not come into common practice until the 1990s, when Mineral Trioxide Aggregate (MTA) was introduced. MTA is a hydraulically active powder that contains primarily tricalcium silicate, dicalcium silicate, and a radiopaque powder, often bismuth oxide. Because of staining of crown dentin by the bismuth oxide component, which may be rendered brown (in NaOCl), gray (in chlorhexidine) or even black (in glutaraldehyde), the radiopacifier has now been replaced with other materials such as zirconia dioxide (zirconia) or tantalum oxide in some commercial formulations. The radiopaque agent, bismuth oxide, zirconia, or tantalum oxide, is important, without which MTA would not be distinguishable on a radiograph. Minor phases of tricalcium aluminate and calcium sulfate are often present. Some MTA-type products contain calcium carbonate, or tetracalcium aluminoferrite. The term MTA is used here to denote all the tri- and dicalcium silicate products.

Tricalcium silicate cements/sealers set by reaction with water and form a highly alkaline (pH of about 12) mixture consisting of a rigid matrix of calcium silicate hydrates and calcium hydroxide.[89] These hydrates form on the surface of the original calcium silicate particles and hydration gradually penetrates inward. When tricalcium silicate cement sets, the dimensional change is less than 0.1% expansion, which helps with creating a barrier, and is especially important for endodontics. Setting time for tricalcium silicate cements is lengthy, about 165 minutes for the initial set and less than 6 hours for the final set, which has been a major drawback for using them in some indications, but not for its use as a sealer. The consistency of the MTA products with water has not been suitable for use as a sealer. Also, the coarseness of the first MTA products, intended for surgical use, has meant they were not suitable for use as a sealer, as their film thickness was too high (>50 μm).

Four tricalcium silicate sealers are currently commercially available: MTA Fillapex (Ângelus Indústria de Produtos Odontlógicos Ltda; Londrina, Paraná, Brazil),[44,143,255,438] iRoot SP (Innovative BioCeramix Inc., Vancouver, Canada; aka Endosequence BC sealer; Brasseler USA),[44,66,231,432,434] Endo CPM Sealer (EGEO SRL, Buenos Aires, Argentina),[141,142,255,336,378] and MTA Plus (Avalon Biomed, Bradenton, Florida). The MTA Plus may be used as a cement[220,334] or a sealer by adjusting the powder:liquid ratio. Table 7-2 lists what is known about the components of these sealers. Other experimental tricalcium

TABLE 7-2
Composition of Tricalcium Silicate Root Canal Sealers

Generex B ProRoot Endo Sealer	MTA Fillapex	Endosequence BC Sealer (iRoot SP)	Endo CPM Sealer	MTA Plus
Powder/gel	**Dual paste**	**Single paste**	**Powder/gel**	**Powder/gel**
Mineral trioxide aggregate (MTA) powder with enhanced radiopacity Water-based gel	Salicylate resin Diluent resin Natural resin Bismuth oxide Silica MTA Pigments	Zirconium oxide Calcium silicates Calcium phosphate monobasic ($CaH_4P_2O_8$) Calcium hydroxide Filler Thickening agents	MTA powder Silicon dioxide Calcium carbonate Bismuth oxide Barium sulfate Propylene glycol alginate Propylene glycol Sodium citrate Calcium chloride Active ingredients	MTA powder Water-based gel

silicate sealers include Generex B (aka ProRoot Endo Sealer),[404] MTAS[64] and MTA Flow.[130] The iRoot SP is premixed and MTA Fillapex has two pastes that are combined in a mixing tip. The Endo CPM Sealer and MTA Plus sealer are powder/gel systems that the user can mix to the consistency desired. The available materials' descriptions are vague; for instance, MTA Fillapex has "natural or diluent resins," with "filler and thickening agents." However, what is believed to be in common is that tricalcium silicate powder is a component of all four sealers.

The MTA Fillapex and iRoot SP sealers have non-aqueous vehicles in which the powders are dispersed. For the tricalcium silicate particles to contribute to sealing, they must become hydrated in the tooth by exchange of the non-aqueous vehicle for water in the root canal. EndoCPM Sealer and MTA Plus sealer rely on mixing with water-based gels just before use, without a non-aqueous resin. The proportions of the tricalcium silicate powders are not known in any of these materials, but are most likely to be highest in MTA Plus because no inert liquid vehicle is included. The inert vehicle is the liquid medium in the pastes of MTA Fillapex and iRoot SP that does not react with the powder. The bioactivity of MTA-type materials has made the use of tricalcium silicate powders in sealers of interest. The possibility of extrusion past the apex with a bioactive material leads to the supposition that healing in the periapical area would occur more readily with a tricalcium silicate product. However, two of the sealers have the tricalcium silicate powders dispersed in a non-reactive organic medium, which may detract from the potential benefit of the bioactive powder. To date, no peer-reviewed papers have been published to establish the benefits of tricalcium silicate powders in sealers in human clinical studies; all studies and reports are based on in vitro testing and in vivo animal models. Calcium carbonate is added to Endo CPM Sealer to reduce the pH from 12.5 to 10.0 after setting. By doing so, the manufacturer claimed that surface necrosis in contact with the material is reduced, thereby optimizing the conditions for the enzymatic action of alkaline phosphatase.[142]

The physical properties of some of these sealers have been published. Table 7-3 contains values that are known for comparison to the ISO 6876/ADA 57 methods and requirements and the AH Plus sealer. The flow, film thicknesses, solubility, dimensional stability, and radiopacity of the new sealers meet the ISO 6876 requirements. The flow of these sealers varies, but so do the values for AH Plus. The dimensional stability is excellent. The solubility of the sealers is higher than AH Plus, which may be attributed to the formation of partially soluble calcium hydroxide within the set tricalcium silicate. The radiopacities are less than AH Plus, which is known for its radiopacity.

The working and setting times of the iRoot SP and MTA Plus materials are longer than the MTA Fillapex sealer. iRoot SP has the longest setting time, and requires diffusion of water from the dentinal tubules into the sealer to set.[256] In a test where water was added to the EndoSequence BC sealer (i.e., iRoot SP), the setting time was reduced to about 150 hours, but the hardness was significantly diminished.[231] The Endosequence BC sealer has been tested for strengthening teeth when used with glass ionomer–coated cones and found to be stronger in vitro.[135]

Biocompatibility tests have been performed with some of these sealers. Endosequence BC sealer was more cytotoxic than AH Plus root canal sealer; the time to become noncytotoxic as judged from mitochondrial enzymatic (succinic dehydrogenase) activity was 5 weeks for Endosequence BC sealer and 3 weeks for AH Plus sealer.[231] Another study found that after setting, the cytotoxicity of MTA Fillapex decreased and the sealer presents suitable bioactivity to stimulate hydroxyapatite nucleation.[323]

Medicated Sealers

Sealers containing paraformaldehyde are strongly contraindicated in endodontic treatment (Fig. 7-17). Although the lead and mercury components may have been removed from these zinc oxide–eugenol formulations over time, the severely toxic paraformaldehyde content has remained a constant. These sealers are not approved by the U.S. Food and Drug Administration[12] and are unacceptable under any circumstances in clinical treatment because of the severe and permanent toxic effects on periradicular tissues.[348] A paste containing 6.5% paraformaldehyde as well as lead and mercury was advocated for use by Sargenti[325-327] and originally marketed as N-2. Lead has been reported in distant organ systems when N-2 is placed within the radicular space.[274] In another study the investigators reported the same results regarding systemic distribution of the paraformaldehyde component of N-2.[38] Removal of the heavy metals resulted in a new formulation: RC2B. Other

TABLE 7-3

Physical Properties of Tricalcium Silicate Root Canal Sealers

Quality	ISO 6876-2001 ADA 57 Requirement	AH Plus*†	MTA Fillapex†	Endosequence BC Sealer	MTA Plus White, Gray‡
Flow (mm)	>20	21, 40	25, 29	23	31
Film thickness (μm)	<50	16	24	22	47
Solubility	<3%	0.1	1.1	2.9	1.9
Dimensional stability	–1% to +0.1%	–0.04	–0.7	+0.09	+0.05
Radiopacity	>3 mm Al				4.9, 5.5
Working time (hours)	No requirement	4:00, 5:30	0:45, 0:30	2:40	0:20
Setting time (hours)	No requirement	11:30, 5:10	2:30, 2:15	>24	10:00

*An epoxy resin–based sealer included for comparative purposes.
†Measured by two authors.
‡If one value is given, white and gray versions of MTA Plus are equal.

paraformaldehyde sealers include Endomethasone, SPAD, and Reibler's paste. The toxic in vivo effects of these materials on the pulp and periapical tissues have been demonstrated over time.[80,261]

In addition to the toxic nature of the material, clinicians employing the material place it with a lentulo spiral. Overextension has resulted in osteomyelitis and paresthesia.[117,202] One of these studies reported irreversible neurotoxicity, manifested as dysesthesia, in cases where paraformaldehyde pastes were forced through the apical foramen into the periapical tissues.[202] The reader is referred to Chapter 29 for further discussion about this harmful material and technique.

SEALER PLACEMENT

Various methods of sealer placement have been advocated, including the master cone, lentulo spirals, files and reamers, and ultrasonics. Investigators compared sealer placement using a file rotated counterclockwise, the lentulo spiral (Fig. 7-18), an ultrasonic file, and coating the master gutta-percha cone.[413] Placement did not differ with the various techniques; however, the investigators noted the most variation in sealer coating was in the apical area.[413] Another study compared sealer placement with a K-type file, the lentulo spiral, and using the master cone in curved canals. Results demonstrated no significant differences in the techniques after obturation; no technique covered more than 62.5% of the canal wall surface.[158] Other investigators reported that ultrasonics produced the best sealer distribution when used circumferentially.[369] These findings were supported by another study that reported ultrasonic placement to be superior to manual placement techniques.[1]

In the past, it has been assumed that instrumented canals need to be thoroughly dried prior to obturation with root-filling materials and sealers. Indeed, there were leakage studies reporting that a better apical seal might be achieved by drying the canals with 95% ethanol prior to obturation.[370] A recent study reported that for some root canal sealers (iRoot SP, AH Plus, Epiphany and MTA Fillapex), better adhesion to the dentinal walls might be achieved by leaving the canals slightly moist (i.e., drying the canals with low vacuum via a Luer-Lok adapter for 5 seconds, followed by one paper point for one second).[256] Similar results were also reported with the use of the EndoREZ sealer.[440] This, however, does not mean that canals should remain totally flooded with water prior to obturation, because such a level of residual moisture would adversely affect the adhesiveness of those root canal sealers to intraradicular dentin.[256]

The method of obturation does not seem to affect the sealer distribution on the canal wall in the apical portion of the canal; however, lateral compaction results in better distribution in the mid-coronal areas when compared with warm vertical compaction.[422] Another well-controlled study reported that none of five evaluated obturation techniques resulted in uniform sealer distribution along the entire length of the core obturation material.[176] Evidence indicates that the method of obturation affects the sealer penetration into tubules. This was exemplified by a study reporting that thermoplastic techniques produced deeper sealer penetration into tubules.[94] Removal of the smear layer enhances sealer penetration into the dentinal tubules.[93]

CORE MATERIALS

Although a variety of core materials have been used in conjunction with a sealer/cement, the most common method of obturation involves gutta-percha as a core material. Regardless of the obturating technique, emphasis should be placed on the process of cleaning and shaping the canal. The materials and techniques described do not routinely create an impervious seal of the canal system; all materials leak to some extent.[2] The choice of the obturation technique(s) depends on the unique circumstances with which each case presents.

The properties of an ideal obturation material have been outlined by Grossman (Box 7-2).[149] Historically, a variety of materials have been employed to obturate the root canal space. Solids, semisolid materials, and pastes have been employed. A common solid material used in the past was the silver cone.

Silver Cones

Jasper introduced cones made of silver, which he claimed produced the same success rate as gutta-percha and were easier to use.[183] The rigidity provided by the silver cones made them easier to place and permitted more predictable length control; however, their inability to fill the irregularly shaped root canal system permitted leakage (Fig. 7-19). Silver cones were believed to possess an oligodynamic property which, if true,

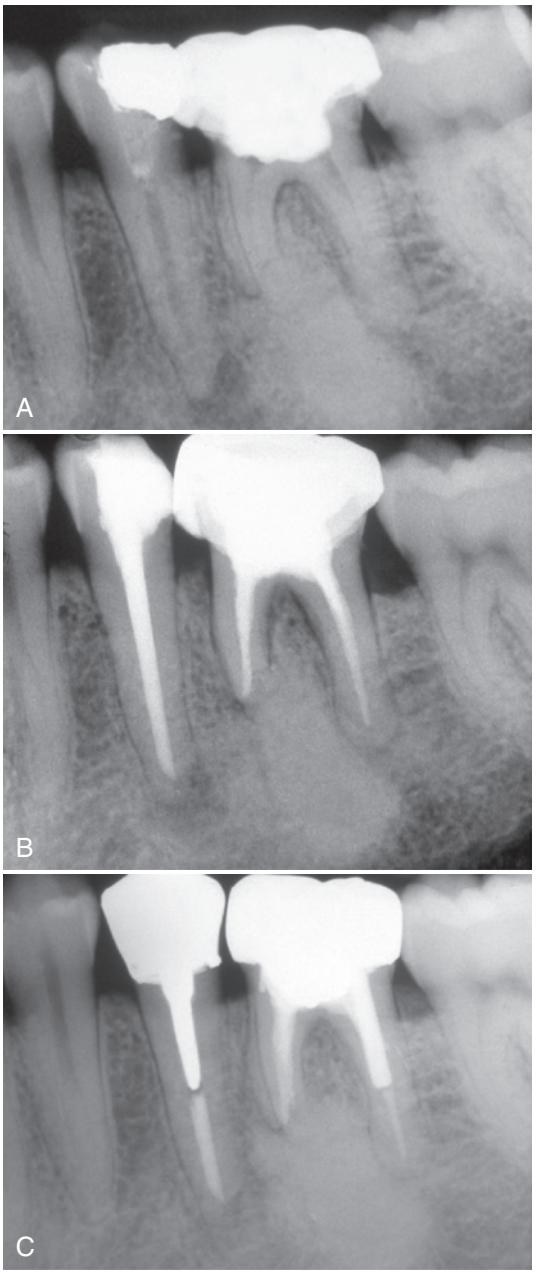

FIG. 7-17 Patient treated with Sargenti paste in her mandibular left second premolar and first molar. **A,** Pretreatment radiograph exhibits an osteolytic response associated with the premolar and a proliferative response associated with the molar. **B,** Posttreatment radiograph of the teeth. **C,** One-year follow-up radiograph exhibiting osseous regeneration apical to the second premolar.

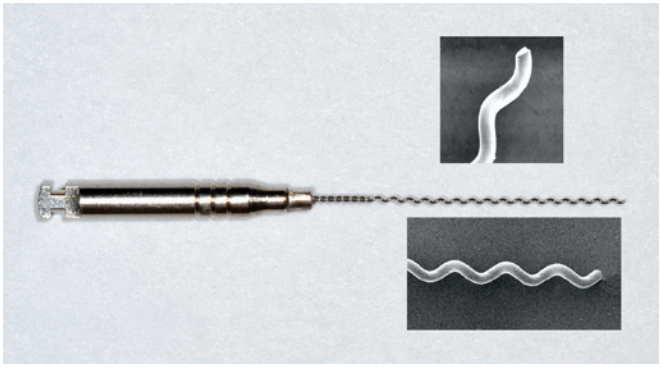

FIG. 7-18 Lentulo spiral used for sealer placement during obturation.

BOX 7-2

Properties of an Ideal Obturation Material

- Easily manipulated and provides ample working time
- Dimensionally stable with no shrinkage once inserted
- Seals the canal laterally and apically, conforming to its complex internal anatomy
- Nonirritating to the periapical tissues
- Impervious to moisture and nonporous
- Unaffected by tissue fluids—no corrosion or oxidization
- Inhibits bacterial growth
- Radiopaque and easily discernible on radiographs
- Does not discolor tooth structure
- Sterile
- Easily removed from the canal if necessary

would have resulted in the destruction of microbes within the root canal system. Unfortunately, it was not true. Moreover, when silver points contact tissue fluids or saliva, they corrode.[48] The corrosion products have been found to be cytotoxic and produce pathosis or impede periapical healing.[344]

With the introduction of rigid silver cones it became possible to easily place them to length. This resulted in clinicians often failing to properly clean and shape the canal before obturation. Treatment failures were the result of leakage and failure to remove the irritants from the root canal system. The use of silver cones today is considered to be below the standard of care in contemporary endodontic practice. For further information about this material and technique, the reader is referred to Chapter 29.

Gutta-Percha

Gutta-percha is the most popular core material used for obturation. Major advantages of gutta-percha are its plasticity, ease of manipulation, minimal toxicity, radiopacity, and ease of removal with heat or solvents. Disadvantages include its lack of adhesion to dentin and, when heated, shrinkage upon cooling. Gutta-percha is the *trans*-isomer of polyisoprene (rubber) and exists in two crystalline forms (α and β).[144] In the unheated β phase, the material is a solid mass that is compactable. When heated, the material changes to the α phase and becomes pliable and tacky and can be made to flow when pressure is applied. A disadvantage to the α phase is that the material shrinks on setting.[340]

Gutta-percha cones consist of approximately 20% gutta-percha, 65% zinc oxide, 10% radiopacifiers, and 5% plasticizers.[126] Attempts have been made to make gutta-percha more antimicrobial by the addition of materials such as iodoform,[75] calcium hydroxide,[230] chlorhexidene,[233] and tetracycline.[247] The clinical effectiveness of adding these materials has not been demonstrated. Moreover, to exert an antimicrobial pharmacological effect, the active ingredient must leach out of the gutta-percha, which could have a detrimental effect on long-term sealability.

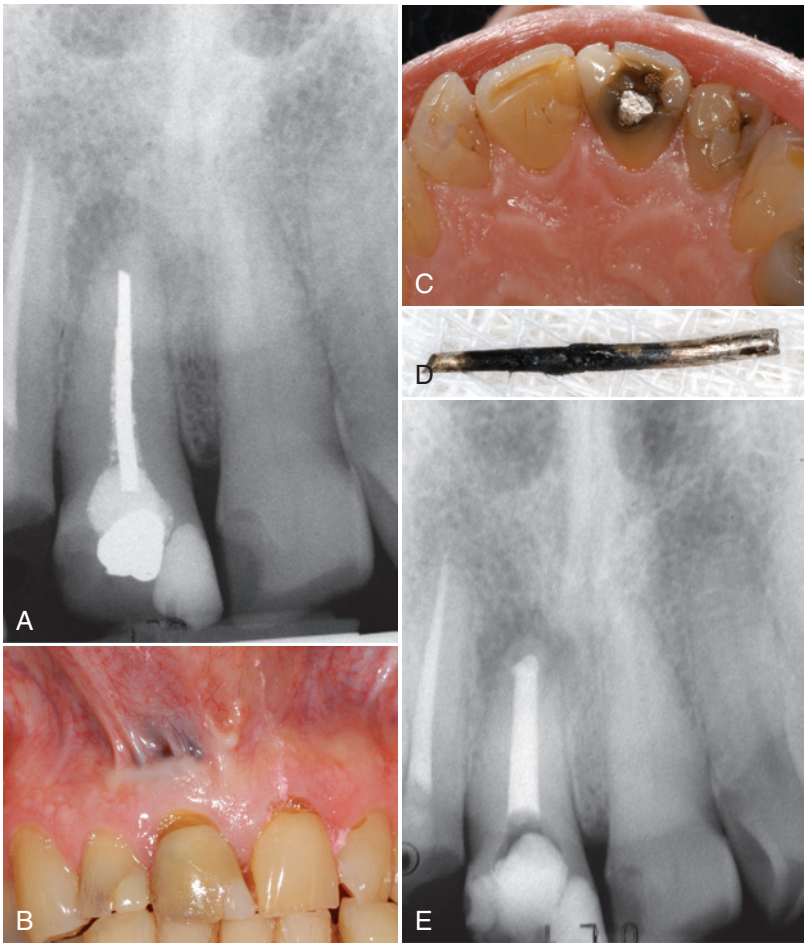

FIG. 7-19 Silver cones are advocated for ease of placement and length control. **A,** Radiograph of a facial maxillary right central incisor obturated with a silver cone. **B,** Tissue discoloration indicating corrosion and leakage. **C,** Lingual view indicates coronal leakage. **D,** Corroded silver cone removed from the tooth. **E,** Posttreatment radiograph of the tooth.

Unlike rubber, room temperature gutta-percha cannot be condensed or made to flow. Compaction results in transmission of forces to the material and the canal wall equally and may result in root fracture. Gutta-percha can be made to flow if it is modified by either heat or solvents such as chloroform. This permits adaptation to the irregularities of the canal walls.

The α form of gutta-percha melts when heated above 65°C. When cooled extremely slowly, the α form will recrystallize. Routine cooling results in the recrystallization of the β form. Although the mechanical properties for the two forms are the same, when α-phase gutta-percha is heated and cooled, it undergoes less shrinkage, making it more dimensionally stable for thermoplasticized techniques. The use of α-phase gutta-percha for obturation has increased as thermoplastic techniques have become more common.

Gutta-percha cones are available in standardized and nonstandardized sizes. Standardized sizes conform to requirements contained in specifications published by the International Organization of Standardization (ISO) or ADA American National Standards Institute (ADA ANSI). The nonstandard nomenclature refers to the dimensions of the tip and body (Fig. 7-20). A fine-medium cone has a fine tip with a medium body. Standardized cones are designed to match the taper of stainless steel and nickel-titanium instruments (Figs. 7-21 and 7-22). A size 40, 0.04 taper cone has a tip of 0.4 mm and a taper of 0.04 mm per millimeter. Unfortunately ISO and ADA ANSI standards allow tolerances and, coupled with a less than completely accurate manufacturing process, the actual cone size varies as does the tip and taper of the master apical file.[140,243]

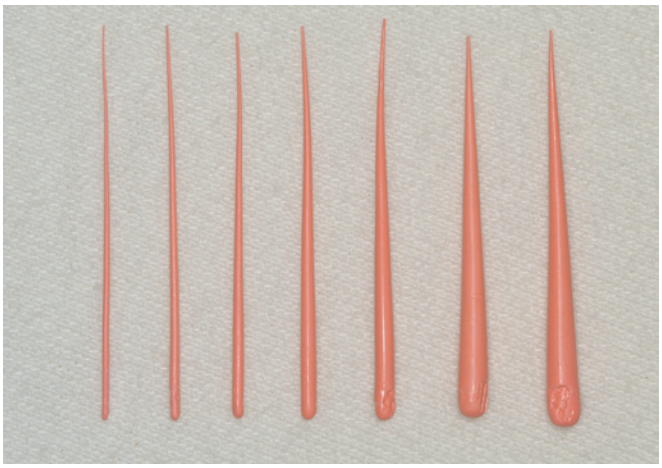

FIG. 7-20 Nonstandard gutta-percha cones: extra fine, fine fine, fine, medium fine, fine medium, medium, large, and extra large.

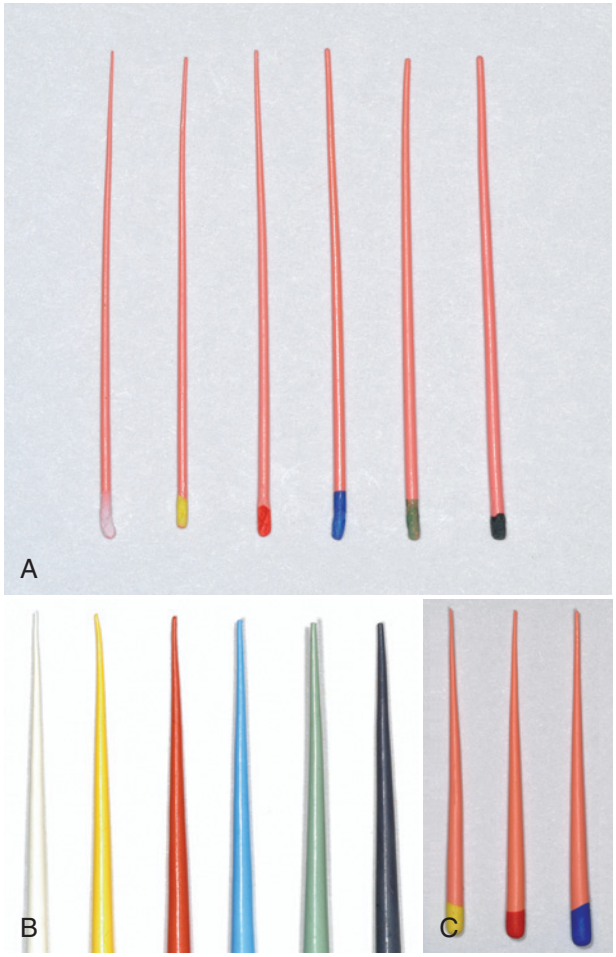

FIG. 7-21 A, Standard gutta-percha cone sizes #15 to #40. B, Standard cones #.06, taper sizes #15 to #40. C, Standard cones Protaper F1, F2, F3.

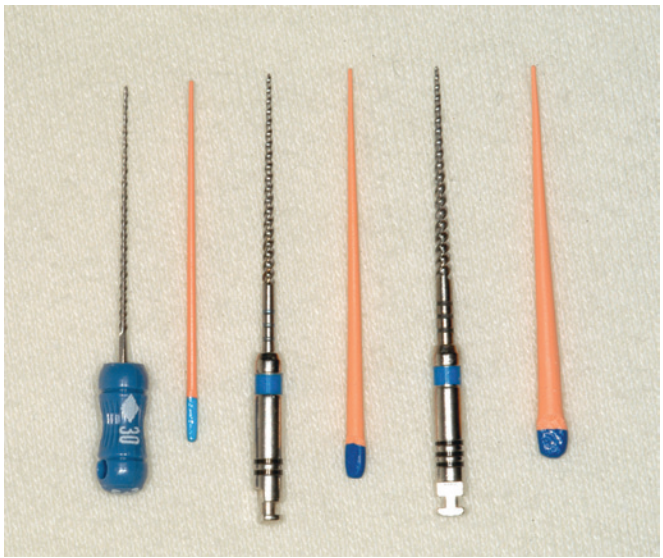

FIG. 7-22 Size #30 standard gutta-percha points exhibiting #.02, #.04, and #.06 tapers.

FIG. 7-23 Epiphany system with the primer, thinning resin, sealant, and standard Resilon points. (Courtesy Pentron Clinical Technologies, Wallingford, Conn.)

Although gutta-percha points cannot be heat sterilized, a study reported that gutta-percha points can be sterilized by placing in 5.25% NaOCl for one minute. This study also reported that 2% glutaraldehyde, 2% chlorhexidine, and 70% ethyl alcohol were not effective in killing *Bacillus subtilis* spores, one of the hallmark organisms used for testing effective antimicrobial effectiveness.[359]

Activ GP

Activ GP (Brasseler USA) consists of gutta-percha cones impregnated on the external surface with glass ionomer (see Fig. 7-14). Single cones are used with a glass ionomer sealer. Available in 0.04 and 0.06 taper cones, the sizes are laser-verified to help ensure a more precise fit. The single cone technique is designed to provide a bond between the dentinal canal wall and the master cone. A bacterial leakage study comparing Activ GP/glass ionomer sealer, Resilon/Epiphany, and gutta-percha/AH Plus demonstrated no statistically significant differences in leakage at 65 days.[125]

Resilon

The resin-based obturation systems RealSeal (SybronEndo), and Resinate (Obtura Spartan, Algonquin, Illinois) have been introduced as alternatives to gutta-percha (Figs. 7-23 and 7-24). Resilon is a high-performance industrial polyester that has been adapted for dental use.

The resin sealer bonds to a Resilon core, and attaches to the etched root surface. The manufacturer claims that this forms a "monoblock" (Fig. 7-25). With traditional obturation techniques, there is a gutta-percha–sealer interface and a tooth-sealer interface. With Resilon the resin sealer bonds to both the canal wall and the Resilon cone. Whether a monoblock is achievable remains controversial.[303]An in-depth review article on the subject of monoblocks indicates that with current materials and techniques, the monoblock has yet to be achieved.[383]

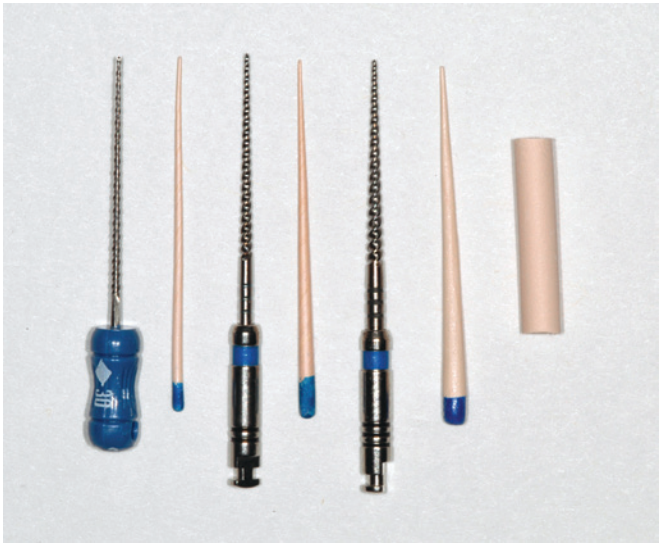

FIG. 7-24 Resilon #.02, #.04, and #.06 tapered points and a thermoplastic plug for use in the Obtura system.

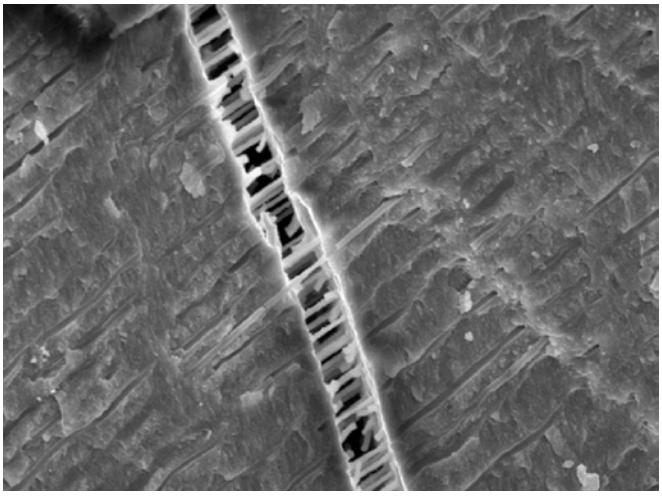

FIG. 7-25 Scanning electron microscopy view of Resilon tags extending into the dentinal tubules.

The system resembles gutta-percha and can be placed by lateral compaction, warm lateral or vertical compaction, thermoplastic injection, or with a core-carrier technique. It consists of a resin core material (Resilon) composed of polyester, difunctional methacrylate resin, bioactive glass, radiopaque fillers, and a resin sealer. Resilon is nontoxic, nonmutagenic, and biocompatible. The core material is available in nonstandard and standard cones and pellets for use in thermoplastic techniques (see Fig. 7-24).

After cleaning and shaping procedures an appropriate master cone is placed into the prepared canal and a radiograph image is exposed to verify the apical position. Because NaOCl may affect the bond strength of the primer, EDTA should be the last irrigant used before rinsing the canal with sterile water, saline, or chlorhexidine.

After drying the canal, a self-etch primer (sulfonic acid–terminated functional monomer, 2-hydroxyethyl methacrylate [HEMA], water, and polymerization initiator) is used to condition the canal walls and prepare them for bonding to the resin sealant (resin matrix of bisphenol A-glycidyl methacrylate [Bis-GMA], ethoxylated Bis-GMA, urethane dimethacrylate [UDMA], and hydrophilic difunctional methacrylates and fillers [70%] of calcium hydroxide, barium sulfate, barium glass, bismuth oxychloride, and silica). Two or three drops are placed in the canal with a pipette, a syringe, or a paper point that wicks the material to the working length. The excess primer is removed, the resin sealer is dispensed onto a mixing slab, and the viscosity is adjusted with the thinning resin. The sealer is applied with a paper point, Resilon point, or lentulo spiral. The canal is then obturated by lateral compaction, warm vertical compaction, or thermoplastic injection. The sealer takes approximately 25 minutes to set, so it is recommended that the coronal surface of the material be light cured for 40 seconds.

When using the System B (SybronEndo) for warm vertical compaction, the temperature setting should be 150°C at a power of 10. With the Obtura II thermoplasticized injection system (Obtura Spartan), the temperature settings vary depending on the needle tip employed. For the 25-gauge needle, a 160°C setting is selected; for the 23-gauge needle, a 140°C setting is used; and for the 20-gauge needle, the setting that is recommended is 120°C to 130°C.

Resilon appears to be comparable to gutta-percha in its ability to seal the radicular space.[22] Investigators evaluated coronal leakage of Resilon, using *Streptococcus mutans* and *E. faecalis* in roots that were filled by lateral and vertical compaction techniques with gutta-percha and AH-26 or Resilon and Epiphany sealer.[355] Resilon showed significantly less coronal leakage when compared with gutta-percha. In another study, investigators used a dog model to assess the ability of Resilon or gutta-percha and AH-26 to prevent apical periodontitis in teeth inoculated with microorganisms. Results indicated periapical inflammation in 18 of 22 roots (82%) obturated with gutta-percha and AH-26, whereas the Resilon group exhibited periapical inflammation in only 4 of 21 roots (19%).[356] Another study demonstrated that teeth filled with Resilon were more resistant to fracture than roots filled with gutta-percha and AH-26 sealer.[387] More recent evidence suggests that Resilon does not strengthen roots.[147,416]

Resilon appears to be biocompatible; implantation in the subcutaneous tissues of rats demonstrated fibrous encapsulation and negligible inflammation at 60 days.[41] One retrospective study compared the success and failure rates between obturation with gutta-percha and Kerr Pulp Canal Sealer and obturation with Resilon or Epiphany, with recall examination between 12 and 25 months. Statistical analysis indicated that the results were indistinguishable.[83] Another study demonstrated that 82 randomly selected clinical cases treated with Resilon produced success rates at 1 year that were comparable to cases treated with gutta-percha.[82] A recent review of the use of Resilon as a root-filling material reported that Resilon cannot be considered yet as an evidence-based replacement for the current gold-standard gutta-percha, due to the lack of long-term clinical outcome studies to demonstrate its clinical superiority over contemporary gutta-percha obturation techniques.[354]

Custom Cones

When the apical foramen is excessively large or the prepared root canal system is large, a custom cone may need to be fabricated (Fig. 7-26). This permits the adaptation of the cone to the canal walls, reduces the potential for extrusion of the core

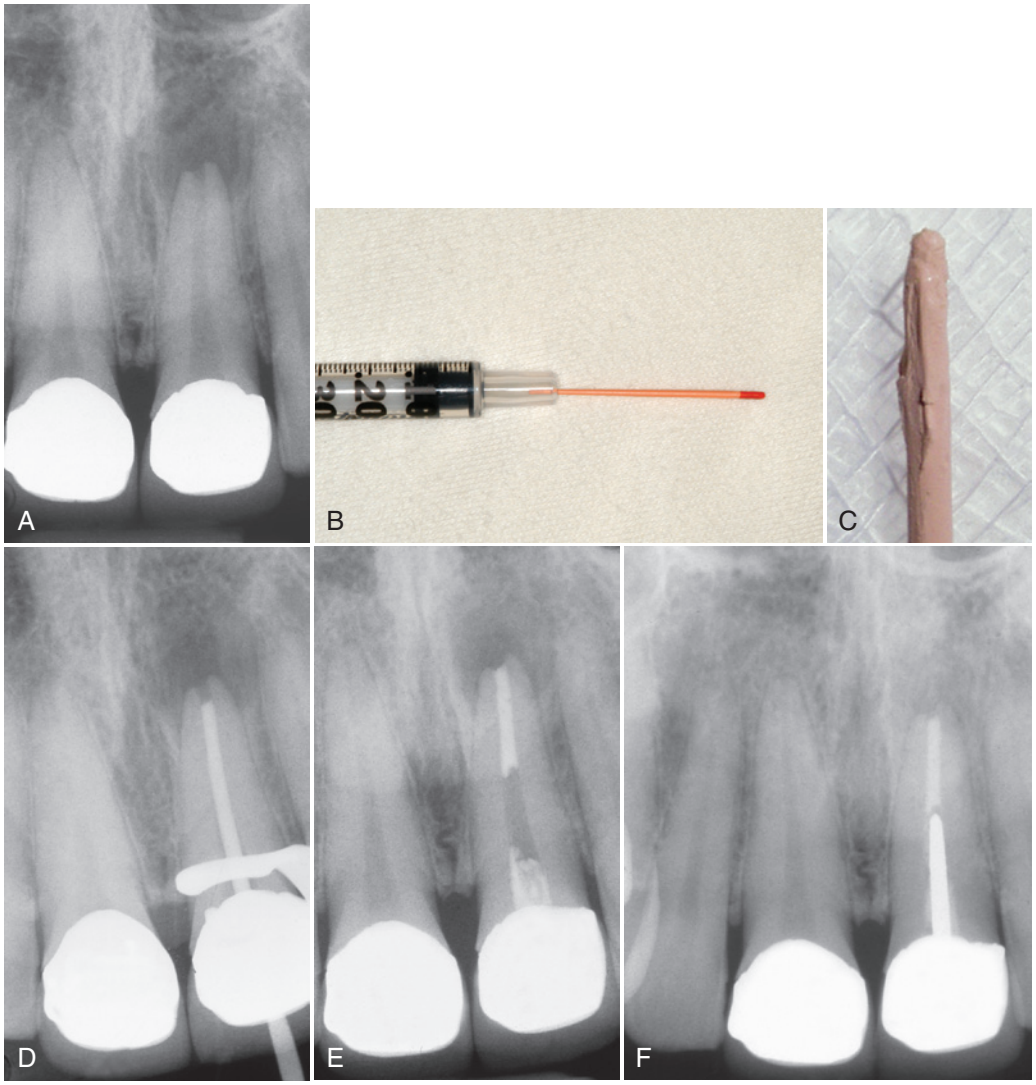

FIG. 7-26 Apical root resorption often results in an open apex requiring fabrication of a custom cone. **A,** Pretreatment radiograph of the maxillary left central incisor with pulp necrosis and chronic apical periodontitis. Apical root resorption is present. **B,** In fabricating a custom master cone a gutta-percha point is fit several millimeters short before softening in solvent and tamping into place. **C,** Softening the apical 2 to 3 mm in chloroform that has been placed in a tuberculin syringe. **D,** The completed custom cone represents an impression of the apical portion of the canal. **E,** The posttreatment radiograph with post space prepared. **F,** A 1-year follow-up radiograph demonstrating osseous regeneration.

material, and may improve the resultant seal.[28,194] The technique involves selection of a master cone and fitting that cone 2 to 4 mm short of the prepared length with frictional resistance. The cone is grasped with locking cotton pliers or a hemostat so that it can be placed into the canal in the same spatial relationship each time. The cone is removed and the tip is softened in chloroform, eucalyptol, or halothane for 1 or 2 seconds, depending on the clinical requirements. Only the outer superficial portion of the cone is softened. The central core of the cone should remain semi-rigid. The cone is then placed into the canal to the working length. The process can be repeated until an adequate impression of the canal is obtained at the prepared length. A radiograph is exposed to verify proper fit and position. An alternative to solvents is softening with heat.[196]

Large root canal systems may necessitate custom fabrication of a large master cone before canal adaptation. This may be accomplished by heating two, or more, large gutta-percha cones and rolling the mass between two glass slabs until an appropriate size is obtained (Fig. 7-27). A spatula may also be used to shape the cone.

METHODS OF OBTURATION

To date, little evidence exists to support one method of obturation as being superior to another based on outcome assessment investigations.[13,264] The prospective Toronto studies have suggested that warm vertical compaction may be superior to lateral compaction[92]; however, definitive evidence is lacking.[286]

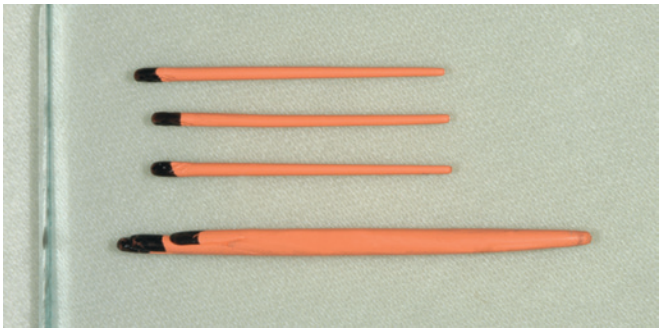

FIG. 7-27 For large canals, several gutta-percha points can be heated and rolled together, using a spatula or two glass slabs.

Lateral Compaction

Lateral compaction is a common method for obturation (Fig. 7-28).[59] The technique can be used in most clinical situations and provides for predictable length control during compaction.[136] A disadvantage is that the technique may not fill canal irregularities[427] as well as warm vertical compaction or other thermoplastic techniques.[424] The procedure can be accomplished with any of the acceptable sealers.

After root canal system preparation, a standard cone is selected that has a diameter consistent with the prepared canal diameter at the working length. Standard cones generally have less taper when compared with nonstandard cones and will permit deeper spreader penetration which will result in a better-quality resultant seal.[288] An alternative is to adapt an appropriately tapered nonstandard cone by cutting small

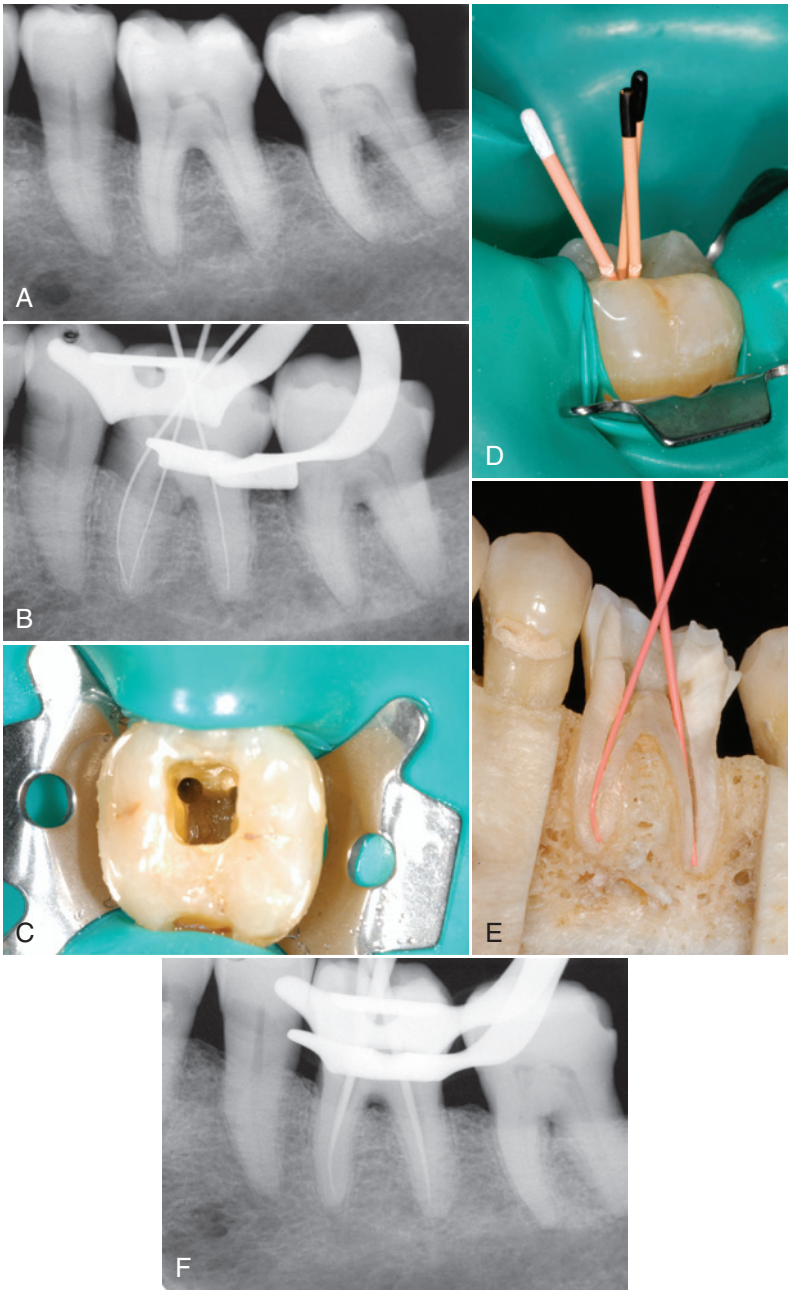

FIG. 7-28 Left first mandibular molar. **A**, Pretreatment radiograph. **B**, Working length radiograph. **C**, Coronal access opening, demonstrating the prepared mesiobuccal canal. **D**, Standardized master cones with coronal reference marked. **E**, Standard master cones fit to length as they exhibit minimal taper and permit deeper penetration of the spreader. **F**, Master cone radiograph.

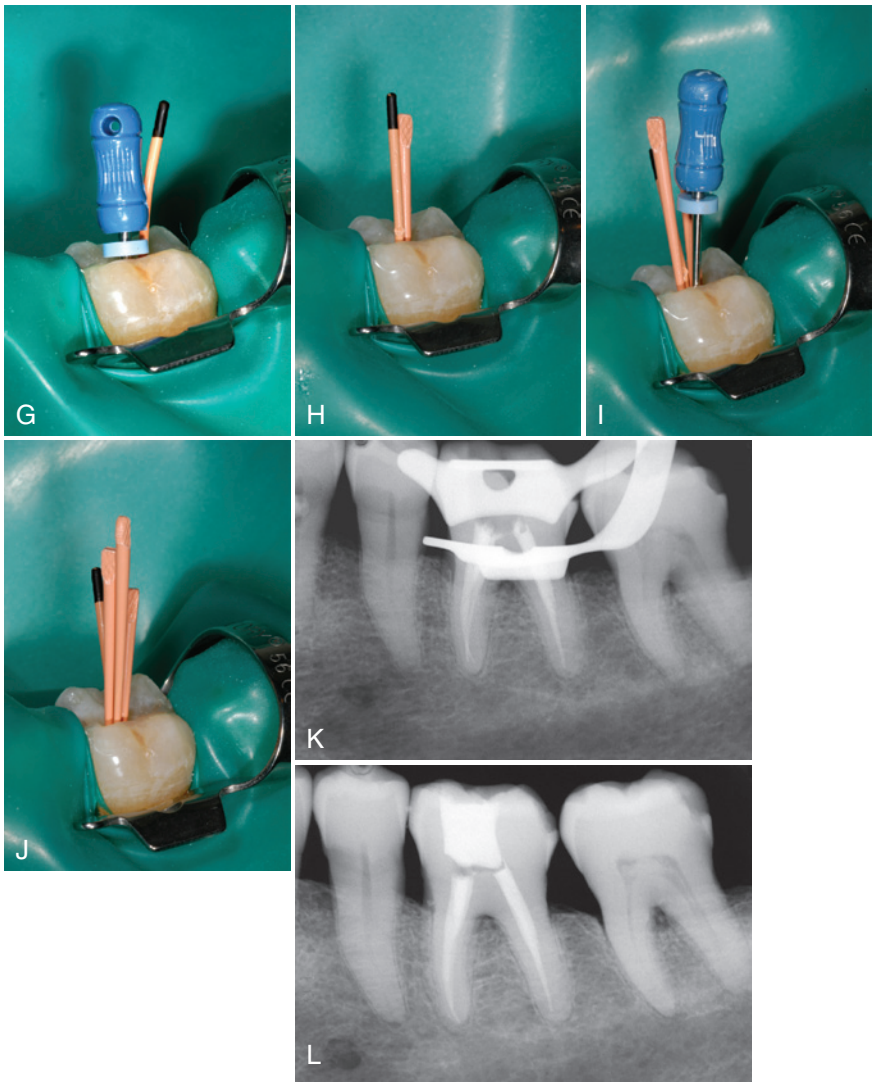

FIG. 7-28, cont'd G, Finger spreader in place. H, Fine-medium accessory cone placed in the space created by the spreader. I, Finger spreader placed in preparation, creating space for additional accessory cones. J, Additional cones are placed until the spreader does not penetrate past the coronal one third of the canal. The cones are then removed at the orifice with heat, and the coronal mass is vertically compacted with a plugger. K, Interim radiograph may be exposed to assess the quality of obturation. L, Posttreatment radiograph demonstrating adequate length, density, and taper. The gutta-percha is removed to the level of the orifice, and a coronal seal has been established with an adequate provisional restoration.

increments from the tip. This "master cone" is measured and grasped with forceps so that the distance from the tip of the cone to the reference point on the forceps is equal to the prepared length. A reference point on the cone can be made by pinching the cone. The cone is placed in the canal, and if an appropriate size is selected, there will be resistance to displacement or "tug back." If the cone is loose it can be adapted by removing small increments from the tip. If the master cone fails to go to the prepared length a smaller cone can be selected. Devices are available to cut cones accurately at a predetermined length (Tip Snip; SybronEndo). When the cone extends beyond the prepared length a larger cone must be adapted or the existing cone shortened until there is resistance to displacement at the corrected working length.

The master cone placement is confirmed with a radiograph. The canal is irrigated and dried with paper points. Sealer is applied to the canal walls, and a spreader is prefitted so as to allow it to be inserted to within 1 to 2 mm of the working length. Appropriately sized accessory points are also selected to closely match the size of the spreader. The correlation between spreader size and nonstandard cones is variable,[53,437] and in small curved canals there does not appear to be a difference in the quality of obturation with nonstandard cones when compared with standard cones.[169,395]

Finger spreaders provide better tactile sensation and are less likely to induce fractures in the root when compared with the more traditional D-11T hand spreader.[88,223,224] In addition to the type of spreader, forces applied, and amount of dentin removed, spreader size may be a factor in root fracture, with large sizes inducing more stress.[294] Spreaders made from nickel-titanium are available and provide increased flexibility,[35] reduced stress,[109] and provide deeper penetration when

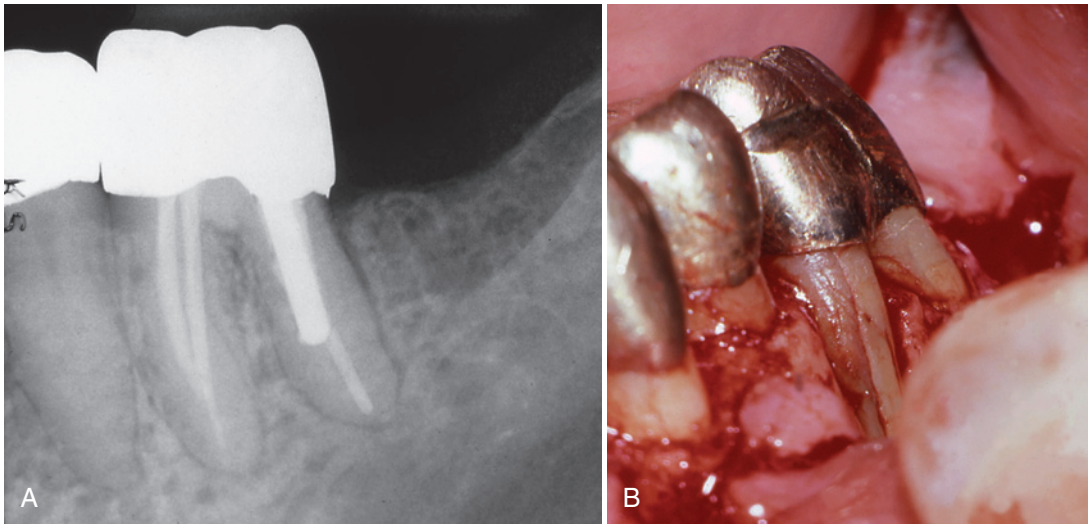

FIG. 7-29 Vertical root fractures can occur with excessive compaction forces. **A,** Follow-up radiograph of a mandibular left first molar. A deep isolated periodontal probing defect was associated with the buccal aspect of the mesiobuccal root. **B,** Flap reflection revealed a vertical root fracture.

compared with stainless steel instruments.[189,341,417] The spreader should fit to within 1 to 2 mm of the prepared length, and when introduced into the canal with the master cone in place, it should be within 2 mm of the working length.[7] There appears to be a correlation between the establishment of a higher quality seal and the depth of spreader penetration.[7,352]

After the spreader has been placed to its maximum depth, it is removed by rotating it back and forth as it is withdrawn. An accessory cone is placed in the space vacated by the spreader. The process is repeated until the spreader no longer goes beyond the coronal one third of the canal. The excess gutta-percha is removed with heat and the coronal mass is compacted with an appropriate unheated plugger. Only light pressure is required during lateral compaction because the gutta-percha is not condensed, and because as little as 1.5 kg of pressure is capable of fracturing a root (Fig. 7-29). In addition to the force applied, investigators have noted that removal of excessive amounts of dentin during preparation is a significant factor in root fracture.[415]

A disadvantage to lateral compaction is that the process does not produce a homogeneous mass. The accessory and master cones are laminated and remain separate. It is envisaged that the space between each of the cones is filled with sealer to aid in establishment of a water-tight seal.

The excess gutta-percha in the chamber is then seared off and vertically compacted with a heated plugger at the orifice or approximately 1 mm below the orifice in posterior teeth. Warm vertical compaction of the coronal gutta-percha enhances the seal.[429] In anterior teeth, the desired level is the cementoenamel junction on the facial surface to avoid aesthetic issues if the dentin should become stained. An alternative to lateral compaction with finger spreaders is ultrasonics.[21] For example, one study found that the technique produced adequate obturation and a 93% clinical success rate.[436]

Another study used ultrasonic-energized files in a warm lateral compaction technique and reported that the amount of gutta-percha, by weight, increased by 33% with two applications of ultrasonics when compared with lateral compaction.[98] Unfortunately, investigators found that the mean internal temperature rise was 29°C at the 6-mm level, with external heat generation exceeding the safe limit of 10°C.

Warm Vertical Compaction

Schilder introduced warm vertical compaction of gutta-percha as a method of obturating the radicular space in three dimensions.[339] Preparation requirements for the technique include preparing a root canal system with a continuously tapering funnel and keeping the apical foramen as small as possible.

The armamentarium includes a variety of pluggers and a heat source. Schilder pluggers come in a variety of sizes (#8 = 0.4 mm, # 8½ = 0.5 mm, etc., for sizes #9, #9½, #10, #10½, #11, #11½, #12) with increasing diameter. The instruments are marked vertically at 5-mm intervals. Various ISO standardized instruments are also available (Fig. 7-30).

The technique involves fitting a master cone short of the prepared working length (0.5 to 2 mm) with resistance to displacement (Fig. 7-31). This ensures that the cone diameter is larger than the prepared canal at the terminus. Nonstandard cones that closely replicate the canal taper are best because they permit the development of hydraulic pressure during compaction. After the adaptation of the master cone, it is removed and sealer is applied to the cone and the walls of the prepared canal. The cone is placed in the canal and the coronal portion is removed with a heated instrument. A heated spreader or plugger is used to remove portions of the coronal gutta-percha in successive increments and soften the remaining material in the canal. The Touch 'n Heat (SybronEndo) (Fig. 7-32), DownPak (Hu-Friedy, Chicago, Illinois), and System B (SybronEndo) (Fig. 7-33) are alternatives to applying heat with a flame-heated instrument because they permit improved temperature control. A plugger is inserted into the canal and the gutta-percha is compacted, forcing the plasticized material apically. The process is repeated until the apical portion has been reached. The coronal canal space is back-filled using small preheated pieces of gutta-percha. The sectional method consists of placing 3- to 4-mm sections of gutta-percha approximating the size of the canal into the root, applying heat, and compacting the mass with a plugger.

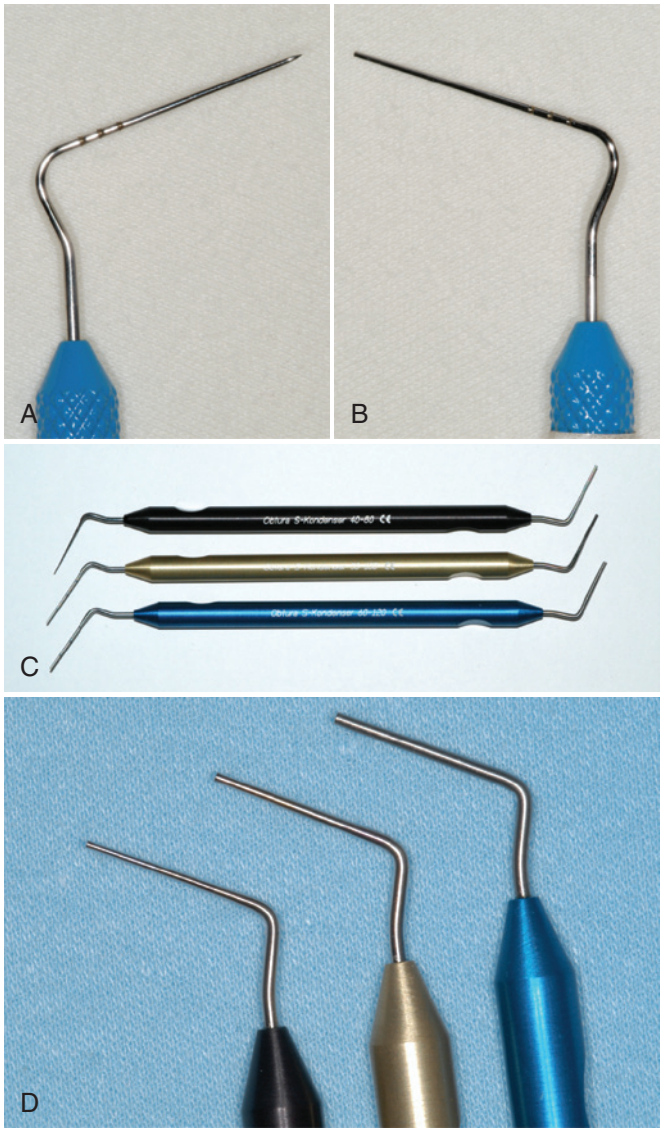

FIG. 7-30 Various pluggers are manufactured for compacting warm gutta-percha. **A**, ISO standard spreader. **B**, ISO standard plugger. **C**, Obtura S Kondensers. **D**, Closeup of Obtura S-Kondensers. (**C** courtesy Obtura Spartan, Algonquin, Illinois.)

One study measured temperature changes in a root canal system with warm vertical compaction.[40] The maximal temperatures occurred coronally and decreased apically. The authors reported that the maximal temperature in the canal was 118°C 8 mm from the apex. At 0 to 2 mm from the apical terminus, the maximal temperature had decreased to 44°C. Another study compared root surface temperatures for warm vertical obturation using the System B heat source, the Touch 'n Heat device, and a flame-heated carrier in maxillary and mandibular incisors and premolars 2 mm below the cemento-enamel junction. System B and the Touch 'n Heat produced a surface temperature rise that was less than 10°C for all maxillary incisors and premolar teeth. The Touch 'n Heat produced a greater than 10°C rise in mandibular incisors. The flame-heated carrier produced temperature changes greater than 10°C in all experimental teeth. Because the critical level of root surface heat required to produce irreversible bone damage is believed to be greater than 10°C the findings suggest that warm vertical compaction with the System B should not damage supporting periodontal structures; however, caution should be exercised with the Touch 'n Heat and flame-heated carriers.[219] The flame-heated spreader has no heat controls and the Touch 'n Heat controls only send heat to the tip and receive no feedback from the tip based on clinical conditions. The System B has a computer built in which allows it to receive feedback from the tip and adjust the tip temperature based on the clinical conditions, which allow the tip to remain at the dialed-in temperature.

The potential for vertical root fracture is also present with warm vertical compaction.[39] The forces developed appear to be equal to lateral compaction. Investigators compared warm vertical compaction and lateral compaction as a function of time. Results indicated that the forces developed with the two techniques were not significantly different. In a follow-up study, the mean value for wedging with warm vertical compaction was 0.65 ± 0.07 kg, whereas for lateral compaction it was 0.8 ± 0.1 kg.

Warm thermoplastic techniques have the advantage of producing movement of the plasticized gutta-percha within the obturated root canal system, resulting in a more homogeneous mass of gutta-percha, and filling irregularities and accessory canals better than lateral compaction.[105,421] This was illustrated in a study that reported a correlation between the quality of adaptation and the depth of heat application and canal size. Heat application close to the apical extent of the preparation produced the best results, and adaptation was better in small canals when compared with wide canals.[420] However, thermoplasticized techniques resulted in more extrusion of obturating materials.[213] There appeared to be no consistent differences between the techniques in sealing the canal space.[421]

Advantages of warm vertical compaction include filling of canal irregularities and accessory canals. Disadvantages include a slight risk of vertical root fracture because of compaction forces, less length control than with lateral compaction, and the potential for extrusion of material into the periradicular tissues. Warm vertical compaction is also difficult in more curved root canal systems, where the rigid pluggers may be unable to penetrate to the necessary depth. Occasionally, the canals have to be enlarged and tapered more to enable rigid carriers to penetrate within 4 to 5 mm of the apex. Additional removal of root canal dentin can weaken the root, making it more susceptible to root fracture.

Continuous Wave Compaction Technique

A variation of warm vertical compaction is the continuous wave compaction technique.[55] The increasing use of nickel-titanium rotary preparation techniques and the fabrication of greater taper standard cones have resulted in more clinicians using thermoplasticizing techniques. The manufacturing of gutta-percha cones to mimic the tapered preparation permits the application of greater hydraulic force during compaction when appropriately tapered pluggers are used. The continuous wave compaction technique employs the System B connected to 0.04, 0.06, 0.08, 0.10, or 0.12 tapered stainless steel dead-soft pluggers (see Fig. 7-33). The 0.06 tapered plugger also approximates the fine nonstandard gutta-percha cone, the 0.08 plugger the fine-medium cone, the 0.10 plugger the medium cone, and the 0.12 plugger the medium-large cone. Pluggers consistent with the ProFile GT instruments (DENTSPLY Tulsa

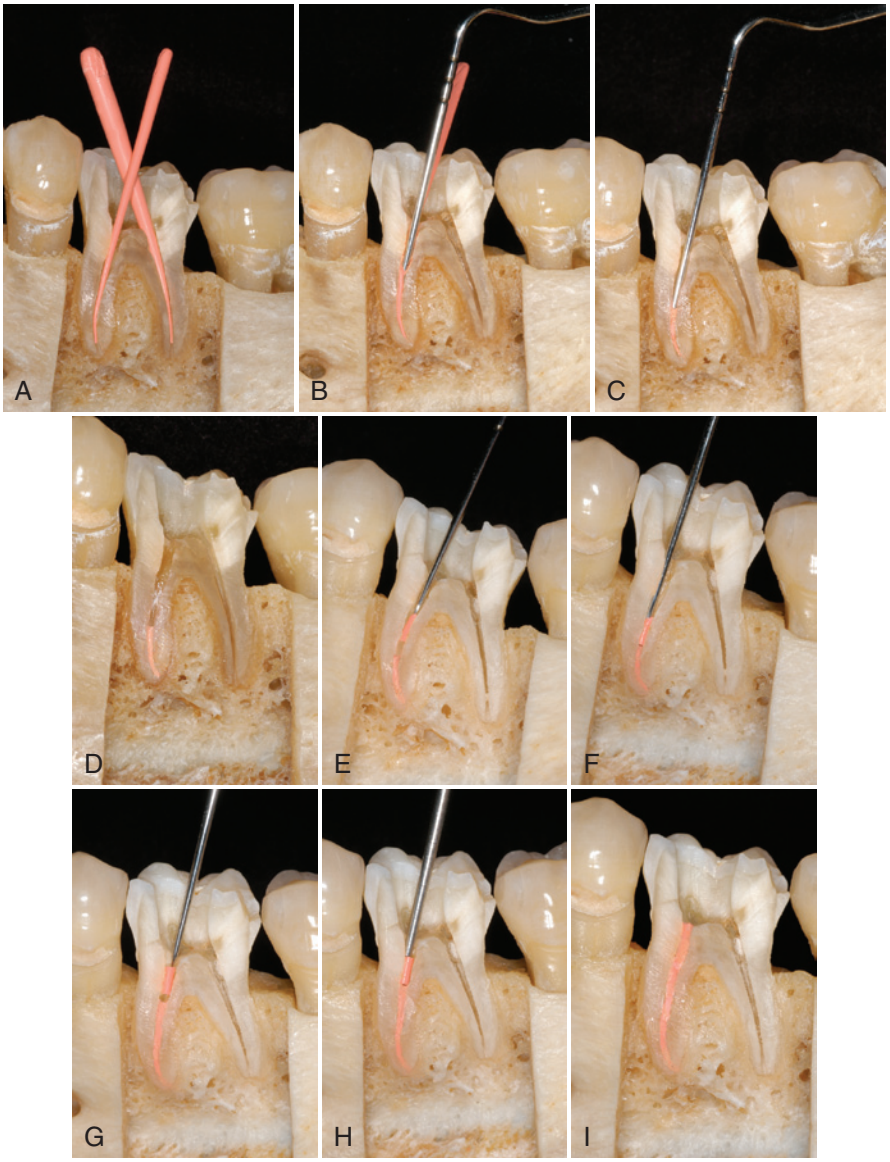

FIG. 7-31 Warm vertical compaction of gutta-percha employs heat and various condensers. **A,** Non-standard cones are selected and fit short of the prepared length because they more closely replicate the prepared canal. **B,** Heated pluggers or spreaders are used to apply heat to the master cone and remove the excess coronal material. **C,** A room temperature plugger is used to compact the heated gutta-percha. **D,** Apical compaction is complete. **E,** A gutta-percha segment is placed in the canal, and heat is applied. **F,** The heated segment is compacted. **G,** The process is repeated for the coronal portion of the canal by placing and heating a segment of gutta-percha. **H,** A plugger is again used to compact the heated material. **I,** Completed obturation.

FIG. 7-32 The Touch 'n Heat unit. (Courtesy SybronEndo, Orange, Calif.)

Dental Specialties), and Autofit gutta-percha cones (SybronEndo) are also available.

The electric heat source permits a variable temperature setting. The recommended temperature setting for the System B unit is 200°C. One study evaluated internal and external temperature changes with the System B unit with varied tips and temperature settings of 200°C, 250°C, and 300°C. At 6 mm from the apex, the System B unit set at 300°C with a fine-medium plugger produced the highest mean internal temperature (74°C). However, the authors noted that the external temperature setting never exceeded the critical 10°C rise at the PDL with any temperature setting or tip configuration.[375] This was confirmed in another study that measured temperature changes 2 mm apical to the cementoenamel junction and 1.5 mm from the apex. Results indicated that temperature changes apically were negligible. The mean change near the cementoenamel junction was 4.1°C.[398]

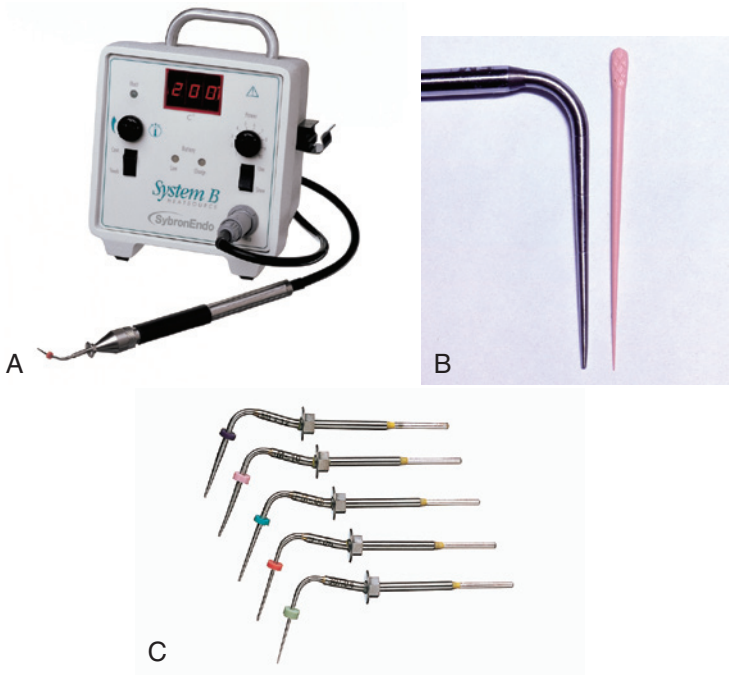

FIG. 7-33 Continuous wave obturation uses the System B unit. **A,** The System B unit. **B,** System B plugger with a nonstandard cone of similar taper. **C,** System B pluggers. (Courtesy SybronEndo, Orange, Calif.)

Another study reported that obturation temperature elevations produced during obturation with System B were significantly less than with traditional warm vertical compaction. An elevation of external root surface temperature by more than 10°C was noted with vertical compaction.[357] Investigators measured the root surface temperatures while using the System B heat source at various temperature settings from 250°C to 600°C. Results indicated that the highest temperature occurred 5 mm from the apex, and this was the only site that exceeded the 10°C rise. On the basis of this study, a temperature setting of 250°C or greater may be potentially hazardous.[101] For example, investigators using a thermocouple and simultaneous infrared analysis of temperatures found that the root surface temperature averaged 13.9°C, whereas the infrared technology indicated a 28.4°C rise at the same sites.[246]

After selecting an appropriate master cone, a plugger is prefitted to within 5 to 7 mm of the prepared length (Fig. 7-34). Placing the plugger deeper into the canal may enhance the flow of gutta-percha according to one investigator.[5] The point of plugger binding should be noted because once the instrument reaches this point the hydraulic forces on the gutta-percha will decrease and recompaction forces on the root will increase. There appears to be a correlation between the depth of the heated plugger relative to the working length and the quality of obturation and filling of canal irregularities.[46,190,424] Increasing the temperature settings does not seem to increase the effectiveness of obturation.[190]

The System B unit is set to 200°C in the touch mode. The plugger is inserted into the canal orifice and activated to remove excess coronal material. Compaction is initiated by placing a cold plugger against the gutta-percha at the canal orifice. Firm pressure is applied and heat is activated with the device. The plugger is moved rapidly (1 to 2 s) to within 3 mm of the binding point (Fig. 7-35). The heat is inactivated while firm pressure is maintained on the plugger for 5 to 10 seconds.

FIG. 7-34 System B plugger fit.

After the gutta-percha mass has cooled, a one-second application of heat separates the plugger from the gutta-percha and it is removed. The pluggers are designed to heat from the tip to their shank, which decreases the potential for dislodging the compacted mass and prevents a second application of heat to the material. Confirmation that the apical mass of gutta-percha is still present in the canal can be established with hand pluggers and/or radiographs. Two hand instruments are manufactured with tip diameters of 0.4 and 0.9 mm and 0.7 and 1.4 mm. It should be noted that with the continuous wave technique the heat source is placed only to within 5 to 7 mm from the tip of the gutta-percha; the apical portion of the gutta-percha remains essentially a single-cone technique as the heat transfer does not take place in the apical 2 to 5 mm of the gutta-percha.[145]

FIG. 7-35 System B activation and compaction.

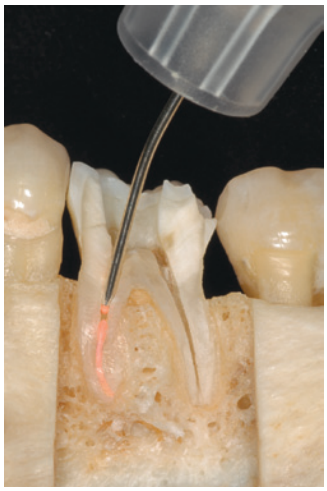

FIG. 7-36 Backfill by the Obtura II thermoplastic injection technique.

In ovoid canals, where the canal configuration may prevent the generation of hydraulic forces, an accessory cone can be placed alongside the master cone before compaction. With type II canals, the master cones are placed in both canals before compaction. A hand plugger is used to stabilize the cone in one canal while the other is being obturated.

Filling the space left by the plugger may be accomplished by a thermoplastic injection technique (Obtura or Ultrafil 3D [Coltène/Whaledent], Calamus [DENTSPLY Tulsa Dental Specialties], Elements [SybronEndo], or HotShot [Discus Dental, Los Angeles, California]) (Fig. 7-36)[187] or by fitting an accessory cone into the space with sealer, heating it, and compacting by short applications of heat and vertical pressure.

Warm Lateral Compaction

Warm lateral compaction of gutta-percha provides for predictable length control, which is an advantage over thermoplastic techniques. The Endotec II device (Medidenta, Las Vegas, Nevada) provides the clinician with the ability to employ length control while incorporating a warm gutta-percha technique (Fig. 7-37). Investigators demonstrated that the Endotec II produced a fusion of the gutta-percha into a solid homogeneous mass.[182] One study evaluated three thermoplasticized filling techniques and lateral compaction, using a bacterial

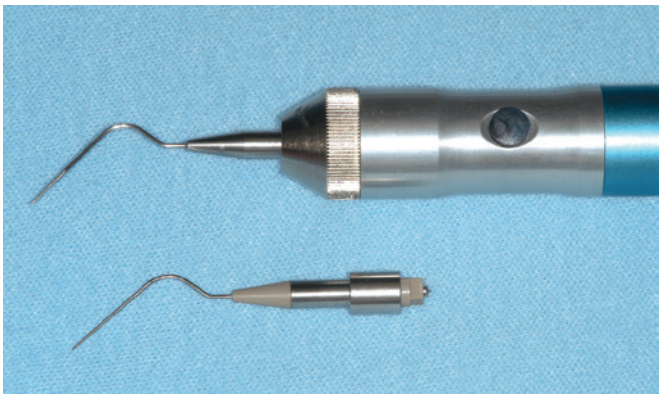

FIG. 7-37 Endotec II device (Medidenta) for warm lateral compaction.

FIG. 7-38 The Rootbuddy device (previously the DownPak device) for heat softening and vibrating gutta percha. (Courtesy Nikinc Dental, Eindhoven, The Netherlands.)

metabolite model, and found the Endotec to be superior to lateral compaction alone, lateral thermocompaction, and the Ultrafil 3D.[197] The use of warm lateral compaction with the Endotec demonstrated an increased weight of gutta-percha mass, by 14.63%, when compared with traditional lateral compaction.[227] Another study found a 24% increase in weight with warm lateral compaction when using the System B device.[260] Using the Endotec II, one investigation reported a statistically significantly better ability of warm vertical and warm lateral compaction techniques, versus cold lateral compaction, to reproduce artificially produced canal irregularities in a split-tooth model.[81] Another group of investigators used the EndoTwinn (Hu-Friedy), an instrument for warm lateral compaction, in a similar experiment. The EndoTwinn instrument also possesses the ability to vibrate the electronically heated tip. They reported that warm lateral compaction, using both heat and vibration, and warm vertical compaction of gutta-percha provided statistically better replication of defects than cold lateral compaction.[211] Hu-Friedy introduced the DownPak* (Fig. 7-38), a variation of the original EndoTwinn that can be used with either warm lateral or warm vertical compaction techniques. Other investigators compared the stress generated with lateral compaction and warm lateral compaction, using the Endotec II, and found that the warm lateral compaction technique created less stress during obturation.[242] An additional concern is the heat generated by the technique. Evaluation of the effects of warm lateral and warm vertical compaction on periodontal tissues demonstrated that neither technique produced heat-related damage.[69]

*Downpak products and service are now supplied by Nikinc Dental, Eindhoven, The Netherlands. It's new name is RootBuddy.

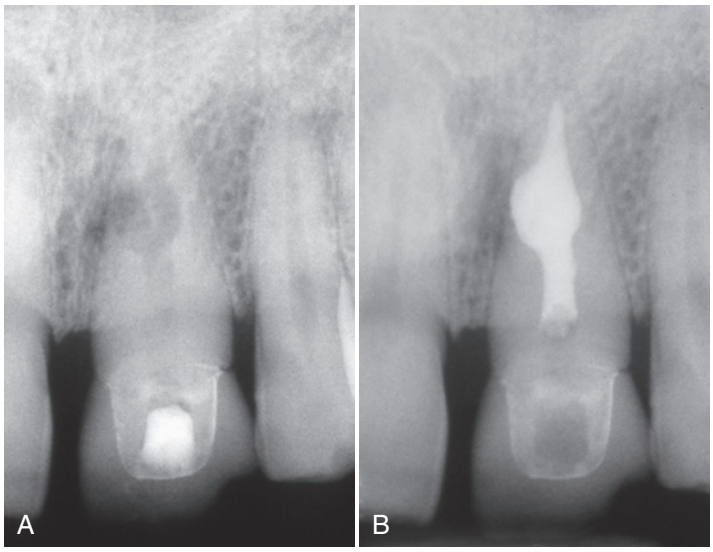

FIG. 7-39 Thermoplastic techniques are often used in cases with significant canal irregularities. **A**, A pretreatment radiograph of a maxillary central incisor exhibiting internal resorption. **B**, Posttreatment radiograph demonstrates a dense obturation of the resorptive defect with gutta-percha.

FIG. 7-40 Obtura III Max System. (Courtesy Kerr, Orange, CA.)

FIG. 7-41 The Calamus thermoplastic unit for heating and injecting gutta-percha. (Courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, Okla.)

The warm lateral compaction technique involves adapting a master cone in the same manner as with traditional lateral compaction. An appropriate-size Endotec II tip is selected. Endotec II tips are available in various taper and tip diameters. The sizes consist of #0.02/20 and #0.02/40. The device is activated and the tip is inserted beside the master cone to within 2 to 4 mm of the apex, using light pressure. The tip is rotated for 5 to 8 seconds and removed. An unheated spreader can be placed in the channel created to ensure adaptation and then an accessory cone is placed. The process is continued until the canal is filled.

Thermoplastic Injection Techniques

Heating of gutta-percha outside the tooth and injecting the material into the canal is an additional variation of the thermoplastic technique (Fig. 7-39). The Obtura III (Fig. 7-40), Calamus (Fig. 7-41), Elements (Fig. 7-42), HotShot (Fig. 7-43), and Ultrafil 3D (Fig. 7-44) are available devices. The Obtura system heats the gutta-percha to 160°C, whereas the Ultrafil 3D system employs a low-temperature gutta-percha that is heated to 90°C.

Obtura III

The Obtura III system (Obtura Spartan) consists of a hand-held "gun" that contains a chamber surrounded by a heating element into which pellets of gutta-percha are loaded (see Fig. 7-40). Silver needles (varying gauges of 20, 23, and 25) are attached to deliver the thermoplasticized material to the canal. The control unit allows the operator to adjust the temperature and thus the viscosity of the gutta-percha. At 6 mm from the apex a study found that the highest internal temperature with the Obtura system was 27°C.[375]

Canal preparation is similar for other obturation techniques. The apical terminus should be as small as possible to prevent extrusion of gutta-percha. The technique requires the use of sealer, and once the canal is dried, the canal walls are coated with sealer, using the last file used to length or a paper point. Gutta-percha is preheated in the gun, and the needle is positioned in the canal so that it reaches within 3 to 5 mm of the apical preparation. Gutta-percha is then gradually, passively injected by squeezing the trigger of the "gun." The needle

FIG. 7-42 The Elements obturation unit for injecting and compacting gutta-percha. (Courtesy Kerr, Orange, CA.)

FIG. 7-43 The battery-powered HotShot unit (Discus Dental) for heating and injecting gutta-percha.

FIG. 7-44 The Ultrafil 3D system consists of an injection syringe, gutta-percha cannulas, and heating unit (Coltène/Whaledent).

backs out of the canal as the apical portion is filled. Pluggers dipped in alcohol are used to compact the gutta-percha. A segmental technique may also be used, in which 3- to 4-mm segments of gutta-percha are sequentially injected and compacted. In either case, compaction should continue until the gutta-percha cools and solidifies to compensate for the contraction that takes place on cooling.

The difficulties with this system include lack of length control. Both overextension and underextension are common results. To overcome this drawback, a hybrid technique may be used, in which the clinician begins filling the canal by the lateral compaction technique. When the master cone and several accessory cones have been placed so that the mass is firmly lodged in the apical portion of the canal, a hot plugger is introduced, searing the points off approximately 4 to 5 mm from the apex. Light vertical compaction is applied to restore the integrity of the apical plug of gutta-percha. The remainder of the canal is then filled with thermoplasticized gutta-percha injected as previously described.

Investigators studied, at 3, 6, and 12 months post treatment, the success rate of 236 teeth obturated with the Obtura system. Results indicated that 96% of the cases were successful, with the highest success rate being in teeth filled flush with the apex (97%) when compared with overextensions (93%) and filling short (93%).[377] Another study compared lateral compaction with Thermafil (DENTSPLY Tulsa Dental Specialties) and Obtura in root canal models and found that the Obtura produced the best adaptation to the canal walls.[408] Other investigators found that continuous wave obturation with the Obtura backfill initially produced a better bacterial seal when compared with lateral compaction, using bilaterally matched teeth and an anaerobic bacterial leakage model.[182]

Ultrafil 3D

Ultrafil 3D (Coltène/Whaledent) is a thermoplastic gutta-percha injection technique involving gutta-percha cannulas, a heating unit, and an injection syringe (see Fig. 7-44). The system employs three types of gutta-percha cannulas. The Regular Set is a low-viscosity material that requires 30 minutes to set. The Firm Set is also a low-viscosity material but differs in that it sets in 4 minutes. The manufacturer recommends compaction after the initial set with both materials. Endoset has a higher viscosity and does not flow as well. It is recommended for techniques employing compaction and sets in 2 minutes. The heater is preset at 90°C and does not require adjustment.

Each cannula has a 22-gauge stainless steel needle that measures 21 mm in length. The needles can be precurved. Cannulas can be disinfected but are not designed for heat sterilization procedures. Heating time varies, but for a cold unit it takes 10 to 15 minutes. In a warm heater the recommended time is 3 minutes. After removing the cannula from the heater the needle should be placed on the hot part of the heater for several seconds. The gutta-percha remains able to flow for 45 to 60 seconds depending on the viscosity.

Calamus

The Calamus flow obturation delivery system (DENTSPLY Tulsa Dental Specialties) is a thermoplastic device equipped with a cartridge system with 20- and 23-gauge needles (see Fig. 7-41). The unit permits control of temperature and also the flow rate. Pluggers are also available for use with the

system. The 360-degree activation switch allows great tactile sensation during use.

Elements

The Elements obturation unit (SybronEndo) consists of a System B heat source and plugger as well as a handpiece extruder for delivering thermoplastic gutta-percha or RealSeal from a disposable cartridge (see Fig. 7-42). The cartridges come with 20-, 23-, and 25-gauge needles for gutta-percha and 20- and 23-gauges for RealSeal.

HotShot

The HotShot delivery system (Discus Dental [now part of Philips Oral Healthcare]) is a cordless thermoplastic device that has a heating range from 150°C to 230°C (see Fig. 7-43). The unit is cordless and can be used with either gutta-percha or Resilon. Needles are available in 20, 23, and 25 gauges.

Carrier-Based Gutta-Percha

Thermafil, Profile GT Obturators, GT Series X Obturators, and ProTaper Universal Obturators

Thermafil (DENTSPLY Tulsa Dental Specialties) was introduced as a gutta-percha obturation material with a solid core (Fig. 7-45). Originally manufactured with a metal core and a coating of gutta-percha, the carrier was heated over an open flame. The technique was popular because the central core provided a rigid mechanism to facilitate the placement of the gutta-percha. Advantages included ease of placement and the pliable properties of the gutta-percha. Disadvantages were that the metallic core made placement of a post challenging and retreatment procedures were difficult. In addition, the gutta-percha was often stripped from the carrier, leaving the carrier as the obturating material in the apical area of the canal. Reports have stated that carrier-based systems result in minimal gaps and voids in the resulting obturation mass.[441] Other reports have stated that there was no difference in healing rates between carrier-based systems and lateral compaction of gutta-percha.[157]

Changes in the carrier systems include the development of a plastic core coated with α-phase gutta-percha (Fig. 7-46) and a heating device that controls the temperature (Fig. 7-47). Obturators are designed to correspond to the ISO standard file sizes, variable tapered nickel titanium rotary files, and the ProFile GT and GT Series X nickel-titanium rotary files (DENTSPLY Tulsa Dental Specialties) (see Fig. 7-46). Size verifiers are available to aid in selection of the appropriate carrier and should fit passively at the corrected working length (see Fig. 7-45).

As with all techniques, a sealer is required. Grossman formulation sealers and resin sealers consistent with AH-26 and AH Plus are acceptable; however, Tubli-Seal and Wach's paste are not recommended.

Removal of the smear layer is strongly recommended (see Chapters 5 and 7) and has been shown to enhance the seal with Thermafil.[29] After drying the canal a light coat of sealer is applied and a carrier is marked, set to the predetermined length. This is accomplished by using the millimeter calibration markings on the carrier shaft. Markings are made at 18, 19, 20, 22, 24, 27, and 29 mm. Gutta-percha on the shaft that may be obscuring the calibration rings can be removed with a surgical blade or knife. The carrier is disinfected with 5.25% NaOCl for 1 minute and rinsed in 70% alcohol.

The carrier is then placed in the heating device. When the carrier is heated to the appropriate temperature, the clinician

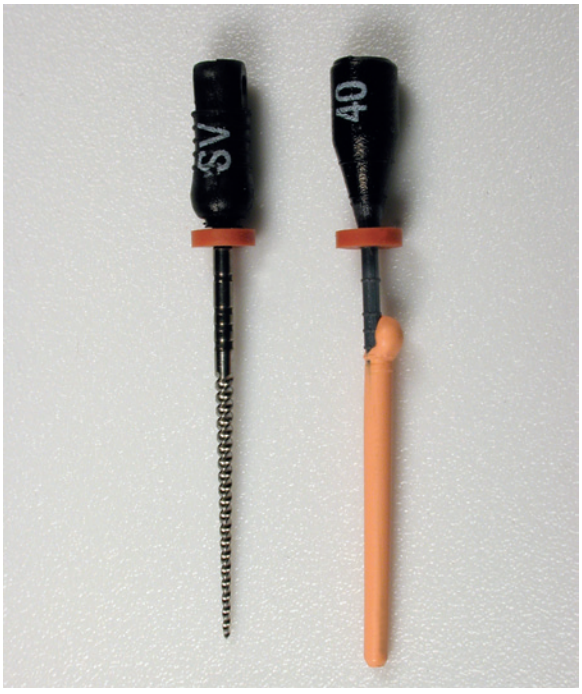

FIG. 7-45 Thermafil carrier and size verifier (DENTSPLY Tulsa Dental Specialties).

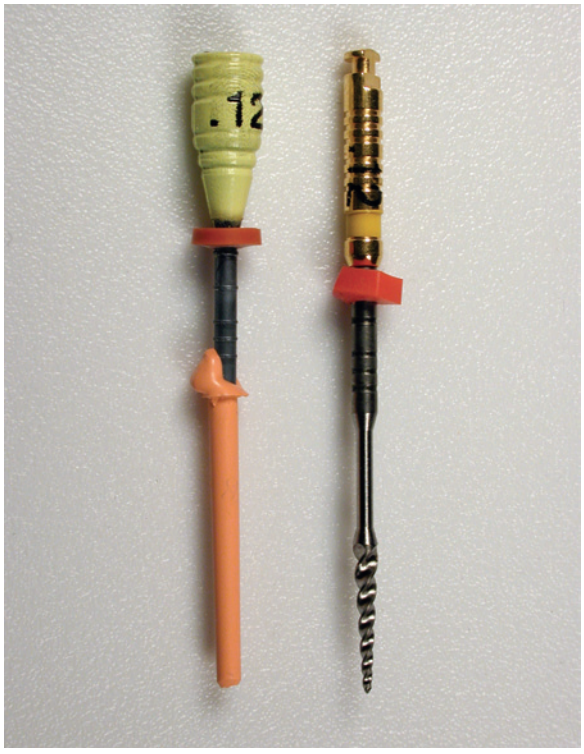

FIG. 7-46 GT obturator and instrument (DENTSPLY Tulsa Dental Specialties).

FIG. 7-47 The Thermafil oven with carrier in place (Thermaprep2 oven. Courtesy DENTSPLY Maillefer, Ballaigues, Switzerland.)

FIG. 7-48 Thermafil carrier placed in the distal canal.

has approximately 10 seconds to retrieve it and insert it into the canal (Fig. 7-48). This is accomplished without rotation or twisting. Evidence suggests that the insertion rate affects the obturation. The fill length and obturation of irregularities increase with increasing insertion rates.[225] A rapid insertion rate enhances obturation.[225]

The position of the carrier is verified radiographically. The gutta-percha is allowed 2 to 4 minutes to cool before resecting the coronal portion of the carrier, which can be several millimeters above the canal orifice. This is accomplished by applying stabilizing pressure to the carrier and cutting the device with an inverted cone, round bur, or a specially designed Prepi bur (DENTSPLY Tulsa Dental Specialties). Heated instruments are not recommended for this process because this may result in displacement.

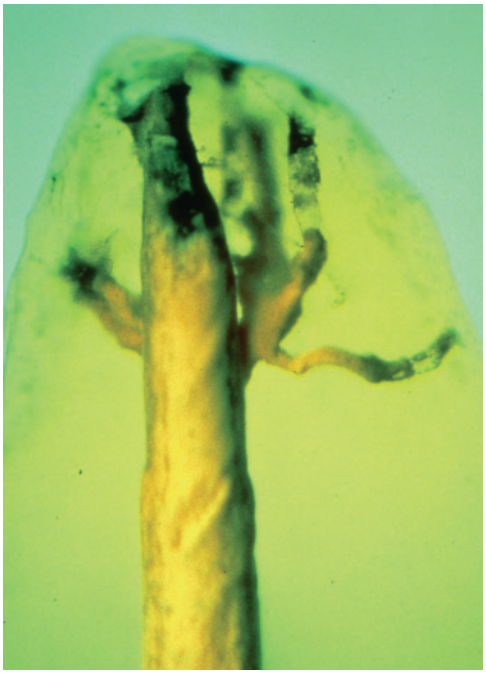

FIG. 7-49 Apical obturation of accessory canals by the Thermafil technique. (Courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, Okla.)

Vertical compaction of the coronal gutta-percha can then be accomplished. When necessary, gutta-percha can be added, heat softened, and compacted. An advantage to this technique is the potential for movement of gutta-percha into lateral and accessory canals (Fig. 7-49)[419]; however, extrusion of material beyond the apical extent of the preparation is a disadvantage.[78,85,213]

Pro-Post drills (DENTSPLY Tulsa Dental Specialties) are recommended if post space is required for restoration of the tooth. The unique eccentric cutting tip keeps the instrument centered in the canal while friction softens and removes the gutta-percha and plastic carrier.

When retreatment is necessary the plastic carrier has a groove along its length to provide an access point for placement of a file. Chloroform and hand files can be used to remove the gutta-percha surrounding the carrier. Rotary #.04 and #.06 nickel-titanium files may also be used to remove the obturation materials. Retreatment rotary nickel-titanium files are available in three different sizes to facilitate removal of gutta-percha and the carrier.

The plastic carriers are composed of two nontoxic materials. Sizes #20 to #40 are manufactured from a liquid crystal plastic. Sizes #40 to #90 are composed of polysulfone polymer. Both have similar physical characteristics, with the polysulfone carriers being susceptible to dissolution in chloroform.

GuttaCore (DENTSPLY Tulsa Dental Specialties), the latest generation of core carriers, uses cross-linked gutta-percha as the carrier of the outer thermoplasticized gutta-percha. This makes retreatment simpler as the clinician can simply drill through the carrier to gain access into the canal space. A recent in vitro study comparing the time required to remove GuttaCore, Thermafil Plus and thermoplasticized gutta-percha from moderately curved root canals with ProTaper retreatment files reported that GuttaCore was removed in significantly less time when compared with the other two techniques.[26]

FIG. 7-50 SuccessFil is an additional carrier system (Coltène/Whaledent).

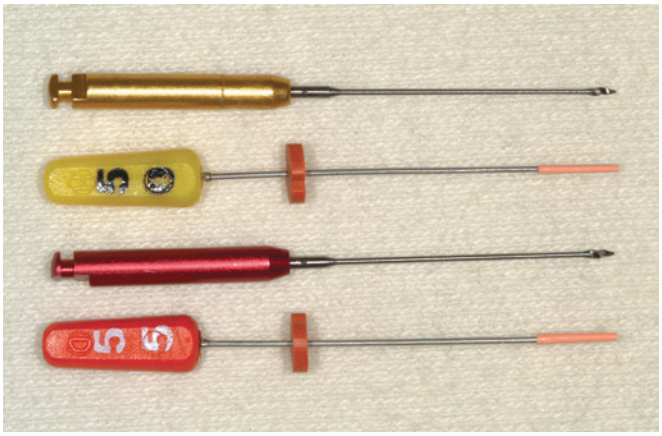

FIG. 7-51 SimpliFill carrier (SybronEndo) and LightSpeed file (LightSpeed Technology, San Antonio, Texas).

SuccessFil

SuccessFil (Coltène/Whaledent) is a carrier-based system associated with Ultrafil 3D (Fig. 7-50); however, the gutta-percha used in this technique comes in a syringe. Carriers (titanium or radiopaque plastic) are inserted into the syringe to the measured length of the canal. The gutta-percha is expressed on the carrier, with the amount and shape determined by the rate of withdrawal from the syringe. Sealer is lightly coated on the canal walls, and the carrier with gutta-percha is placed in the canal to the prepared length. The gutta-percha can be compacted around the carrier with various pluggers depending on the canal morphology. This is followed by severing of the carrier slightly above the orifice with a bur.

SimpliFill

SimpliFill (SybronEndo) is gutta-percha or Resilon manufactured for use after canal preparation with LightSpeed instruments (Fig. 7-51). The carrier has an apical plug with 5 mm of gutta-percha. The technique involves fitting a carrier that is consistent with the master apical rotary file (SybronEndo) to within 1 to 3 mm of the prepared length (Fig. 7-52). The apical gutta-percha plug can be modified by clipping the end in 1-mm increments to obtain an appropriate fit if the plug is too small.

FIG. 7-52 SimpliFill fitted to 1 to 3 mm from the prepared length.

Once the cone is fitted it is withdrawn and sealer is applied to the canal walls. AH Plus is recommended. The SimpliFill carrier is slowly advanced to the prepared length. This may require firm pressure. With the plug at the corrected working length the handle is quickly rotated a minimum of four complete terms in a counterclockwise direction to separate the shaft from the apical gutta-percha. The coronal space can then be filled with gutta-percha, using lateral compaction or the warm thermoplastic technique. When using lateral compaction it is recommended that the first cone be the same size as the SimpliFill carrier. This sectional technique is efficient, and leakage potential is similar to that of other common techniques.[324]

Thermomechanical Compaction

McSpadden introduced an instrument, the McSpadden Compactor, with flutes similar to a Hedström file but in reverse. When activated in a slow-speed handpiece, the instrument would generate friction, soften the gutta-percha, and move it apically. Rotary compactors similar in design have been developed and advocated. To increase flexibility, the instrument is available in nickel-titanium.

The technique requires fitting a master cone short of the prepared length and applying sealer. A compactor is selected on the basis of the size of the canal and inserted alongside the gutta-percha cone 3 to 4 mm from the prepared length. The handpiece is activated, and the friction of the rotating bur heats the gutta-percha. The pliable mass is compacted apically and laterally as the device is withdrawn from the canal.

Advantages include simplicity of the armamentarium, the ability to fill canal irregularities,[161,198,232,331,332] and time. Disadvantages include possible extrusion of material, instrument fracture,[270] gouging of the canal walls, the inability to use the technique in curved canals, and possible excessive heat generation.[27,124,159,245,332,333] Microseal condensers (SybronEndo) and the Gutta Condenser (DENTSPLY Tulsa Dental Specialties) are variations of this product.

Solvent Techniques

Gutta-percha can be plasticized with solvents, such as chloroform, eucalyptol, or xylol. Disadvantages include shrinkage caused by evaporation, voids, the inability to achieve predictable length control of the obturating material, and irritation of periradicular tissues. The Callahan and Johnston technique involved dissolving gutta-percha in chloroform and placing the mixture into the canal with a syringe.[60] A gutta-percha cone was then softened by immersion in this mixture and placed into the canal and the mass solidified as the solvent evaporated. Unfortunately, shrinkage occurred with the evaporation process.[299] Obturation techniques using solvents have been abandoned and replaced with materials and methods that exhibit minimum shrinkage.

Pastes

Pastes fulfill some of the criteria outlined by Grossman[149] and can adapt to the complex internal canal anatomy; however, the flow characteristic can result in extrusion or incomplete obturation. The inability to control the material is a distinct disadvantage, and when extrusion occurs it can be corrected only by surgical intervention. In addition, pastes are sometimes used as a substitute for complete cleaning and shaping procedures, and the addition of paraformaldehyde results in severe toxicity.

Immediate Obturation

Apical barriers may be necessary in cases with immature apical development, cases with external apical root resorption, and cases where instrumentation extends beyond the confines of the root. Dentin chips, calcium hydroxide, demineralized dentin, lyophilized bone, tricalcium phosphate, hydroxyapatite, and collagen have been advocated for placement as a barrier in canals exhibiting an open apex. The barriers are designed to permit obturation without extrusion of materials into the periradicular tissues but are often incomplete and do not seal the canal.[320]

Dentin chips appear to confine materials to the canal space during instrumentation/obturation and may encourage development of a biologic seal.[115,320] Enhanced healing, minimal inflammation, and apical cementum deposition have been noted histologically.[275] A concern with this technique is contamination of the dentin with bacteria or other nonhost materials, because investigators found that infected dentin adversely affected healing.[173]

Calcium hydroxide has also been extensively used as a common apical barrier. Calcium hydroxide has been shown to induce an apical barrier in apexification procedures. Calcifications similar to dentin plugs have been noted at the apical foramen.[295] Calcium hydroxide has the advantage of being free of bacterial contamination and may provide a better, although imperfect, apical seal.[406]

Immature teeth exhibiting pulp necrosis or teeth with apical resorption traditionally were treated with calcium hydroxide to establish an apical barrier (apexification) before obturation. Studies have demonstrated that teeth treated with calcium hydroxide for prolonged periods are more susceptible to fracture.[11,316] Immediate obturation is an alternative to apexification. An apical barrier material should confine obturation materials to the canal space[115] and enhance healing by inducing cementum and bone formation.[172,275,295] Mineral trioxide aggregate (MTA) (ProRoot MTA; DENTSPLY Tulsa Dental Specialties) has been successfully employed as an apical barrier material before obturation (Fig. 7-53).[350]

FIG. 7-53 Mineral trioxide aggregate is available as ProRoot MTA. This material is advocated for use in apexification, repair of root perforations, repair of root resorption, root-end fillings, and pulp capping. (Courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, Okla.)

After cleaning and shaping procedures, the root canal system is dried and a small increment of MTA is placed to the radiographic apex and the location verified through a radiograph. If the material is overextended, it can be easily irrigated out with sterile saline. If it is short of the radiographic apex, it can be compacted with prefitted pluggers to move it to the radiographic apex. The material is compacted into the apical portion of the root to form a barrier. After the material sets, any thermoplasticized technique can be used to backfill with gutta-percha without concern of overextension (Fig. 7-54). Other investigators have reported that hand compaction of MTA provided better adaptation to the canal walls with fewer voids when compared with ultrasonic placement.[9] Another investigation reported that ultrasonic placement of a 4-mm apical plug of MTA improved the seal[430] and that placement of a composite resin as the obturation material enhanced the seal and strengthened the root.[216]

Mineral trioxide aggregate is sterile, biocompatible, and capable of inducing hard tissue formation.[112,174] The technique has been shown to be clinically successful and can be accomplished quickly, eliminating the need for numerous patient visits and possible coronal recontamination during the many months required for apexification.[73,87,138]

Clinical evidence supports this technique. In a study comparing calcium hydroxide with mineral trioxide aggregate in 15 children, each having paired immature teeth with necrotic pulps, two teeth treated with $Ca(OH)_2$ exhibited pathosis on recall examination, whereas all the teeth treated with MTA were clinically and radiographically successful.[114] A prospective clinical study found the success rate of MTA barriers and immediate obturation in 43 cases to be 81%.[358] In another study, 85% of 20 teeth with immature apical development were considered healed and 5% were considered healing after immediate barrier placement.[171] The time required for healing is

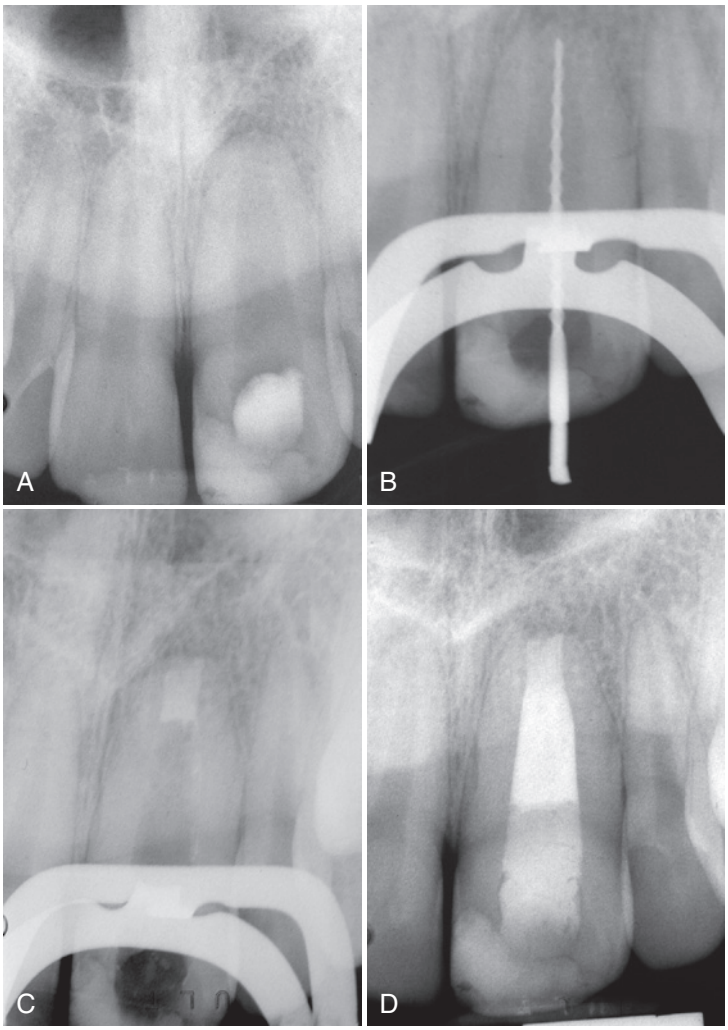

FIG. 7-54 Immediate obturation employs a barrier technique to prevent extrusion when the apex is open. This case involves a maxillary left central incisor with pulp necrosis caused by trauma. **A,** Pretreatment radiograph demonstrates a large canal with an open apex. **B,** Working length is established and the canal is prepared. **C,** Mineral trioxide plug is placed. **D,** The canal is obturated with gutta-percha.

comparable to traditional apexification with $Ca(OH)_2$ and treatment time is reduced.[298]

Another option for treating immature teeth with pulp necrosis involves regenerative endodontic techniques (see Chapter 10).[160,184,391,414] Advantages include continued increasing thickness of the canal walls, continued apical root development, and apical closure.[351] The technique involves copious irrigation, minimal canal preparation, and the use of an antibiotic paste as an interim medication. At a subsequent visit, bleeding is induced in the canal by placing a file beyond the root apex. When the bleeding comes within 3 mm of the canal orifice, the clot is lined with a resorbable membrane and then covered with MTA. When the MTA is set, a definitive restoration can be placed to ensure a coronal seal.[17]

CORONAL ORIFICE SEAL

No matter which technique is used to obturate the canals, coronal microleakage can occur within a short time through seemingly well-obturated canals,[68,389] potentially causing infection of the periapical area. Early research efforts focused on the quality of the seal in the apical part of the root canal system to prevent percolation of apical fluids. However, contemporary research efforts have identified the greater importance of maintaining a coronal seal to prevent bacterial leakage.[185] Leakage studies indicate that the coronal seal can be enhanced by the application of supplemental restorative materials over the canal orifice[293,407,418] and by placing a definitive coronal restoration as soon as is feasible.[305]

Cavit (3M ESPE) has traditionally been advocated as an acceptable material. One study demonstrated that placement of 3.5 mm of Cavit or Super EBA cement (Bosworth, Skokie, Illinois) decreased bacterial leakage by 85% and 65%, respectively, when compared with unsealed controls, which all leaked at 45 days.[293] In an animal study in which the access openings were left open for 8 months, placing a dentin-bonded composite resin or IRM in the orifice decreased periapical inflammation from 89% for teeth without orifice plugs to 39% with plugs.[428] Another study demonstrated in a dog model that MTA placed in the orifice decreased inflammation in teeth inoculated with bacteria.[235]

Another method to retard leakage through the obturated canal, should failure of the coronal restoration occur, is to cover the floor of the pulp chamber with a lining of bonded material after removal of excess gutta-percha and sealer to the canal orifice.[31,71,221] This results in the formation of a hybrid layer with microtags of resin in the tubules. A resin-modified glass ionomer cement is placed approximately 1 mm thick over the floor of the pulp chamber and polymerized with a curing light. Alternatively, a self-etching dentin adhesive and a flowable resin composite may be used. Investigators found that this procedure resulted in none of the experimental canals showing bacterial leakage at 60 days.[72] A material with a different color than dentin may be used for identification, in case subsequent placement of a post is required.

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PART II

The Advanced Science of Endodontics

CHAPTER 8
Nonsurgical Retreatment

CHAPTER 9
Periradicular Surgery

CHAPTER 10
Regenerative Endodontics

CHAPTER 11
Evaluation of Outcomes

CHAPTER 12
Structure and Functions of the Dentin-Pulp Complex

CHAPTER 13
Pulpal Reactions to Caries and Dental Procedures

CHAPTER 14
Microbiology of Endodontic Infections

CHAPTER 15
Pathobiology of Apical Periodontitis

CHAPTER 16
Root Resorption

CHAPTER 17
Diagnosis of Nonodontogenic Toothache

CHAPTER 18
Management of Endodontic Emergencies

CHAPTER 19
Managing Iatrogenic Endodontic Events

CHAPTER 8

Nonsurgical Retreatment

ROBERT S. RODA | BRADLEY H. GETTLEMAN

CHAPTER OUTLINE

Etiology of Posttreatment Disease
Diagnosis of Posttreatment Disease
Treatment Planning
Nonsurgical Endodontic Retreatment
Coronal Access Cavity Preparation
Post Removal
Regaining Access to the Apical Area
Removal of Separated Instruments
Heat Generation During Retreatment Procedures
Management of Canal Impediments
Finishing the Retreatment
Repair of Perforations
Prognosis of Retreatment
Summary

Nonsurgical root canal therapy has become a routine procedure in modern dentistry. Technical and scientific advances in endodontics have resulted in the retention of millions of teeth that would otherwise be lost. Even as advances in surgical and prosthetic restorative care have made tooth replacement less onerous than in the past, it is universally accepted that a natural tooth with a good prognosis is a superior choice to loss and replacement.

Unfortunately, not all treatments result in optimum long-term healing. Given the large numbers of treatments performed, the very small rate of unsuccessful outcomes translates into relatively large numbers of patients requiring further treatment. Dental clinicians should be able to diagnose persistent or reintroduced endodontic disease and be aware of the options for treatment. If they wish to approach treating these teeth, they should have the appropriate armamentarium and be capable of performing these specialized techniques at the highest level (Fig. 8-1). Also, clinicians must always have a scientifically sound, evidence-based rationale for every treatment decision that is made so that they may best serve the patients who entrust them with their care. The purpose of this chapter is to provide information to allow the reader to maximize the likelihood of success in the treatment of persistent endodontic disease.

ETIOLOGY OF POSTTREATMENT DISEASE

In the past, undesirable outcomes of endodontic therapy were described as failures. Clinicians quote failure rates based on published success/failure studies. Using the words *success* and *failure* may be a holdover from a time when clinicians felt they needed to congratulate themselves on their successes and blame themselves for the failures of their treatment endeavors. This thought process does not reflect reality and can be potentially destructive. There are many instances in which treatments performed at the highest level of clinical competence result in an undesirable outcome, and there are other instances in which a procedure is performed well below a scientifically acceptable standard and yet provides long-term success.[203] We must begin to dissect the science from emotion and ego, and this separation may start with nomenclature. Friedman stated that "most patients can relate to the concept of disease-treatment-healing, whereas failure, apart from being a negative and relative term, does not imply the necessity to pursue treatment."[64] He has suggested using the term *posttreatment disease* to describe those cases that would previously have been referred to as treatment failures. This will be the term used in the remainder of this chapter to describe persistent or reintroduced endodontic disease.

Almost 16 million root canal procedures were performed in 1999,[29] and, with success rates varying between 86% and 98%,[65,66] it has been shown to be a reliable treatment option. Conversely, the incidence of posttreatment disease, although small, translates into a large number of cases where further treatment is needed. When faced with such a situation, the clinician must determine the etiology of the persistent pathosis and devise a rationale and strategy for treatment.

There are many causes for "failure" of initial endodontic therapy that have been described in the endodontic literature (Fig. 8-2). These include iatrogenic procedural errors such as poor access cavity design, untreated canals (both major and accessory),[266] canals that are poorly cleaned and obturated,[37,112] complications of instrumentation (ledges, perforations, or separated instruments),[231] and overextensions of root-filling materials.[164] Coronal leakage[131,147,196,236,248] has also been blamed for posttreatment disease, as has persistent intracanal and extracanal infection[166,216,232] and radicular cysts.[162] These etiologies may be obvious at the time of diagnosing the diseased, root-filled tooth, or they may remain uncertain until the completion of successful therapy. Occasionally, the cause of posttreatment disease may take years to become discernible or may ultimately never be known. The most important causative factors for the clinician, however, are those related to treatment planning and determination of prognosis.

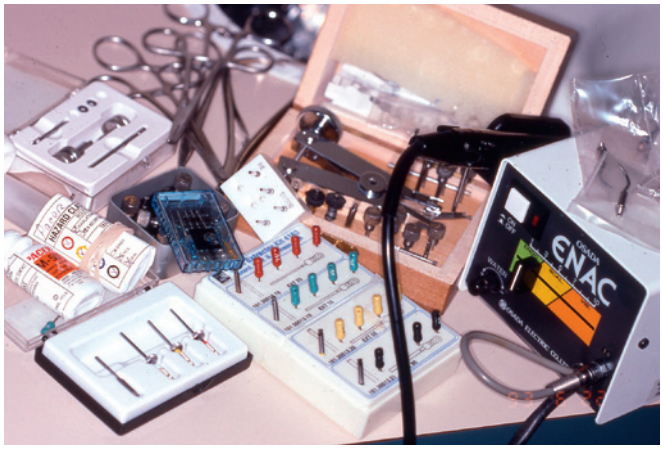

FIG. 8-1 Some of the armamentarium needed to perform retreatment at the highest level.

To treatment plan effectively, the clinician may place the etiologic factors into four groups[231] (Fig. 8-3):

1. Persistent or reintroduced intraradicular microorganisms
2. Extraradicular infection
3. Foreign body reaction
4. True cysts

Persistent or Reintroduced Intraradicular Microorganisms

When the root canal space and dentinal tubules are contaminated with microorganisms or their by-products and if these pathogens are allowed to contact the periradicular tissues, apical periodontitis ensues. As stated earlier, inadequate cleaning, shaping, obturation, and final restoration of an endodontically diseased tooth can lead to posttreatment disease. If initial endodontic therapy does not render the canal

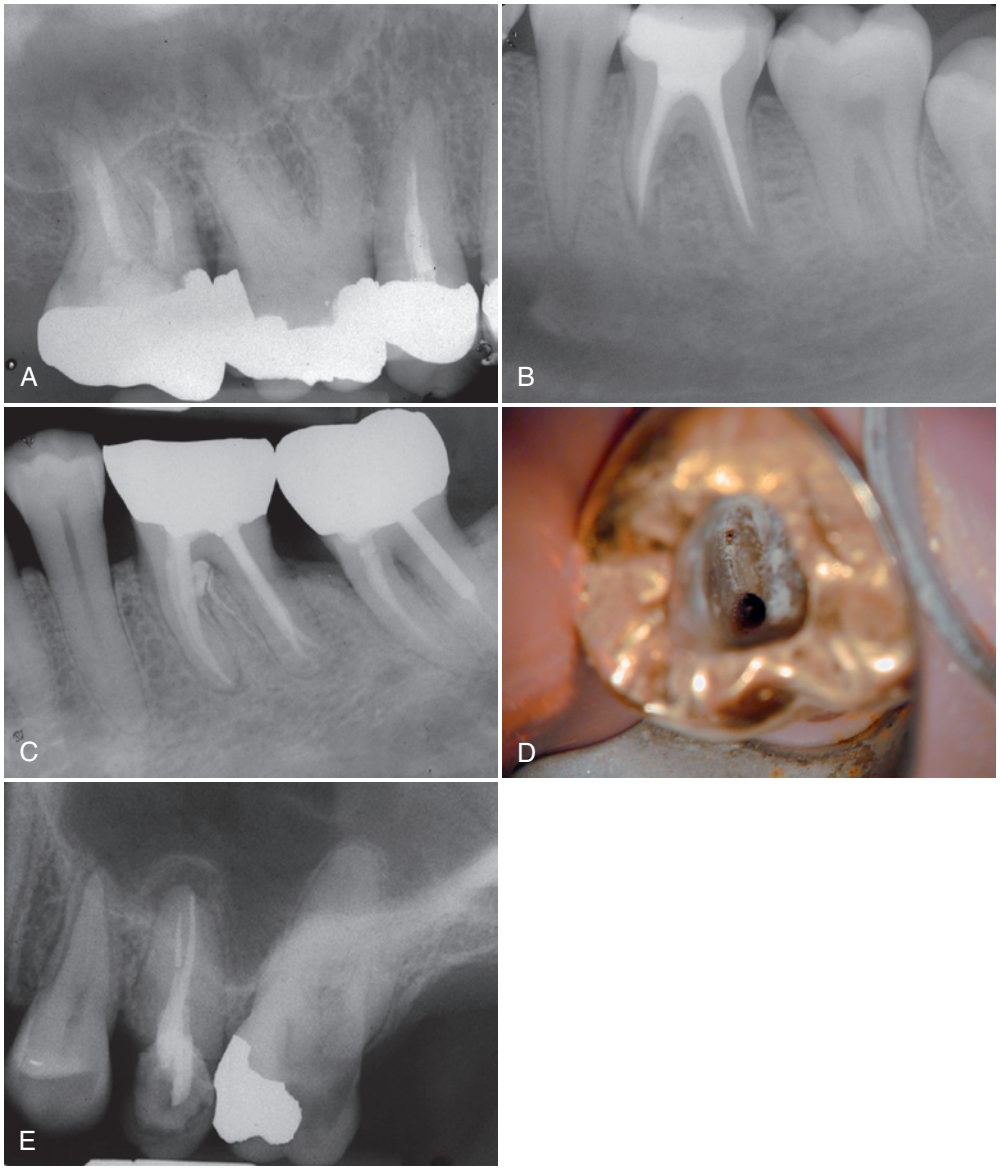

FIG. 8-2 Clinical presentations of posttreatment disease. A, Canals that are poorly cleaned, shaped, and obturated. B, Mesial canal with apical transport, ledge, and zip perforation. C, Strip perforation of the mesial root. D, Missed MB2 canal in an upper molar. E, Suspected coronal leakage of bacteria and a separated file.

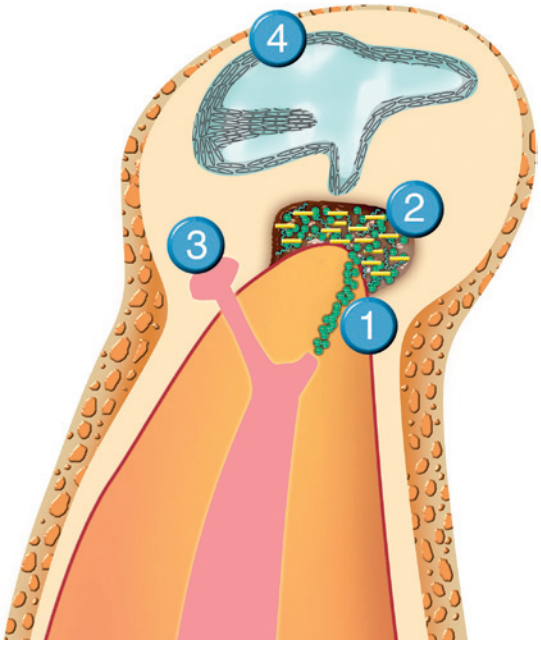

FIG. 8-3 The causes of posttreatment disease. *(1)* Intraradicular microorganisms. *(2)* Extraradicular infection. *(3)* Foreign body reaction. *(4)* True cysts. (Redrawn with permission from Sundqvist G, Figdor D. In Orstavik D, Pitt-Ford TR, editors: *Essential Endodontology,* London, 1998, Blackwell Science, p 260.)

space free of bacteria, if the obturation does not adequately entomb those that may remain, or if new microorganisms are allowed to reenter the cleaned and sealed canal space, then posttreatment disease can and usually does occur. In fact, it has been asserted that persistent or reintroduced microorganisms are the major cause of posttreatment disease.[163] Many iatrogenic treatment complications, such as creation of a ledge or separation of an instrument, result in persistence of bacteria in the canal system. It is not the complication itself, however, that results in persistent disease; rather, it is the inability to remove or entomb the microorganisms present that creates the pathologic state. While infected root canals of endodontically untreated teeth generally contain a polymicrobial, predominantly anaerobic flora,[230] cultures of infected, previously root-filled teeth produce very few or even a single species (see also Chapter 14). The infecting flora are predominantly gram positive, not anaerobic, and a commonly isolated species is *Enterococcus faecalis,*[71,182] which has been shown to be resistant to canal disinfection regimens.[16,33] Interestingly, if the previous root canal treatment is done so poorly that the canal space contains no obturating material in the apical half of the root canal space, its flora is more typical of the untreated necrotic infected pulp than that of classic "failed" root canal therapy.[231] Though posttreatment disease has been primarily blamed on bacteria in the root canal system, certain fungi, notably *Candida albicans,* are found frequently in persistent endodontic infections and may be responsible for the recalcitrant lesion.[214]

Extraradicular Infection

Occasionally bacterial cells can invade the periradicular tissues either by direct spread of infection from the root canal space via contaminated periodontal pockets that communicate with the apical area,[212] extrusion of infected dentin chips,[99] or by contamination with overextended, infected endodontic instruments.[257] Usually, the host response will destroy these organisms, but some microorganisms are able to resist the immune defenses and persist in the periradicular tissues, sometimes by producing an extracellular matrix or protective plaque.[249] It has also been shown[166,216,232] that two species of microorganisms, *Actinomyces israelii* and *Propionibacterium propionicum,* can exist in the periapical tissues and may prevent healing after root canal therapy.

Foreign Body Reaction

Occasionally, persistent endodontic disease occurs in the absence of discernible microorganisms and has been attributed to the presence of foreign material in the periradicular area. Several materials have been associated with inflammatory responses, including lentil beans[211] and cellulose fibers from paper points.[125] In the seemingly endless debate about which endodontic obturation technique is superior, there has been much discussion about the effect of overextended root canal filling materials upon apical healing. Outcomes assessments generally show that filling material extrusion (root filling flush to the radiographic apex or gross overextension) leads to a lower incidence of healing (see also Chapter 7).[63,215] Many of these cases involved not only overextension but also inadequate canal preparation and compaction of the root filling whereby persistent bacteria remaining in the canal space could leak out. Gutta-percha and sealers are usually well tolerated by the apical tissues, and if the tissues have not been inoculated with microorganisms by vigorous overinstrumentation, then healing in the presence of overextended filling materials can still occur.[63,70,137]

True Cysts

Cysts form in the periradicular tissues when nests of epithelial cells, retained from tooth development, begin to proliferate due to the chronic presence of inflammatory mediators. These epithelial cell rests of Malassez are the source of the epithelium that lines cystic walls, and cyst formation may be an attempt to help separate the inflammatory stimulus from the surrounding bone.[181] The incidence of periapical cysts has been reported to be 15% to 42% of all periapical lesions,[162,224] and determining whether a periapical radiolucency is a cyst or the more common periapical granuloma cannot be done with available radiographic methods.[23] There are two types of periapical cysts: the periapical true cyst and the periapical pocket cyst. True cysts have a contained cavity or lumen within a continuous epithelial lining and are therefore isolated from the tooth, whereas with pocket cysts, the lumen is open to the root canal of the affected tooth. True cysts, due to their self-sustaining nature, probably do not heal following nonsurgical endodontic therapy[112,165] and usually require surgical enucleation (Fig. 8-4).

When a patient presents with posttreatment disease, clinical decision making depends on determining the cause of the persistent disease and then making an assessment of how best to treat the pathologic condition. The following section presents a rationale and methods for performing endodontic diagnosis that offer the greatest likelihood of a successful outcome.

DIAGNOSIS OF POSTTREATMENT DISEASE

It has been stated that "there may be different ways of treating a disease; however, there can be but one correct diagnosis."[9]

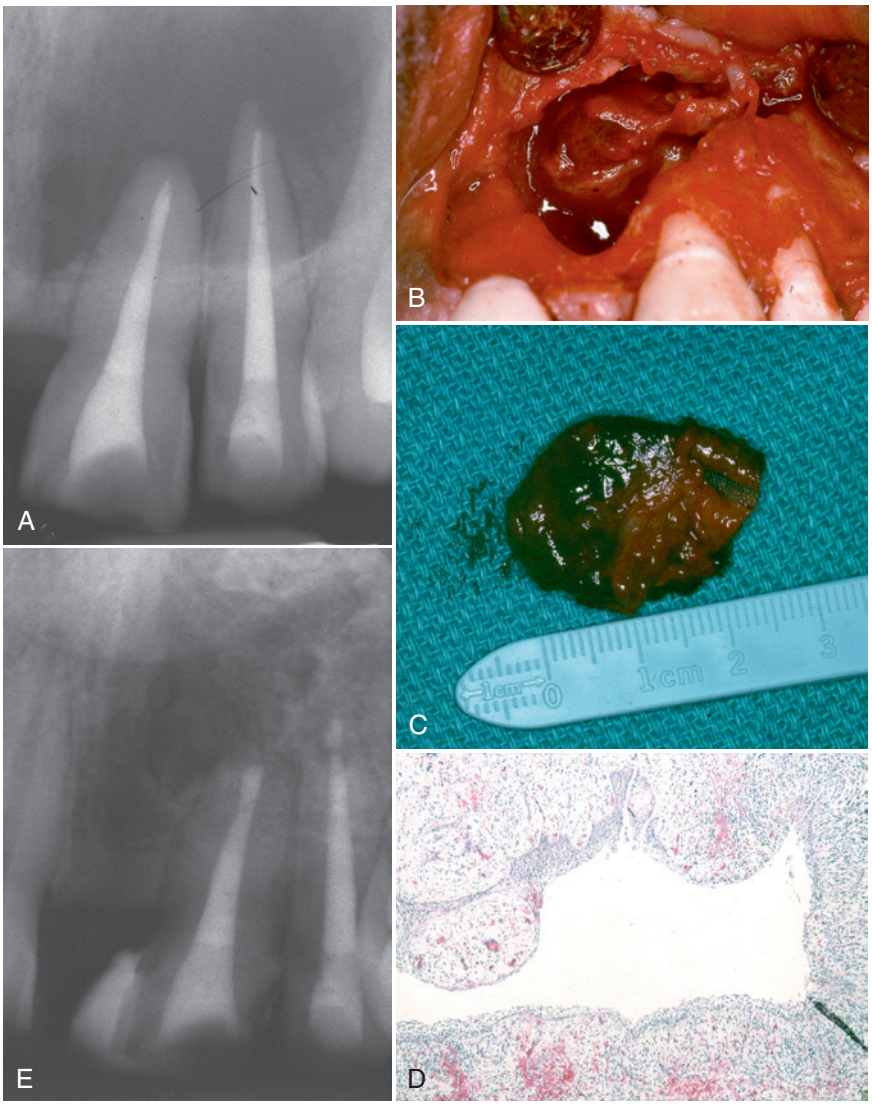

FIG. 8-4 A, Apparently good nonsurgical retreatment with large persistent lesion. B, Surgical exposure of apical lesion in situ. C, Large lesion removed in toto. D, Histopathologic section confirming cystic nature of the lesion. E, A 4-year post-operative film showing apical scar formation due to the large size of the lesion. The teeth were asymptomatic and in function.

The proper diagnosis is probably the most important portion of any endodontic procedure (see also Chapter 1). This is not as bold of a statement as one may first suspect when consideration is given to what the patient may undergo if treatment is performed based on an incorrect diagnosis (Fig. 8-5). To make a correct diagnosis, the clinician must rule out nonodontogenic etiology, perform all of the appropriate tests, properly interpret the patient's responses to these tests, derive at a definitive diagnosis, and decide on treatment options. When performing a diagnosis in endodontic cases where there is no history of previous endodontic therapy, both a pulpal and periradicular diagnoses are necessary. In cases of persistent disease, the diagnosis may not be as straightforward as the clinician may be dealing with partially treated pulp canals, missed canals, and many other types of problems associated with the previous treatment. These must be included in the diagnostic description for each case.

Endodontic diagnosis was thoroughly discussed in Chapter 1, and the reader is referred there for further details on these procedures. The diagnostic method requires collecting subjective information, developing objective findings, and using these to arrive at a diagnosis and plan of treatment.

The subjective information is collected by questioning the patient and then actively listening to the responses. Of particular interest in cases of suspected posttreatment disease is whether the patient recalls the use of aseptic techniques during the previous endodontic therapy. If a rubber dam was not used, for example, and this can be confirmed with a call to the previous clinician, nonsurgical retreatment will almost certainly be necessary because the canals can be assumed to be contaminated regardless of how aesthetically pleasing the previously filled case may appear on the radiograph. The diagnostician should be careful to avoid or to minimize communicating to the patient any negative feelings he or she has toward the

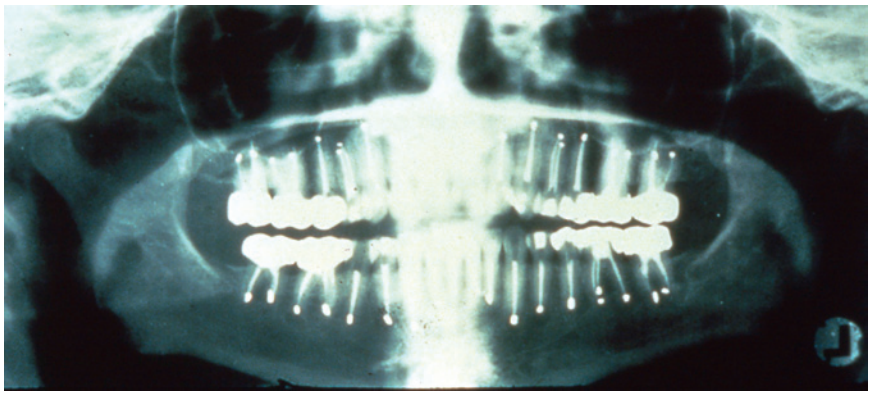

FIG. 8-5 This patient was misdiagnosed for years and underwent unnecessary endodontic therapy. The actual cause of the patient's complaint was nondental pain. (Courtesy Dr. Ramesh Kuba.)

previous treatment, however bad it may seem. This approach allows the patient to become more comfortable with the current clinician and the proposed corrective treatment. An irate patient is an irate patient, and negativity will color the patient's emotional state, level of trust, and ability to accept current or future treatment plans. If the patient asks a direct question about the previous treatment, an honest answer is necessary, but avoid the temptation to imply superiority by disparaging the former clinician. To state the situation honestly and correctly without being inflammatory, use a sentence such as "Well, it may be that your previous dentist (endodontist) had some difficulty with that tooth. Let's see if we can figure out what could have been the problem."

Following a thorough review of the patient's health history, the next step is to gather all of the objective information needed to obtain an accurate diagnosis. This information will include the clinical and radiographic examination. The clinical examination should include a visual extraoral and intraoral examination, and a thorough periodontal evaluation. Visual examination is greatly aided by magnification and illumination, which can allow the clinician to identify significant conditions invisible to the naked eye, such as fine fractures on root surfaces (Fig. 8-6). Exposed dentin from recession and narrow based probing defects may be the result of an endodontic infection draining through the sulcus; however, they sometimes indicate vertical root fracture.[40] The presence of occlusal wear facets indicates the presence of occlusal trauma that may complicate diagnosis and treatment outcome by predisposing the tooth to fracture,[90] and it has been associated with posttreatment disease.[113] Further information on diagnosis and management of cracks and fractures is provided in Chapter 21.

Radiographic assessment is obligatory. Even though radiographs may be a critical aid to the clinician, they should never be the sole support for a conclusive diagnosis. They are only one piece of the puzzle in determining endodontic etiology.[58] In cases with previous endodontic therapy, radiographs are useful in evaluation of caries, defective restorations, periodontal health, the quality of the obturation, existence of missed canals, impediments to instrumentation, periradicular pathosis, perforations, fractures,[238] resorption, and canal anatomy. Radiographs should be properly exposed and have a sharp, clear image. They should include the tooth and surrounding tissues, and multiple angulated films should be used to determine endodontic etiologies using the buccal object moves most

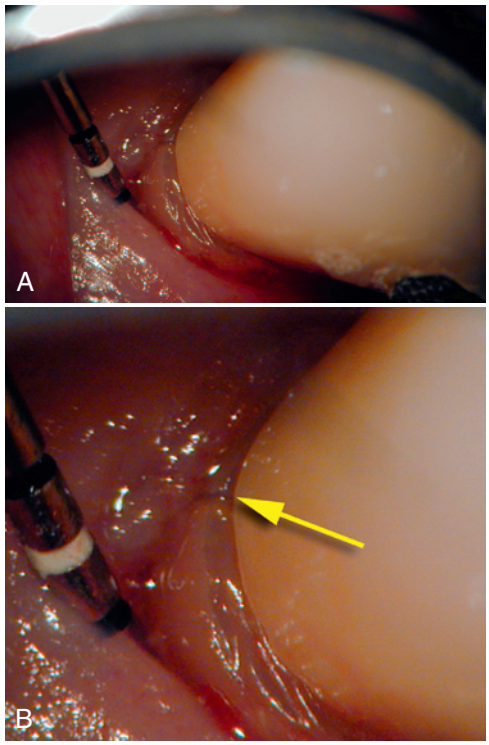

FIG. 8-6 A, Buccal aspect of a premolar with posttreatment disease. B, Higher magnification reveals a vertical fracture.

rule (Fig. 8-7).[80] Bitewing radiographs are useful for determining periodontal bone height and looking for caries or fractures. All sinus tracts should be traced with a cone of gutta-percha followed by a radiograph to localize their origin.[111]

Cone beam computed tomography (CBCT) has been introduced into endodontics, and its usefulness in the management of endodontic retreatment is unquestioned. It has provided a quantum leap in our ability to determine the causes of posttreatment apical periodontitis by giving the clinician, for the first time, the ability to easily, safely, and inexpensively visualize the tooth and surrounding structures in three dimensions (3D). CBCT use in endodontics is discussed in detail in Chapter 2, but when faced with a tooth needing retreatment, it is especially helpful. The CBCT allows the clinician to determine the true size, extent, and position of periapical and resorptive lesions

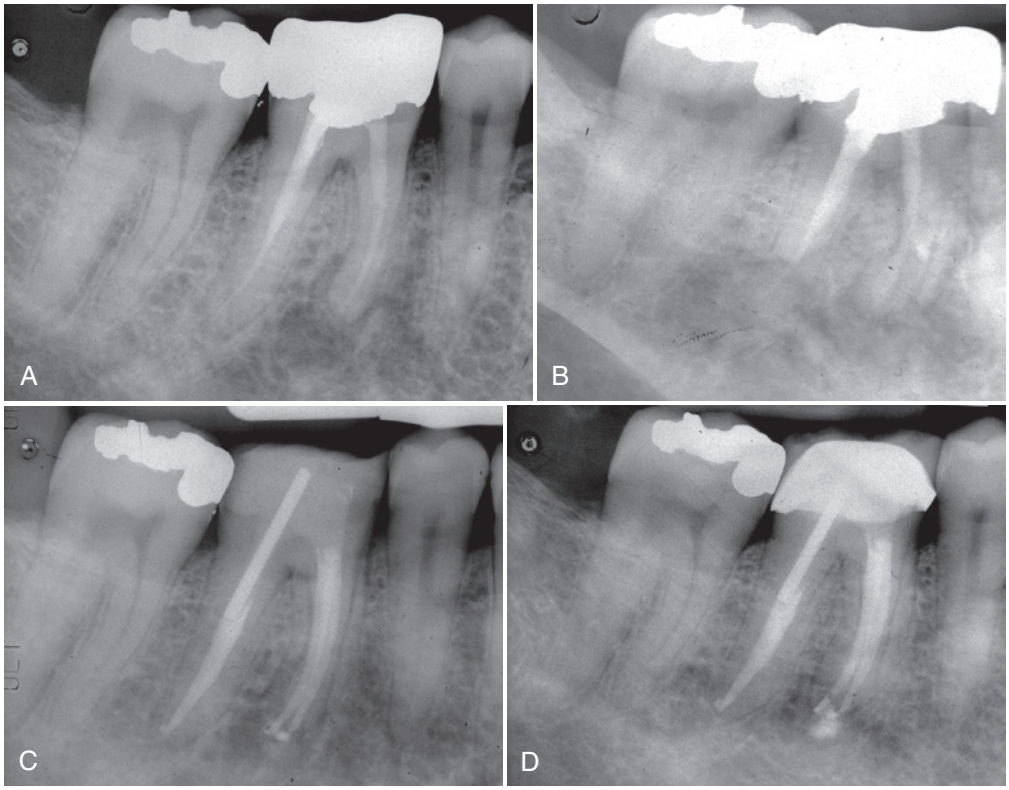

FIG. 8-7 **A**, Posttreatment disease. Previous endodontic therapy performed 3 years previously. **B**, Distal angle radiograph reveals asymmetry indicating the presence of an untreated mesiobuccal canal. **C**, Immediate postobturation film showing treated MB canal. **D**, A 14-month postoperative view. The patient was asymptomatic.

and gives added information about tooth fractures, missed canals, root canal anatomy, and the nature of the alveolar bone topography around teeth.[42] CBCT technology has greatly enhanced presurgical diagnosis and treatment planning, because the relationship of adjacent anatomic structures such as the maxillary sinus and inferior alveolar nerve to the root apices can be clearly visualized. This helps the clinician to decide on when to perform endodontic retreatment surgically or nonsurgically. CBCT is more accurate than periapical radiography in the diagnosis of apical periodontitis, and it can reveal the details of the lesions and adjacent structures, thus providing enhanced clinical diagnosis and treatment planning.[42,175,176]

There are many manufacturers and brands of CBCT machines on the market today, but the most useful ones for endodontic retreatment are those that produce the clearest image with the highest resolution.[155] These would be the small field of view (FOV) machines that image a small volume and use the smallest picture element (voxel) dimensions available. Radiation exposure to the patient with these machines is in the range of 23 to 488 μSv,[142] which is very small, but the "as low as reasonably achievable" (ALARA) principle applies, so its use in every diagnostic case cannot be encouraged. In a joint position statement in 2010, the American Association of Endodontists and the American Association of Oral and Maxillofacial Radiologists stated that "CBCT should only be used when the question for which imaging is required cannot be answered adequately by lower dose conventional dental radiography or alternate imaging modalities."[1] When managing posttreatment disease, however, almost every case will benefit from the use of three-dimensional imaging.

One major cause of posttreatment apical periodontitis is untreated canals, and the CBCT gives an unprecedented ability to discover those (Fig. 8-8). In one study, endodontist evaluators failed to identify at least 1 root canal system in approximately 4 of 10 teeth when using images obtained by conventional digital radiography compared with images from cone-beam computed tomography.[152]

Avoiding treatment, which will lead to a predictable failure, is beneficial for both the clinician and the patient. The ability to obtain 3D images of teeth will help the clinician to avoid these mishaps. The diagnosis of root fractures frequently frustrates the clinician, as a definitive diagnosis is often difficult and treating these teeth has a high likelihood of a poor outcome. Although visualizing root fractures in teeth with root fillings is still not predictable using CBCT,[45,50,96,122] the patterns of bone loss indicative of root fracture can sometimes be seen,[275] and this helps the clinician to infer their presence. The prognosis for the treatment of root resorption is directly related to the extent of the resorption, and this usually cannot be determined accurately using conventional radiography. Using small FOV CBCT, however, the extent of the lesions and the prognosis can be determined,[54] usually saving the patient from an exploratory procedure that may be doomed to fail (Fig. 8-9).

Though most clinicians believe that CBCT is not necessary for every patient treated, there are many retreatment situations where the additional information gained (relative to conventional radiography) is extremely valuable. In the future, specific protocols for use will be developed; but for now, the authors recommend that clinicians use their best judgment on when to use this new technology.

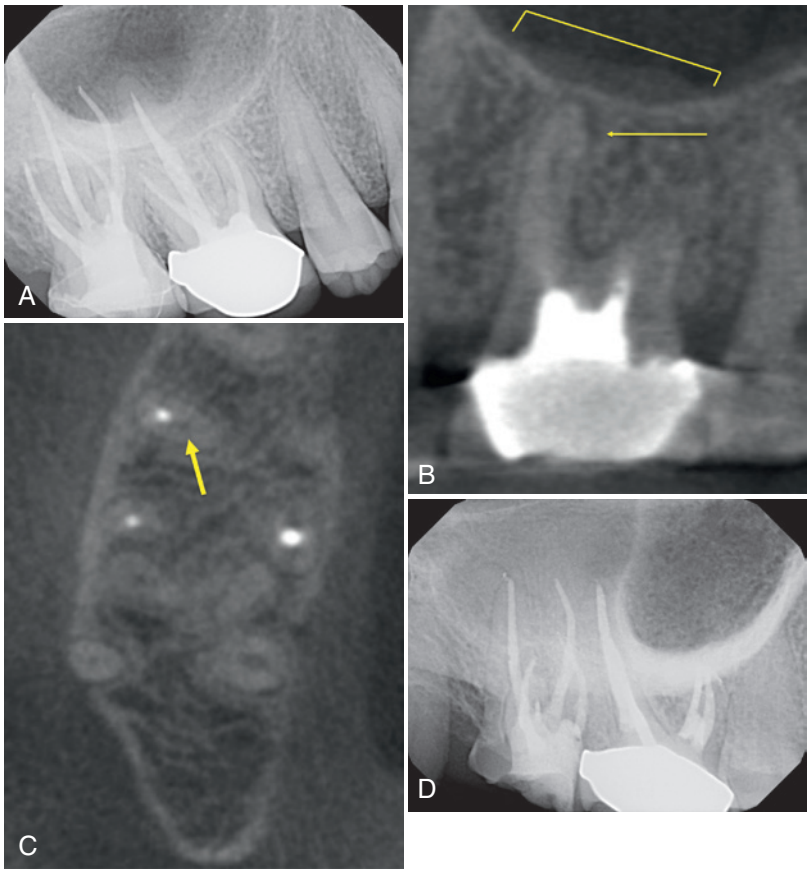

FIG. 8-8 A, Preoperative radiograph of symptomatic tooth #3. B, Sagittal slice of tooth #3 showing periodontal ligament thickening and associated sinus mucosal thickening. C, Axial slice showing untreated MB2 canal *(arrow).* D, Treated case.

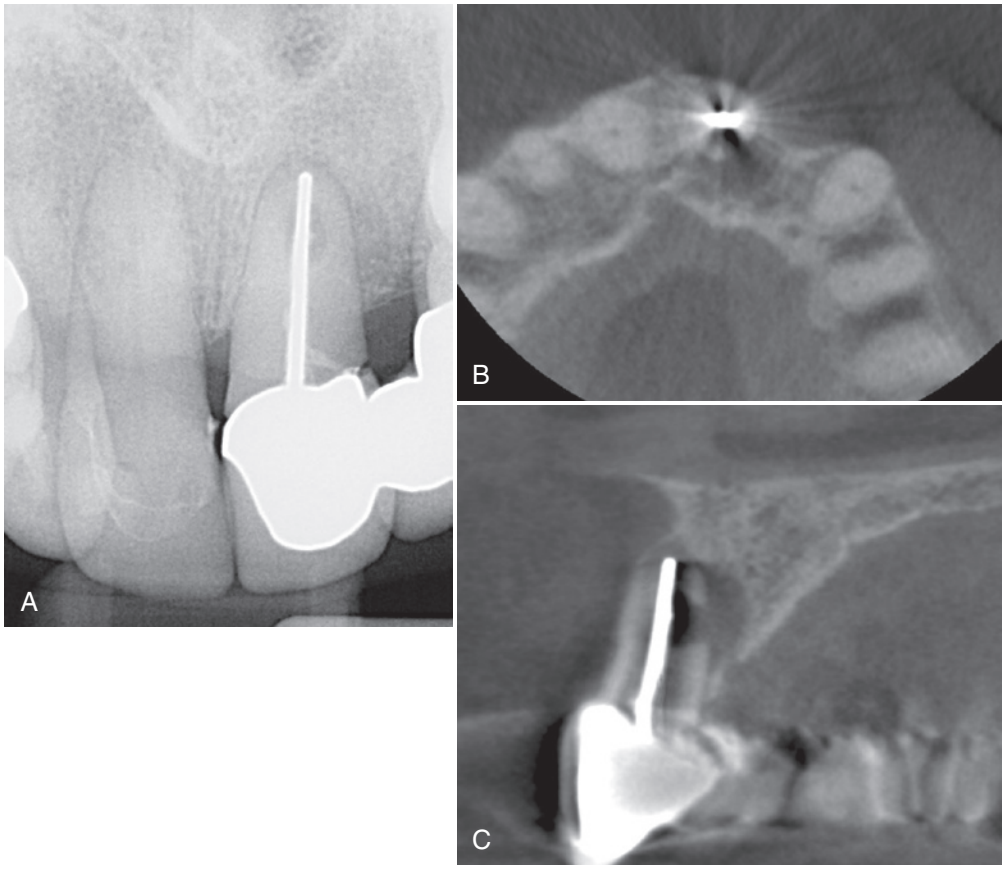

FIG. 8-9 A, Preoperative radiograph showing suspected small area of resorption associated with an endodontically treated tooth filled with a silver cone. B, Axial slice showing how beam hardening artifact from the metallic root filling obscures the image. C, Sagittal view showing very large palatally oriented external root resorption. The prognosis for retention of this tooth was poor, and the patient elected to extract it.

Comparative testing is the next procedure performed to collect objective information about the pulpal and periradicular status. Most useful are the periradicular tests that include percussion, bite, and palpation.[256] These allow the diagnostician to begin developing a sense of the status of the periradicular tissues. These tests are of great importance anytime an endodontic diagnosis is needed. However, they are of even greater importance when evaluating teeth that have been previously treated with endodontic therapy due to the lack of significant and consistent evidence that can be gained from pulp vitality tests in these cases. If a tooth exhibits percussion tenderness, it may be due to persistent endodontic disease, but recent trauma or occlusal trauma may also cause this finding,[90] as can periodontal disease.[256]

Pulp vitality tests are often of little value when examining teeth with previous endodontic therapy. However, if the patient's chief complaint reveals the need for these tests, they must be performed because it is possible that the pain may be referred from a nearby vital tooth and not from the root canal–treated tooth. When there is vital tissue remaining in the canals of a previously root-filled tooth, either by way of a completely missed canal or from an improperly cleaned canal, patients may complain of sensitivity to heat or cold.[90] Pulp vitality tests should then be performed to assess the situation. They are also useful in testing adjacent and opposing nonendodontically treated teeth to rule out those as etiologies for poorly localized pain. Once the tissue is removed from the pulp chamber after root canal therapy, the results of these tests should almost always be negative, even with radicular pulp remaining. Thus, a negative response with previously treated teeth is not necessarily conclusive; whereas a positive response usually means there is responsive pulp tissue remaining in the tooth.[90] Care is always warranted in interpreting pulp test results, however, as false positive and negative results may occur.[204] As with cold tests, the same limits apply to heat tests as far as the reasons for false results and accuracy relative to retreatment cases.

The remaining pulp vitality tests—electric pulp test, test cavity, and direct mechanical dentin stimulation—are of even lesser value than thermal testing when evaluating teeth that have already received endodontic therapy. These are usually precluded by the existing restoration or endodontic therapy.

When all diagnostic information is collected, a diagnosis must be developed. It is important to record the diagnosis in the patient's record so that anyone reading the record can discern the clinician's rationale for treatment. The pulpal diagnosis will usually be recorded as previous endodontic treatment, but the periradicular diagnosis will vary depending on the clinical picture presented. In the case of previous endodontic treatment, however, a brief note about the suspected etiology of the persistent disease is warranted.

TREATMENT PLANNING

Once the diagnosis is complete, the cause of the persistent disease will usually become apparent. At this point in the clinical process, information must be given to the patient by the clinician as to what treatment options are available and the likely outcomes of each choice. The patient is then allowed to make a decision based on his or her own perceptions of the options, not by the clinician's opinion as to what is "best" for the patient. The reader is reminded, however, that if the cause of the posttreatment condition remains unknown despite thorough diagnostic workup, then any decision results in an empirical "trial and error" type of treatment. This approach should be avoided if possible, and prior to definitive treatment, consultation with an endodontist or other colleague is in order. This consultation may be as simple as a brief conversation or even referral of the patient, but a second opinion is extremely useful in these situations. In most instances due to the interdisciplinary nature of modern dentistry, consultation with other clinicians who are treating the patient becomes a necessity to enhance the potential for successful treatment outcomes.

Occasionally, a patient will have persistent symptoms that mimic posttreatment disease, but these symptoms are actually the result of nonendodontic conditions such as occlusal trauma, concurrent periodontal disease, or nondental pain conditions. Appropriate diagnostic procedures should allow the clinician to sift through these options and treat accordingly.

The patient harboring true endodontic posttreatment disease has four basic options for treatment, which are as follows:

1. Do nothing
2. Extract the tooth
3. Nonsurgical retreatment
4. Surgical retreatment

The first option is to do nothing with the condition and allow it to take its course (Fig. 8-10). This approach is sometimes a useful, short-term option if the etiology of the

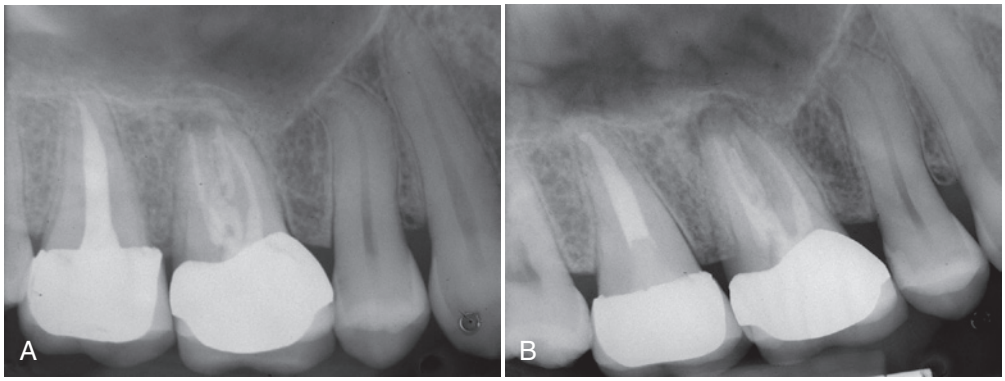

FIG. 8-10 A, Radiograph indicating presence of asymptomatic persistent apical periodontitis 7 years after initial treatment. The patient elected no treatment at that time. B, The 6-year follow-up. Lesion has enlarged, and the tooth has become symptomatic.

condition remains unknown and the clinician feels that another diagnostic sampling would help with diagnosis. Even though most clinicians would find this approach to be a less than desirable long-term course of action, the decision belongs to the patient. The clinician is bound, however, to ensure that the patient has complete information about what will happen if nothing is done. The events in the progression of the disease and a reasonable timeline are necessary, and the conversation needs to be thoroughly documented in the patient record to avoid possible subsequent accusations of abandonment. The question of whether the clinician is required to follow up with the patient or dismiss the patient from the practice is one that each clinician must make based on the clinician's experience, judgment, and knowledge of the patient.

Extraction of the tooth is usually considered a viable option. Advances in both prosthetic reconstruction techniques and dental implantology have made extraction and replacement a more desirable option in certain cases where previously "heroic" (read "expensive with an unknown prognosis") methods were needed to "save" the tooth. This alternative, however, provides results that are inferior, more expensive, and much more time consuming than preserving the natural tooth. The average titanium root-form implant restoration can take up to 6 months to finish, not counting preimplant site preparation, which can add months more. Despite published long-term success rates for dental implants,[4] postimplant disease does occur[4,85,86] (Fig. 8-11) and can leave the patient with few options. The cost of implant treatment is high and usually not covered under dental benefit plans, so the net financial impact on the patient is large. Implant aesthetics can be inferior to that of natural teeth in the aesthetic zone of the mouth, and some patients are just not candidates for implant procedures.[4] Fixed partial dentures are another replacement alternative with a long history of successful use, but negative outcomes are also possible. Most concerning to the endodontist is the likelihood that retainer fabrication procedures will result in endodontic disease of the abutment teeth,[154] which may potentially occur at a rate of up to 10%[150,253] (Fig. 8-12). Removable partial dentures are a less desirable option to the patient because they are generally less comfortable, usually require a long period of patient adaptation, and frequently result in damage to adjacent oral tissues (tooth, gingiva, and mucosa) if not meticulously cleaned. Due to these factors, patient compliance with removable dentures is relatively low, and their use is declining. Occasionally, a patient will choose to have a tooth extracted and not pursue replacement. This decision is usually disastrous for the patient, but there are a few situations where this choice is a reasonable alternative. Diseased maxillary second molars with no opposing tooth, or with an opposing tooth in class I or class III occlusion that articulates with another tooth, may be extracted without concern for future inappropriate movement of the remaining teeth, which can be so occlusally and periodontally damaging. In most instances, however, removal of a tooth will result in the need for replacement, and unless the tooth is hopelessly nonrestorable, retaining the tooth with endodontic procedures is better for the patient.

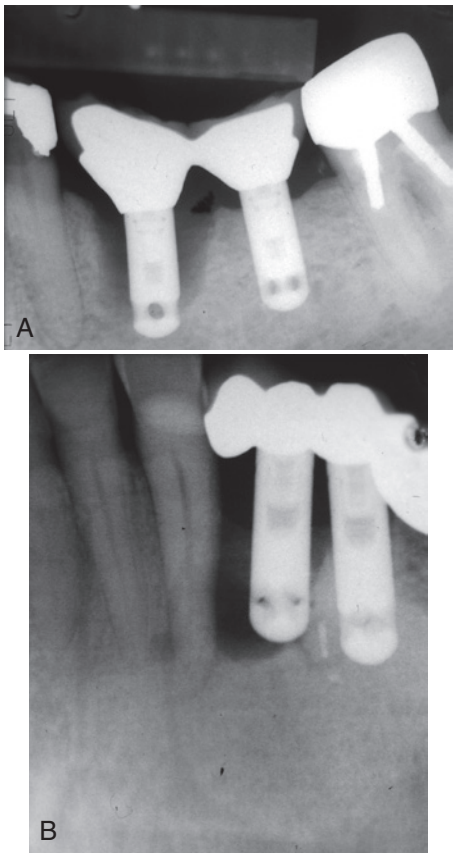

FIG. 8-11 A, Classic peri-implantitis. The implant needed removal. B, Another peri-implantitis. Note the endodontically treated root tip apical to the implant that may have contributed to the persistent disease. Perhaps apicoectomy should have been performed.

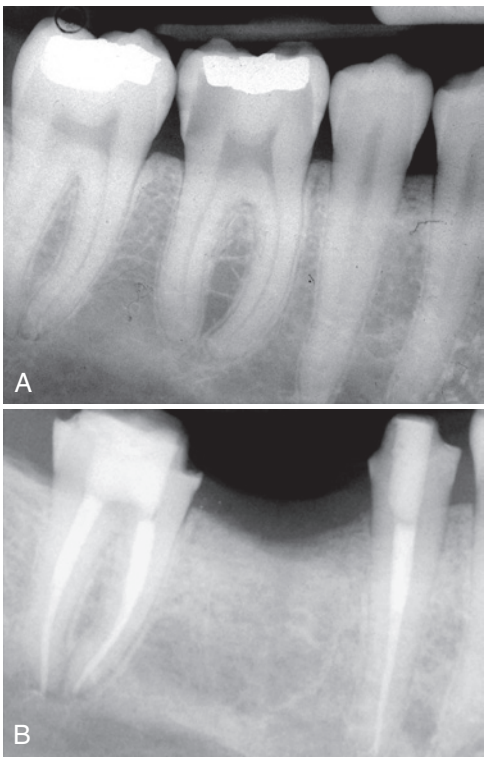

FIG. 8-12 A, Preoperative film showing deep caries approaching the pulp. The patient's holistic dentist advised extraction and replacement rather than endodontic therapy to retain the tooth. B, Fixed partial denture fabrication procedures resulted in irreversible pulpitis on both abutments requiring endodontic therapy.

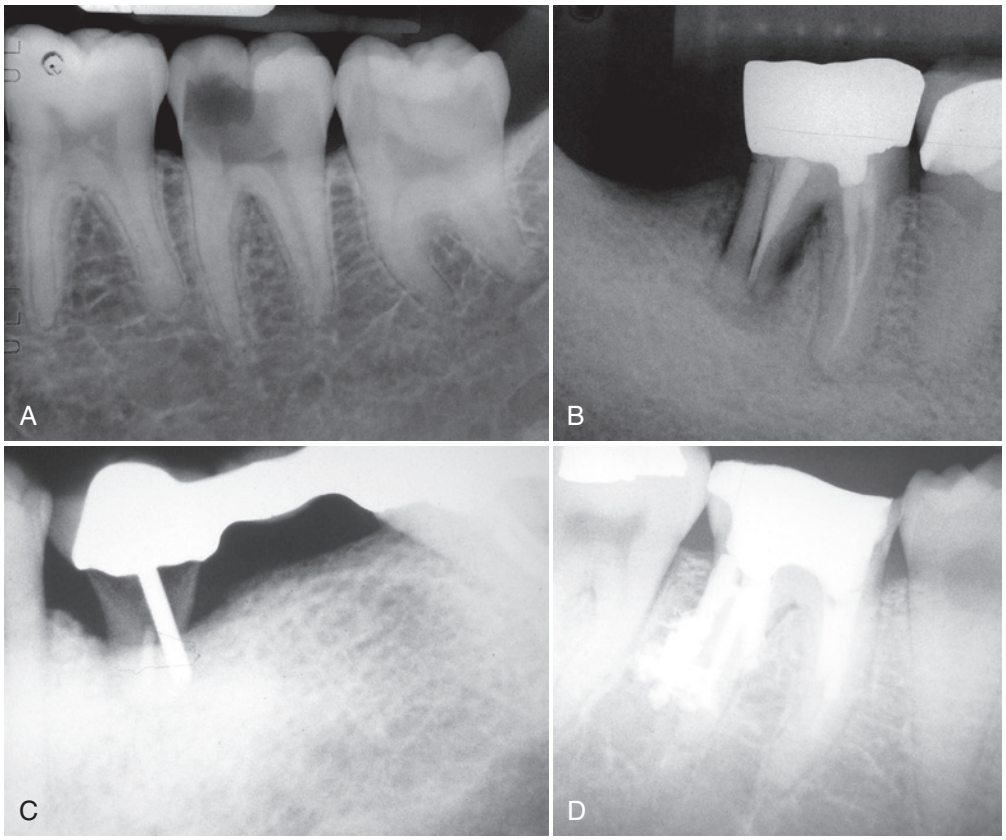

FIG. 8-13 **A,** Deep caries approaching the furcation and the biologic width. Necessary crown-lengthening surgery would open the furcation to bacterial invasion and persistent periodontal disease. **B,** Distal root vertical fracture resulting in a split root. **C,** Severe caries and post perforation. Inadequate root structure remaining to restore. **D,** Multiple distal root perforations so weaken the root as to make it nonrestorable. Note that cases A, B, and D could have resective endodontic surgery such as hemisection, but long-term prognosis is poorer than for extraction and replacement.[31,128]

Various situations may render a tooth nonrestorable (Fig. 8-13); however, the line of demarcation between restorable and nonrestorable is a movable one, depending on who is evaluating the tooth. There are several widely agreed upon situations that render a tooth nonrestorable. These include extensive caries or coronal fracture approaching or entering the furcation or the biologic width. This situation may render preprosthetic periodontal procedures ineffective (leaving a furcation involvement or poor crown-to-root ratio, for example) or, worse, removes bone that would otherwise be useful for implant procedures. Terminal periodontal disease (extensive pocketing or mobility) or root fracture[39] generally result in loss of the tooth despite all efforts at treatment. If the patient has a life-threatening endodontic infection with extensive trismus, most oral surgeons are going to extract the tooth rather than allow less aggressive management. Some previously root-filled teeth may have endured procedural complications, such as a nonretrievable separated instrument or irreparable ledge formation. In combination with the proximity to vital anatomic structures, such as the inferior alveolar canal, endodontic retreatment, either surgical or nonsurgical, may not be feasible and extraction may be the only option. These situations are, fortunately, quite rare, and in most instances, teeth presenting with posttreatment disease can be retained with endodontic procedures.

Once the decision has been made to retain the tooth, there are several choices for treatment. These can be grouped together into either nonsurgical or surgical endodontic treatments. The surgical options can be further broken down into periradicular curettage, apical root resection (with or without root filling), root amputation or hemisection, and intentional replantation (extraction/replantation).[89,170] Occasionally, a situation arises that will require both nonsurgical and surgical types of treatment to effect healing. The American Association of Endodontists has published guidelines that may help the clinician with clinical decision making.[7] However, the choice of which option to undertake will be determined by the clinician's experience, knowledge, patient considerations, and the preoperative diagnosis. If the etiology of the posttreatment disease can be made known, the choices become more obvious. In a previous section, four basic etiologies were presented. If the suspected etiology is in the first group, which is persistent or reintroduced microorganisms, then several choices are available. However, if the cause of the posttreatment disease is persistent extraradicular infection, foreign body reaction, or the presence of a true cyst, then nonsurgical root canal therapy has little likelihood of allowing healing to occur, and surgical methods should be employed.[231] The problem for the clinician is that in most instances, it cannot be determined which of these etiologies exist, so the treatment becomes more empirical.

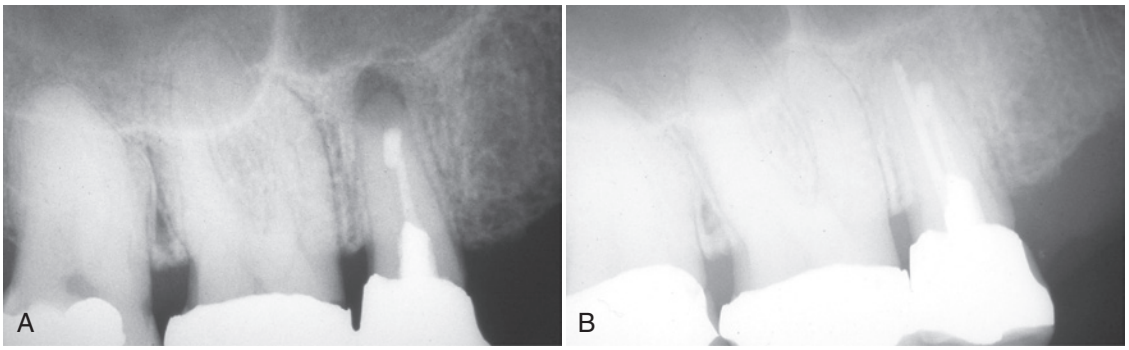

FIG. 8-14 A, Posttreatment disease following apical surgery. Off-center positioning of the root filling indicates the presence of a second, untreated canal. B, One year following nonsurgical retreatment showing complete healing.

The choice of nonsurgical retreatment versus apical surgery becomes the focus of the decision in most instances. Outcomes assessment studies provide some help in making this decision. The reported healing rates of nonsurgical retreatment range between 74% to 98%,[66,222,136,221,208] but with apical surgery alone, only 59% heal completely.[63] When apical surgery is preceded by orthograde retreatment, however, the incidence of complete healing rises to 80%.[63] In general, nonsurgical retreatment will be the preferred choice, because it seems to provide the most benefit with the lowest risk. It has the greatest likelihood of eliminating the most common cause of posttreatment disease, which is intraradicular infection. Nonsurgical retreatment is usually less invasive than surgery and has a less traumatic postoperative course. There is less likelihood of incurring damage to adjacent vital structures such as nerves, adjacent teeth, and sinus cavities. However, nonsurgical retreatment may be more costly than surgical treatment, especially if large restorations must be sacrificed during disassembly procedures prior to the retreatment. In addition, the amount of time needed for retreatment is usually longer than surgical intervention. There are times when the clinician may not be able to achieve the complete elimination of microorganisms from the canal space, and complete obturation may not be possible. Apical surgery is chosen, therefore, when nonsurgical retreatment is not possible or when the risk-to-benefit ratio of nonsurgical retreatment is outweighed by that of surgery.[64,144]

There are many factors to consider when deciding whether to retreat surgically or nonsurgically (see also Chapter 9). The patient must be fully aware of the proposed treatment and the alternatives, and he or she must be motivated to follow through with all treatment including the final restoration. The patient must have adequate time to undergo the required procedures. If he or she does not, then apical surgery alone may be indicated although the patient must be made aware of the potentially compromised nature of the treatment. Clinicians must be armed with the best equipment and knowledge available, and critical self-evaluation should allow experienced clinicians to know what they can treat and what they cannot. The tooth must be restorable and retreatable. Attempting nonsurgical retreatment on teeth where there is little likelihood of improving the previous treatment provides little benefit to the patient. Thus, in disease situations where there is an apparently adequate root filling and no evidence of coronal leakage, surgery may be indicated. If the previous treatment falls below any acceptable standard and there is no evidence of apical periodontitis, then there is no indication for any treatment unless a new coronal restoration is planned. In that case, conservative retreatment is indicated, and the reported success rates are very high.[63,66] If there has been a previous procedural complication, such as a ledge that cannot be bypassed or a separated instrument that cannot be removed, then surgery may become a better option. Most times, however, it is still prudent to attempt the retreatment because ledges or separated instruments that appear impenetrable on diagnostic films can frequently be bypassed. Even if they cannot, nonsurgical retreatment can enhance the success of subsequent apical surgery as noted previously. The clinician must be careful not to worsen the situation by overly vigorous attempts to treat the previous complication, as root perforation, worsening of a ledge, or another separated instrument may be the result. Previous failed apical surgery should be retreated nonsurgically and then followed up because many surgical failures are due to poorly cleaned and filled canal systems[187] (Fig. 8-14). In many instances, performing the surgery a second time can be avoided altogether. If there is evidence of root fracture (narrow-based probing defect or a "J"-shaped radiolucency encompassing the root apex and progressing in a coronal direction[237] (Fig. 8-15), then nonsurgical retreatment would be unlikely to improve that situation. Apical exploratory surgery may be necessary, which could result in root resection or even extraction of the tooth.

Each case should be approached as a unique set of considerations that need to be reviewed and interpreted prior to selecting a treatment method. Once the selected option is undertaken, however, the prudent clinician is always watchful, as additional pieces of information can be discovered during treatment that may modify previous decisions.

NONSURGICAL ENDODONTIC RETREATMENT

The primary difference between nonsurgical management of primary endodontic disease and that of posttreatment disease is the need to regain access to the apical area of the root canal space in the previously treated tooth. After that, all of the principles of endodontic therapy apply to the completion of the retreatment case. Coronal access needs to be completed, all previous root-filling materials need to be removed, canal obstructions must be managed, and impediments to achieving full working length must be overcome. Only then can cleaning

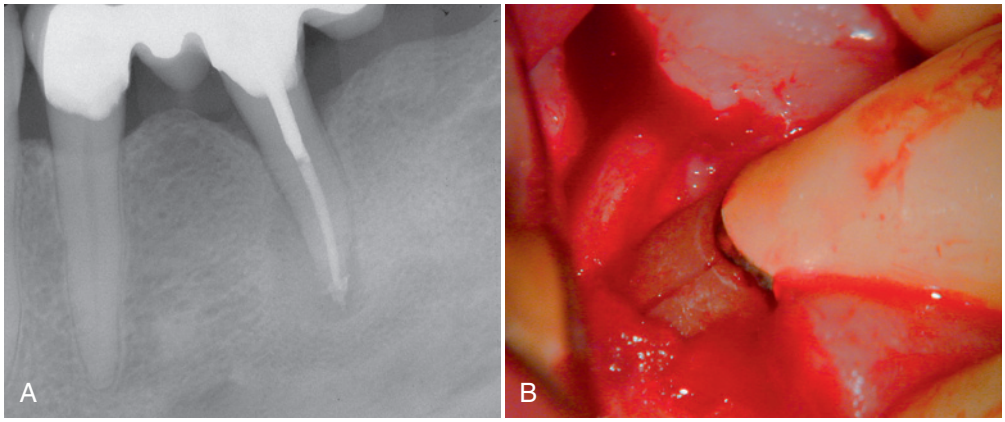

FIG. 8-15 A, "J"-shaped radiolucency possibly indicating root fracture. B, Exploratory surgery confirms presence of vertical root fracture.

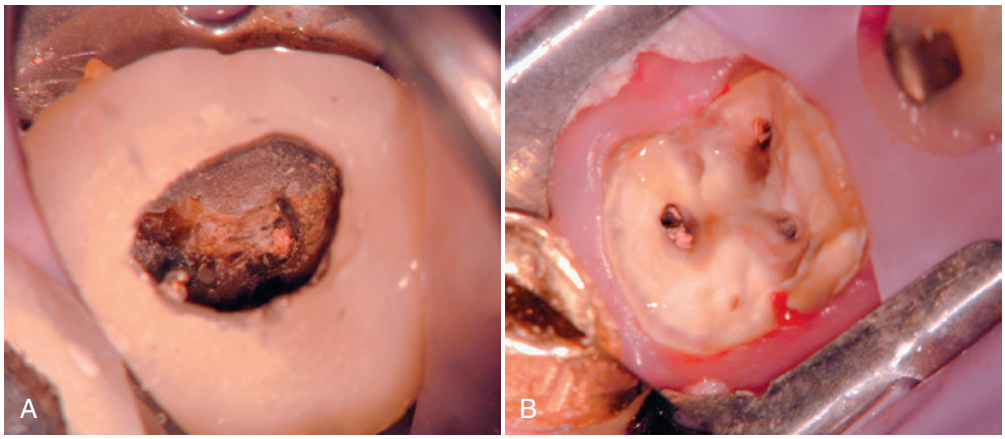

FIG. 8-16 A, Limited visibility and access with crown present. B, Enhanced visibility and access with crown off. Note that isolation was achieved by using a Silker-Glickman clamp and sealing putty.

and shaping procedures be instituted that will allow for effective obturation and case completion. The remainder of this chapter is devoted to these topics in the order that they generally present themselves to the clinician treating the previously root-filled tooth.

Coronal Access Cavity Preparation

Retreatment access has been called coronal disassembly[187,188] because of the frequent need to take apart or remove the previous coronal and radicular restoration. Following initial endodontic therapy, most teeth require and receive a full coverage restoration, and many times that restoration is supported by a post and core. Coronal-radicular access for retreatment is much more complicated in these cases when compared with endodontically treated teeth that have been minimally restored. The goal of the access preparation is to establish straight-line access to the root canal system while conserving as much tooth structure as possible. The ideal access preparation allows for instruments to enter the canals without being deflected by the access cavity walls. This is reasonably easy to achieve when the tooth is completely intact and a pulp chamber is present, because surface and internal anatomic landmarks can guide the search for the canals. Unfortunately, when endodontic retreatment is necessary, the tooth structure has almost always been altered and is commonly quite misrepresentative of the original anatomy of the tooth.

When presented with a tooth in need of retreatment that has a full coverage restoration, the decision for the clinician becomes whether to attempt to preserve the restoration or to plan its replacement. This decision is made simpler if there is a defect or caries associated with the restoration or if the treatment plan calls for a new crown. The old one is simply removed and replaced later in the treatment sequence (Fig. 8-16). When the crown is considered to be satisfactory, the decision becomes more complex. If the restoration is maintained, the cost for replacement can be avoided, isolation is easier, the occlusion is preserved, and the aesthetics will be minimally changed. Even if the crown requires replacement, the clinician may elect to retain it during the endodontic retreatment to allow for better isolation with the rubber dam. Unfortunately, retreatment may be more difficult with the crown in place, as this could lead to an increased chance for an iatrogenic mishap due to restricted visibility. In addition, removal of canal obstructions, such as posts, will be more difficult, and there is an increased chance the clinician may miss something important such as hidden recurrent caries, a fracture, or an additional canal. To preserve the restoration, two approaches can be taken: access through the crown or crown removal and replacement when retreatment

is completed. The simplest choice is to prepare an access cavity through the existing crown, although there is a significant risk of damaging the restoration resulting in the need to replace it.[160] This risk must be communicated to the patient prior to instituting therapy. If the clinician decides to access through the existing restoration, there are several choices of access burs to use, depending on what material the preparation will be cut through. If the access will be primarily cut through metal (amalgam alloy or cast metal) or composite resin, carbide fissure burs such as the #1556 are usually chosen. With many restorations, it is advisable to consider using a combination of burs to achieve access. For example, when a porcelain fused to metal crown is encountered, a round diamond is used to cut through the porcelain layer. Once the metal substructure is encountered an end-cutting bur, such as the Transmetal bur (Dentsply Maillefer, Ballaigues, Switzerland) or the Great White bur (SS White, Piscataway, NJ) can be used to cut through to and remove the core material efficiently. An important consideration for the clinician is the potential for porcelain fracture, which may occur during the preparation or possibly at a later date after completion of the treatment. This damage is especially common with porcelain jacket crowns. Restorations fabricated completely of porcelain are becoming more and more popular, thus creating added concern due to the increased likelihood of crack formation during access. Porcelain is a glass, and drilling through this material will create many microfractures, which in turn, may weaken the structure of the restoration, making it more prone to future failure.[94] Copious coolant water spray and the use of diamond burs are recommended during access through porcelain to minimize occurrence of this event.[235] In a novel approach, Sabourin and colleagues[189] recently showed that, compared with the use of drills, air abrasion produced almost no defects in the porcelain structure of all ceramic crowns when used for endodontic access. It was, however, significantly more time consuming to access through the crown this way.

If the decision is made to remove the crown for reuse, the visibility is increased, allowing for much easier removal of canal obstructions and a decrease in the potential for operator error; however, rubber dam clamp placement and tooth isolation may become a bigger problem. Also, despite all of the varying techniques and armamentaria available for removal of an existing restoration, the procedure remains unpredictable and many times can also result in damage to the restoration or the inability to remove it at all.

The clinician must decide how to remove the crown. If the crown is of no value, even as a temporary, then the clinician can take the easiest road and simply cut it off. However, if the crown is to be preserved, then a more conservative approach must be used. Two considerations, which may influence the decision about removal of a crown or bridge, are what material the restoration is made of and what is it cemented with. Conservative removal efforts are difficult with traditional, all-metal restorations cemented with nonbonded cements. This situation has been even more of a concern lately due to the increasing popularity of tooth-colored restorations, mainly different types of porcelain or porcelain fused to metal (PFM) restorations, which are being bonded to the tooth. These restorations are less likely to withstand the stresses of removal than those fabricated entirely of metal, and restorations that are bonded are much more difficult to remove due to the adhesive strengths of bonding agents. Each new generation of bonding agent is stronger than the previous, making removal increasingly more difficult as cosmetic dentistry advances.

Many devices have been developed specifically for the conservative removal of crowns. Some of the more commonly used devices are forceps that have been designed specifically for crown removal such as the K.Y. Pliers (GC America, Alsip, IL) (Fig. 8-17), which use small replaceable rubber tips and emery powder to enable a firm grasp of the crown without damaging it. Other instruments of this type include the Wynman Crown Gripper (Integra Miltex, York, PA), the Trial Crown Remover (Hu-Friedy, Chicago, IL), and the Trident Crown Placer/Remover (CK Dental, Orange, CA). Unfortunately, a crown that has been cemented with long-term cement or has been bonded to the tooth will usually not be removed with one of these instruments. There are also forceps designed specifically to engage the margins of the crown while using an adjacent tooth as a fulcrum. Squeezing the handles together will cause the crown to be elevated off of the tooth. The Roydent Bridge Remover (Roydent, Rochester Hills, MI) works in this fashion and can be effective in crown removal, but care must be taken to avoid damage to fine, fragile margins, especially on porcelain crowns. Another type of instrument can be engaged under the margin, and a subsequent impact delivered at this site will dislodge the restoration. The Easy Pneumatic Crown and Bridge Remover (Dent Corp, White Plains, NY) and the Coronaflex (KaVo, Lake Zurich, IL) create this impact from compressed air, whereas the Morrell Remover (Henry Schein, Melville, NY) applies the force manually using a sliding weighted handle. The ATD Automatic Crown & Bridge Remover (J. Morita, Irvine, CA) uses vibrations to break the crown-to-preparation bond, and the Crown-A-Matic (Peerless International, N. Easton MA) delivers a shock impulse to loosen the crown. As mentioned earlier, crown margin damage may result, as can inadvertent extraction of the tooth if the periodontium is compromised[183] (Fig. 8-17, *E*). A different approach to conservative crown removal involves drilling a small hole through the crown to allow a device to thread a screw through the hole. This approach creates a lifting force that separates the crown and the tooth. The instruments that work in this manner are the Metalift (Classic Practice Resources, Baton Rouge, LA), the Kline Crown Remover (Brassler, Savannah, GA), and the Higa Bridge Remover (Higa Manufacturing, West Vancouver, BC, Canada). Although very effective on metal crowns, these instruments may cause damage to porcelain occlusal surfaces on PFM restorations, and their use in both anterior teeth and in all porcelain restorations is generally precluded.

Another interesting technique designed to remove a crown without causing damage is performed using the Richwil Crown & Bridge Remover (Almore, Portland OR). This material is a water-soluble resin, which is softened using warm water (Fig. 8-18). The small block of material is placed on the crown to be removed, and the patient bites into this material until the resin cools and hardens at which point the patient opens his or her mouth, generating enough force to pull the crown off. The clinician must be careful to avoid using this technique when the opposing tooth is extensively restored, because the opposing restoration may inadvertently be removed during the procedure. None of these techniques works in every case, and they may produce damage to the restoration being removed or possibly others. These are, however, methods that are available and may work while permitting reuse of the restoration.

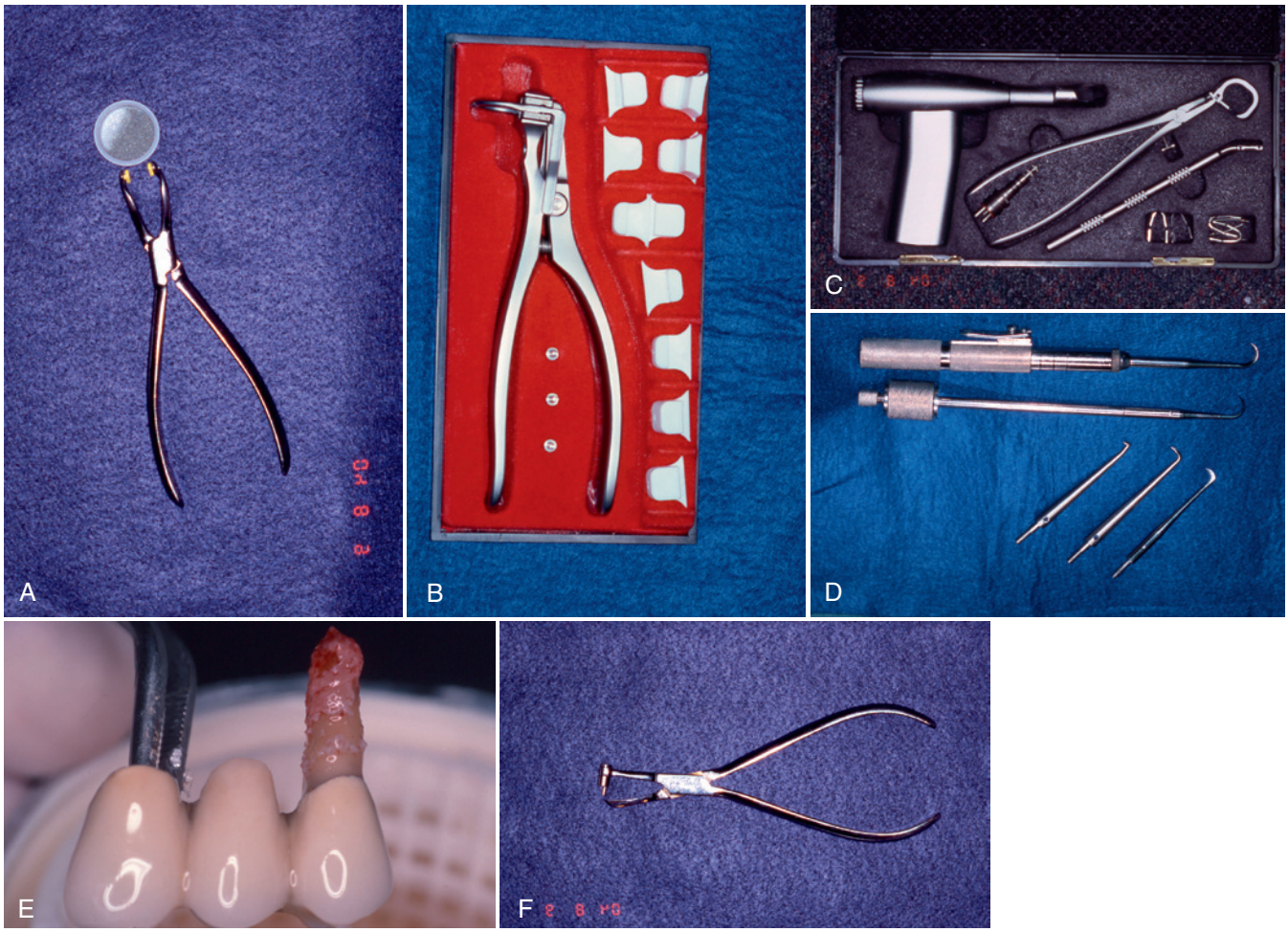

FIG. 8-17 **A,** KY Pliers (GC America) and supplied emery powder. **B,** Roydent Bridge Remover (Roydent). **C,** CoronaFlex Kit (KaVo). **D,** *(top)* Crown-A-Matic (Peerless International); *(bottom)* Morrell Crown Remover (Henry Schein) with interchangeable tips. **E,** Tooth inadvertently extracted using a crown/bridge remover. Endodontic therapy was performed in hand and the tooth was replanted, a procedure known as unintentional replantation. **F,** Kline Crown Remover (Brasseler USA).

Post Removal

Once the access is prepared, it is common to encounter a post, as posts are frequently used in the restoration of endodontically treated teeth. The clinician may encounter many different types of posts during retreatment (Fig. 8-19). They can be classified into two categories: prefabricated posts and custom cast posts. Historically, cast posts were more commonly used than prefabricated posts; however, since the 1990s, cast posts have become much less popular.[201] The main reason for this decrease is the convenience of placing the prefabricated post immediately after post preparation as opposed to waiting for a laboratory to fabricate the casting. There is also less likelihood of the interappointment contamination that frequently occurs with temporary post/core/crowns that are needed for cast/custom post and core fabrication. Prefabricated posts come in a variety of shapes, designs, and materials. The shapes can be subclassified into two groups: parallel sided or tapered. The design of posts also can be subclassified into active (threaded), passive, vented, fluted, and acid-etched groups. There are also many materials that have been used to fabricate posts, such as stainless steel, gold, titanium, ceramic, zirconium, and fiber-reinforced composite posts. Cast posts, which are fabricated in a laboratory, will always be made up of precious or nonprecious metal alloys. These posts also come in a variety shapes and configurations because they are custom manufactured for each root in which they are placed. Most of these have some degree of taper, and many will be cast in one piece with the core included.

In addition to the shape, design, and material of posts, there are two more very important factors that will have some influence on the clinician's ability to remove them. These factors are the adhesive material used to cement the post and the location in the arch of the tooth that requires post removal.

The same concerns regarding cements that were discussed in the section on crown removal apply to post removal. The main consideration is whether the post was cemented with traditional cement or bonded with a composite resin and dentin-bonding agent. Several post systems on the market today, such as the ProPost (Dentsply, York, PA), use acid-etched metal posts that are bonded into the canal with cements, such as Panavia (Kuraray America, New York, NY) or C&B Metabond (Parkell, Edgewood, NY). Removal of these posts is

FIG. 8-18 A, Richwil Crown and Bridge Remover (Almore). B, Using hot water to soften the material. C, The remover is placed on the restoration to be removed and the patient bites into the material. D, Image showing the removed crown adhering to the material.

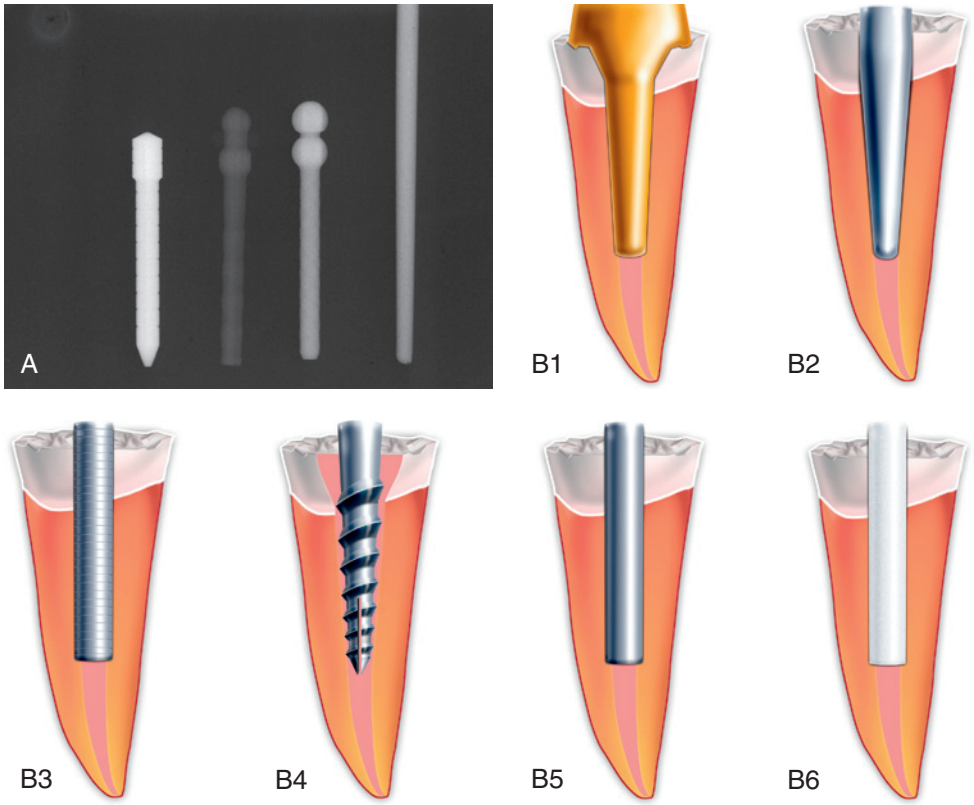

FIG. 8-19 A, Relative radiopacities of post materials: *left* to *right*—stainless steel, fiber post, titanium post, gutta-percha. B, Diagrammatic representation of post types: *(B1)* custom cast, *(B2)* tapered, *(B3)* parallel, *(B4)* active, *(B5)* passive/metal, and *(B6)* passive/nonmetal. (Diagrams courtesy DENTSPLY Endodontics.)

extremely difficult and occasionally impossible, regardless of which technique is used.[84] One study has shown that heat generation with ultrasonic vibration may help to decrease retention of resin cemented posts,[73] but concern for heat-generated periodontal ligament damage may preclude this technique.[201]

With regard to location, the more posterior in the arch, the more difficult the post is to remove. This predicament is a result of accessibility. The more accessible the tooth is, the easier the post is to remove because the clinician will have more techniques and instruments available to use.[2] Also, the more anterior the tooth is, the less the opposing occlusion will interfere with post removal.

Post Removal Techniques

After initial access and after the post to be removed has been located, the clinician is faced with the decision of how to remove it. Many techniques have been developed for the sole purpose of post removal. Regardless of which technique is chosen, there is one simple yet extremely important rule to follow: it is not only what is removed but what is left behind that is important. This rule applies to the removal of all intracanal obstructions. The reason for this rule is to make sure that the remaining tooth, after removal of the obstruction, can be restored predictably with a good long-term prognosis. For example, there is little use in successfully removing a post and leaving behind a root that is eggshell thin and prone to fracture (Fig. 8-20).

The first step in post removal is to expose it properly by removing all adjacent restorative materials. With preformed posts, the bulk of the core material around the post and within the chamber can be removed with a high-speed handpiece using cylindrical or tapered carbide or diamond burs. When the majority of the restorative material is removed, a less aggressive instrument, such as a tapered bur in a slow-speed handpiece or a tapered, midsized ultrasonic tip, should be used to remove the last of the embedding core material. This process is greatly facilitated by use of magnification and illumination. Once there is minimal restorative material remaining, a smaller-sized ultrasonic instrument should be used to minimize the risk of removing unnecessary tooth structure or thinning of the post. The more post that is left, the more options for removal, and the more tooth structure that is left, the more options for restoration. At this point, a high-speed bur is too risky to use. When the core is cast in one piece with the post, a high-speed instrument can perform this process to generate a shape that can facilitate removal.

Once the post is well isolated and freed from all restorative materials, the clinician can begin the retrieval process. There are many instruments and kits on the market that can be used to remove posts; however, prior to using one, the retention of the post should be reduced. The clinician can usually continue to use the same medium-sized ultrasonic tip that was used to get to this point. Using this instrument at the interface between the post and the tooth (the cement line) and constantly moving it around the circumference of the post will disrupt the cement structure along the post/canal wall interface and decrease post retention facilitating removal,[19,32,119] although the effects of ultrasonic vibration may be minimal in reducing retention of well-fitted, long, large-diameter titanium posts.[20] Titanium has a lower modulus of elasticity than stainless steel, so it may dampen the ultrasonic vibrations, which may decrease the effectiveness of the ultrasonic; however, one study failed to duplicate this effect.[98] Nonetheless, care should be taken not to push the ultrasonic tip against the post with too much force, as this will dampen the ultrasonic wave and actually reduce the effectiveness of this technique. Taking away a small amount of the dentin around the coronal aspect of the post is not critical at this time, as this will aid in the reduction of post retention without unduly weakening the root. If the root is thin, however, and the amount of space between the cement line and the root surface is restricted, the size of the tip that can be used may be limited. Unfortunately, the smaller tips are not only less effective for post removal, but they are also more prone to breakage. At this point, the ultrasonic handpiece should be used with copious air-water spray as a coolant. Due to the heat that can be generated from this procedure, the tip should be removed from the access every 10 to 15 seconds to allow the use of an air/water syringe not only to clean the area of debris but also to reduce the temperature produced that could potentially cause damage to the periradicular tissues.[201,274] If a rubber dam is in place, the area around the post may be flooded with a solvent such as chloroform prior to activating the ultrasonic instrument, as this will help dissolve the cement around the post. Using a solvent in conjunction with removal of cemented obstructions may prove beneficial because the ultrasonic energy produced will set up shock waves in the solvent and make it penetrate

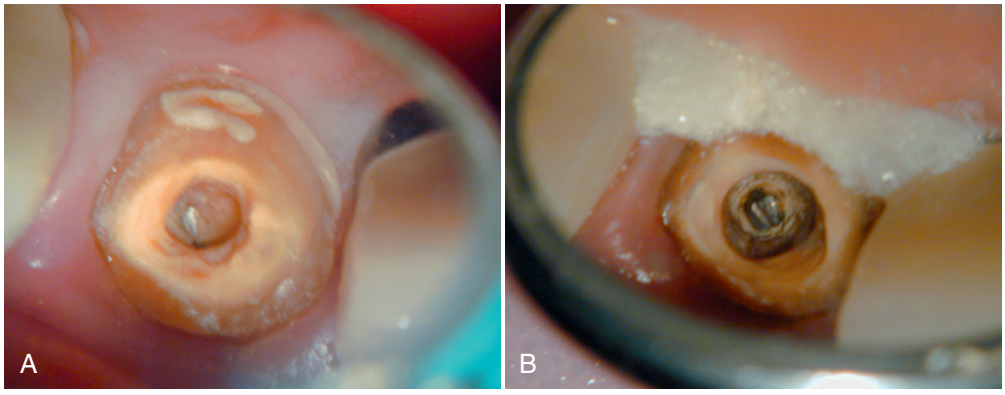

FIG. 8-20 A, Broken post (incisal view before excavation). B, Root has been so thinned and weakened by excavation procedures that restorability is questionable.

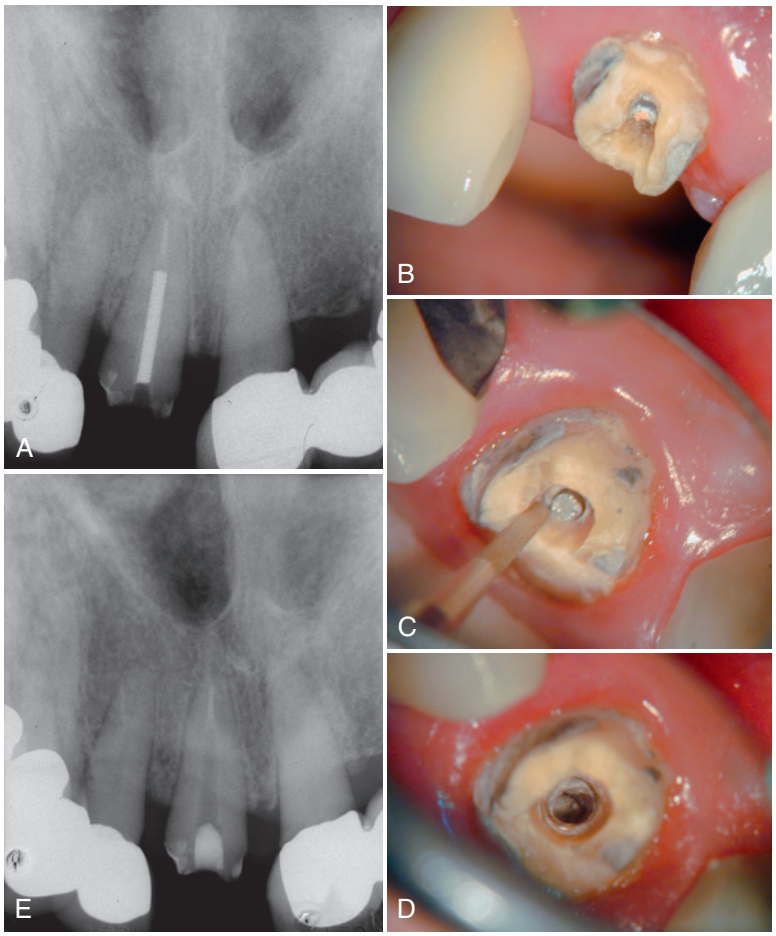

FIG. 8-21 A, Radiograph of fractured post. B, Fractured post, labial view. C, Ultrasonic troughing. D, Post removed by ultrasonic alone. E, Check film confirming complete post removal.

deeper into the canal space, exerting a faster solvent action on the cement.[78]

Using an ultrasonic instrument in this fashion is not simply helpful in reducing post retention; this may also prove to be all that is needed to remove the post. Many times, after judicious use of the ultrasonic instrument, the post will loosen and actually spin out of the preparation, completing post removal (Fig. 8-21). In addition, if post removal cannot be accomplished in this manner, the resulting post exposure will be very beneficial in contributing to the predictable use of other techniques, as many of the instruments to be discussed involve using a trephine bur to shape the coronal end of the post. Ultrasonic exposure will facilitate this process. Another instrument to consider for exposing and loosening a post is the Roto-Pro bur (Ellman International, Hicksville, NY) (Fig. 8-22). There are three shapes available, all of which are six-sided, noncutting tapered burs that are used in a high-speed handpiece around the circumference of the post. The vibrations created when the noncutting flutes come in contact with the post decrease the retention of the post, facilitating its removal.

If retention reduction does not remove the post, some form of vice is needed to pull the post from its preparation. Many post removal kits are available on the market today with varying degrees of effectiveness. One such device is the Gonon Post Removing System (Thomas Extracteur De Pivots, FFDM-Pneumat, Bourge, France), which is a very effective instrument for removing parallel or tapered, nonactive preformed posts.[145,191] This kit utilizes a hollow trephine bur that is aligned with the long axis of the post and placed over its newly exposed end. The trephine then cuts in an apical direction, shaving off the post's outer layer not only to remove tooth structure adjacent to the post but also to reduce the circumference of the post to a specific size and shape. This procedure is necessary to allow a specific, matched-size extraction mandrel to create or tap a thread onto the exposed milled portion of the post. Once the extraction mandrel with its associated washer/bumpers (Fig. 8-23) is attached to the post, the extraction forceps or vice is applied to the tooth and post. Turning the screw on the handle of the vice applies a coronal force in a similar fashion as a corkscrew removes a cork from a bottle of wine. This method is effective because all the force is applied to the bond between the tooth and the post, ideally in the long axis of the root. The main problem with this technique is the size of the vice that can make access in the molar region and between crowded lower incisors difficult. Also, if the extraction force applied is not directed in the long axis of the root, root fracture may occur.[35]

The Thomas Screw Post Removal Kit (Thomas Extracteur De Pivots, FFDM-Pneumat) (Fig. 8-24) is an instrument designed specifically for the removal of active or screw posts. The trephine burs are identical to those used with the Gonon Post Removal System, although the extraction mandrels are threaded in the opposite direction. The mandrels are reverse threaded to enable them to tap onto the screw post in a counterclockwise direction so that continued torquing force while creating the thread will unscrew the post.

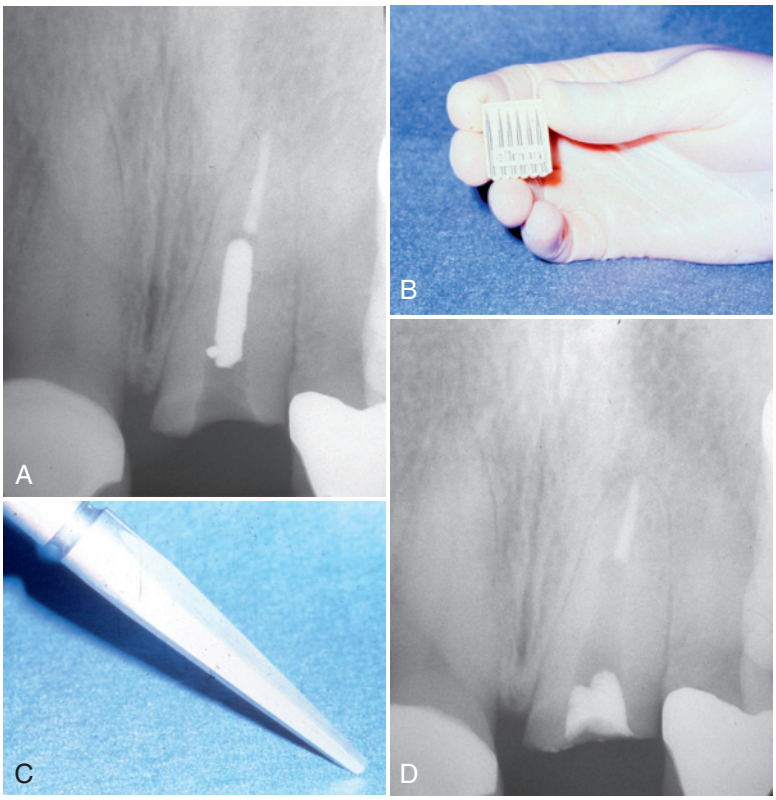

FIG. 8-22 A, Radiograph of fractured post. B, Roto-Pro Kit. C, Roto-Pro Bur. D, Post removed by vibration of the instrument alone.

The Ruddle Post Removal System (Sybron Dental Specialties)[187] (Fig. 8-25) and the Universal Post Remover (Thomas Extracteur De Pivots, FFDM-Pneumat) were designed to combine the properties of both the Gonon and Thomas kits. Both of these very similar kits are useful not only for removing parallel or tapered passive types of posts but also for removing screw posts. They can even be adapted to remove large separated instruments in the coronal straight portion of a large canal. These kits also use a trephine bur to machine the post to a specific size that will dictate which mandrel to use. These mandrels tap in the counterclockwise direction so that the same taps can be used for both passive and active posts. Once the mandrel is tapped onto the post, the extraction jaws, or vice, can be applied and activated, enabling removal of passive posts, or the tap is continuously rotated counterclockwise to unthread screw-type posts.

Another device that works in a similar fashion as the Gonon and the Ruddle Post Removal System is the JS Post Extractor (Roydent Dental Products). The biggest advantage of this kit is the size, as this is the smallest of the kits that work using a pulling action, which may help in cases where access is difficult. However, this kit does have one disadvantage: it does not have as large of a variety of trephine burs and extraction mandrels as some of the others. Therefore, the size of the post may be a limiting factor.

Another post removal device is the Post Puller, also known as the Eggler Post Remover (Automaton-Vertriebs-Gesellschaft, Germany)[228] (Fig. 8-26). This device works in a similar manner as some of the others; however, there are no trephine burs or extraction mandrels. The design of this instrument enables it to be used more efficiently with the crown removed. In addition, the design also allows this instrument to be used for cases in which the post and core are cast as one unit. This device consists of two sets of jaws that work independently of one another. With this device, both the post and the tooth are reduced to allow attachment of the post puller. Because there are no trephine burs, this reduction is done with a high-speed handpiece and bur. Next, the first set of jaws are attached to the post while the second set of jaws push away from the tooth in line with the long axis of the tooth removing the post from the canal.[228] Care must be taken to align the pulling forces of this instrument with the long axis of the root to prevent fracture,[35] and also, this technique is not recommended for the removal of screw posts. In a survey of the Australian and New Zealand Academy of Endodontists, this was the most commonly used technique for post removal.[34] However, in a survey of the American Association of Endodontists, this was one of the least used techniques.[229] Clearly, techniques that are common in one country are not always that common in another.

The increased popularity of cosmetic dentistry has created an impetus toward the use of tooth-colored posts that are fabricated from ceramic, zirconium, or various types of fiber-reinforced composite. Unfortunately, as with all posts, cosmetic posts also will need to be removed periodically. Neither the use of the Gonon Kit nor ultrasonic instruments allows for removal of fiber posts, whereas the use of a high-speed bur to channel down through the post may result in a high rate of root perforation.[178,201] The use of the Largo Bur (Dentsply)[75] and the Peeso drill[178] to remove these posts has been advocated, and most of the post manufacturers have removal burs in the kit.[47] These manufacturers' removal kits have been shown to be more efficient at removing their own fiber posts than the use of diamond burs and ultrasonics.[138] In addition, a new bur, the

FIG. 8-23 Gonon post removal technique. **A,** Fractured post in a lower incisor. **B,** Tooth isolated with a rubber dam. **C,** Gonon Kit. **D,** Ultrasonic exposure of the post. **E,** Domer bur creating a shape that the trephine bur can engage. **F,** Trephine bur milling the post. **G,** Extraction device tapping a thread onto the post. Note the three bumpers needed to protect the tooth from the vice. **H,** Vice applied. Turning the screw on the vice opens the jaws, creating the extraction force. **I,** Post removed.

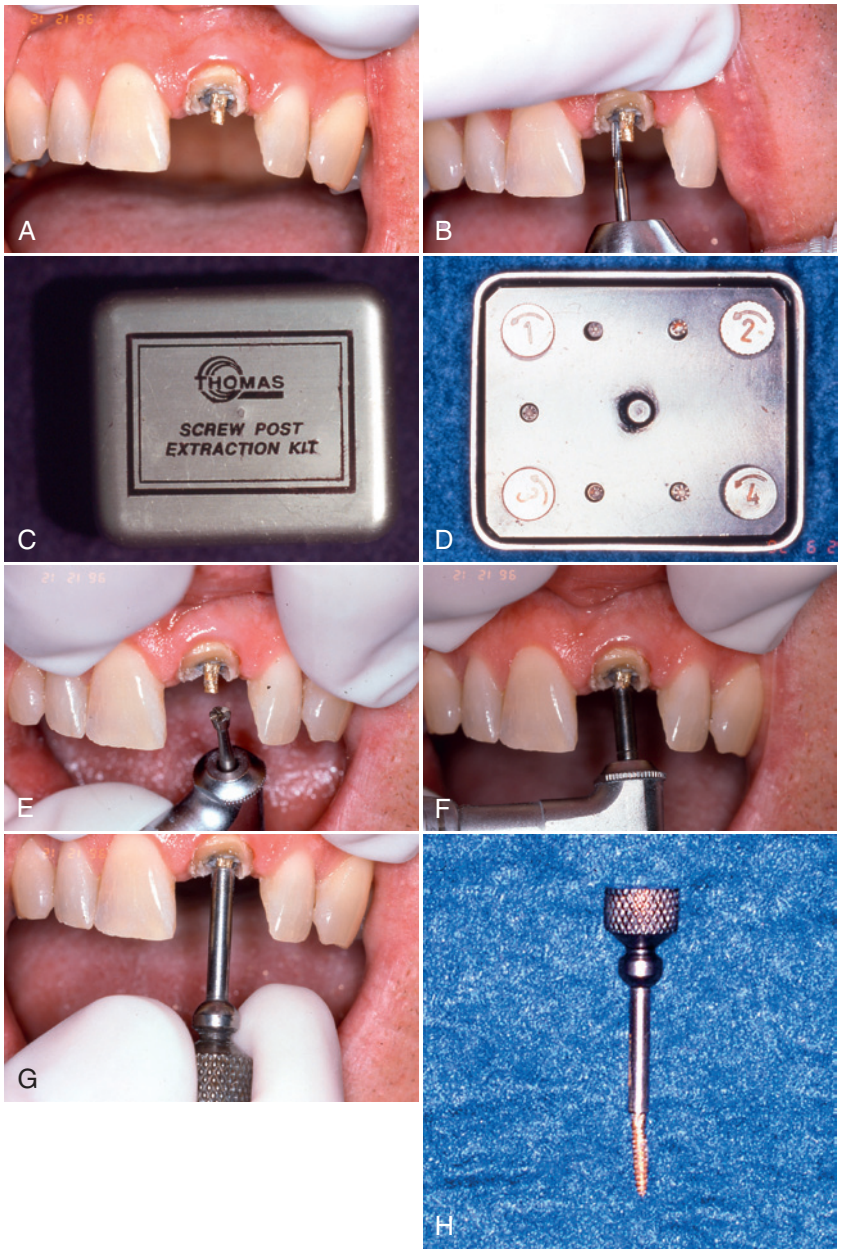

FIG. 8-24 Thomas screw post removal technique. **A,** Broken screw post. **B,** Head of post being contoured to a roughly cylindrical shape. **C** and **D,** Thomas Post Removal Kit. **E,** Domer bur creating a shape that the trephine bur can engage. **F,** Trephine bur milling the post. **G,** Application of counterclockwise rotational force using the wrench. **H,** Post removed.

GyroTip (MTI Precision Products, Lakewood, NJ), has been designed for the specific purpose of removing fiber-reinforced composite posts (Fig. 8-27). These drills consist of a heat-generating tip designed to soften the matrix that binds the fibers within the fiber-reinforced post. The fibers within the post are parallel, which assists the axial travel of the drill through the center of the post. The fluted zone of the drill allows the fibers to be safely removed, creating access to the root canal filling. Above the fluted zone, a layer of plasma-bonded silica carbide reduces the heat generation that would otherwise occur if a smooth carbide surface were rotating in contact with enamel or dentin. This abrasive zone also provides for a straight-line access preparation and facilitates the placement of a new post. Ceramic and zirconium posts are usually impossible to retrieve. They are more fragile than metal posts, and though ceramic posts may be removed by grinding them away with a bur (a procedure with a high risk of root perforation), zirconium has a hardness approaching that of diamond and cannot be removed by this method.[201]

Regardless of the post type or retrieval method used, once the post has been removed, the final step in exposing the underlying root filling material is to ensure that none of the post cement remains in the apical extent of the post space. This step can be easily accomplished by visualizing the cement using magnification and illumination and then using a straight ultrasonic tip to expose the underlying canal filling.

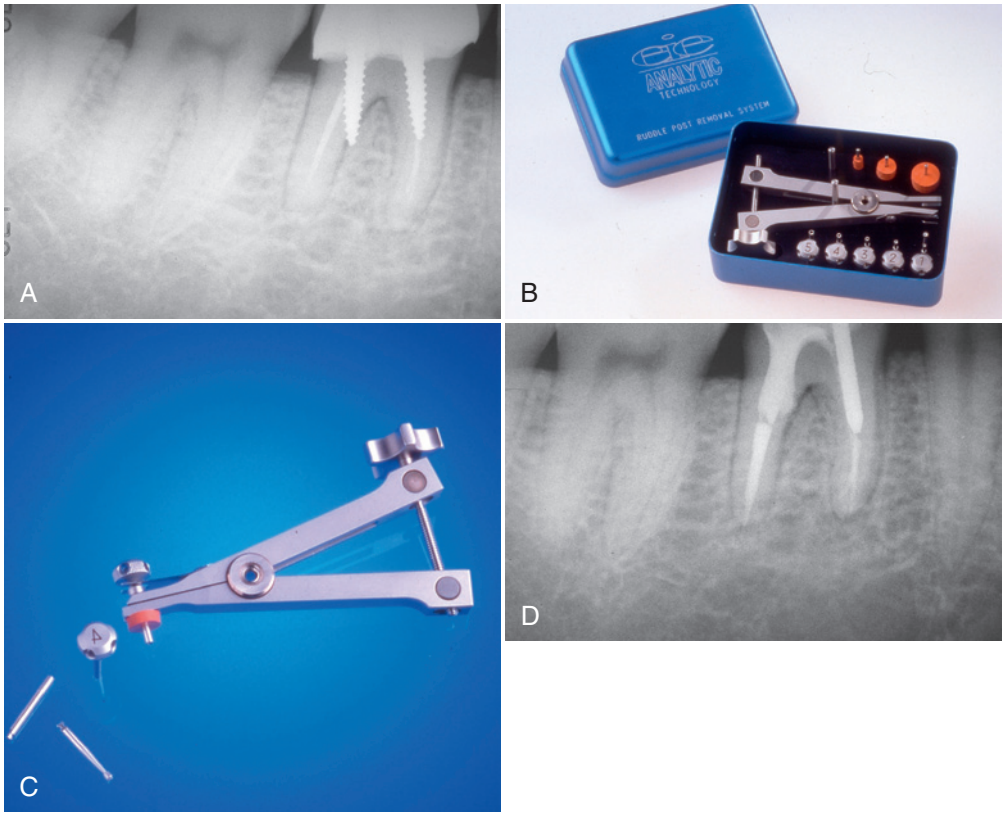

FIG. 8-25 A, Perforated post requiring removal. B and C, Ruddle postremoval kit. D, Post removed and perforation repaired. (B and C, Courtesy Kerr, Orange, CA.)

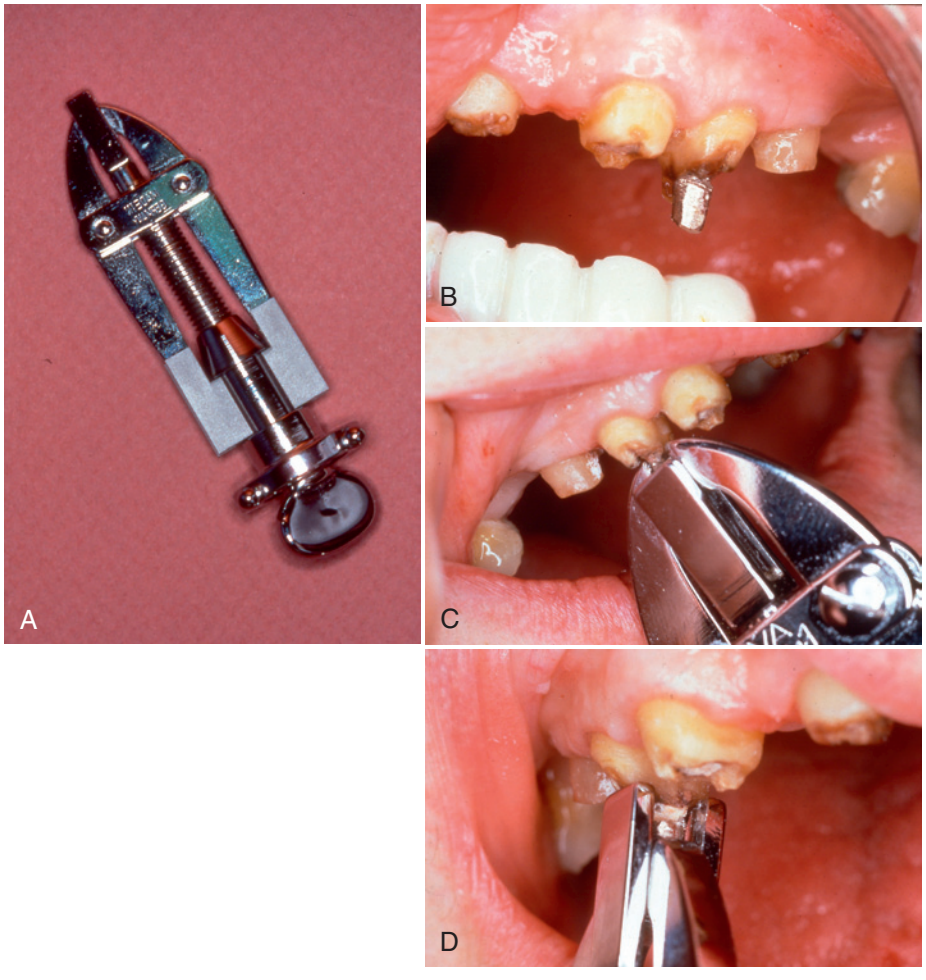

FIG. 8-26 A, Eggler Post Remover. B, Post has been contoured with a high-speed bur. C, Eggler Post Remover grasping the post. D, Elevating the post. (Reprinted with permission form Stamos DE, Gutmann JL: Revisiting the post puller, *J Endod* 17:467, 1991.)

FIG. 8-27 GyroTip technique **A**, Broken fiber post in an extracted tooth. **B**, Radiograph of test tooth with post in place. **C**, Creating a pilot hole. **D**, GyroTip instrument. **E**, GyroTip cutting through the fiber post. Note alignment with long axis of post. **F** and **G**, Post removed. **H**, Clinical case showing fiber post perforation into furcation area. **I**, Post removed with the GyroTip. **J**, The 1-year follow-up of MTA repair.

Potential Complications of Post Removal

As with many dental procedures, post removal has risks. These risks include fracture of the tooth, leaving the tooth nonrestorable, root perforation, post breakage, and inability to remove the post.[229] An additional concern is ultrasonically generated heat damage to the periodontium.[201]

Even though there may still be some who feel posts strengthen teeth, it is widely accepted that they do not.[201] Actually, it has been shown that post preparation alone weakens teeth.[250] Therefore, it seems obvious that any additional work, which may require removal of further tooth structure, will further weaken the tooth, increasing the likelihood of fracture. An in vitro study showed that cracks can form in radicular dentin during post removal using both the Gonon Kit and ultrasonics, but there was no significant difference between these two groups, and teeth with posts that were not removed.[6] The authors speculated that the potential for vertical root fracture might increase; however, the clinical significance of this

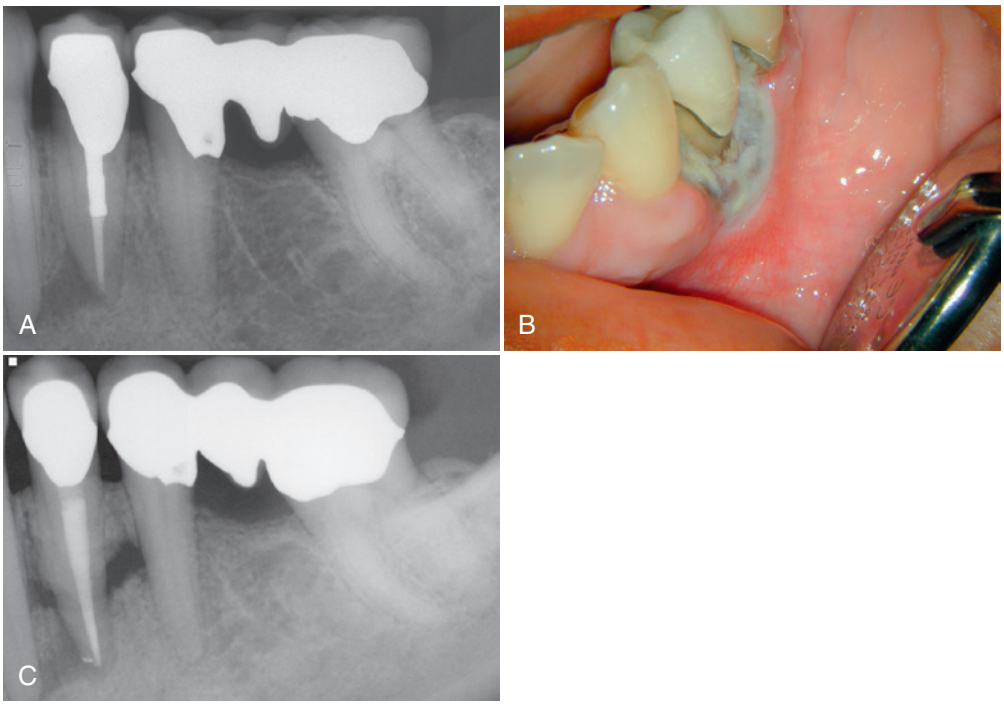

FIG. 8-28 Tissue damage from heat generated by ultrasonic application to a post during removal. The ultrasonic tip was applied to the post for no more than 5 minutes at high power with the assistant applying a constant water spray. **A**, Preoperative radiograph. **B**, and **C**, These images were taken 1 month after the retreatment. Note sloughing bone visible on Fig. 8-28, *C*, The tooth was lost 1 month later. (Reprinted with permission form Schwartz RS, Robbins JW: Post placement and restoration of endodontically treated teeth: a literature review, *J Endod* 30:289, 2004.)

remains unknown. A more recent study, however, concluded that the incidence of root fracture during post removal was extremely low and that with good case selection, post removal is, in fact, a predictable procedure.[2] If, however, post removal would also leave the remaining tooth structure in a state that may not be predictably restored with a good prognosis and if this situation can be predicted ahead of time, surgery may be the preferred treatment option.

Perforation is an additional possible complication that can happen during post removal, especially if the post is removed by simply attempting to drill it out with high-speed burs.[178] If perforation occurs, the clinician should repair it immediately, as the prognosis will worsen as the time between perforation and repair lengthens.[24,206] Once a perforation occurs, the clinician must reconsider the prognosis and determine whether the tooth should be salvaged. Terminating the procedure and pursuing a different treatment option could be considered at this point. Extraction and replacement with an implant or a fixed prosthesis was a treatment option prior to initiating the retreatment, and some may consider this treatment the best option once a perforation has occurred. However, with the development of mineral trioxide aggregate (Pro-Root MTA, Dentsply, York, PA), perforations can be repaired with a favorable prognosis.[184] The techniques and materials for perforation repair will be discussed in detail in a later section of this chapter.

Another complication is separation of the post, causing removal of the coronal segment leaving a small portion of the post with even less accessibility. This separation will decrease the likelihood of removal and occurs more frequently when attempting to retrieve titanium posts.[201]

The use of ultrasonic energy for prolonged periods of time can generate excessive amounts of heat. The heat generated can cause damage to the surrounding periodontium.[78,201] This damage may be as serious as both tooth and permanent bone loss (Fig. 8-28). For this reason, stopping periodically to cool off the area with a water spray is necessary. This will be discussed in detail in a later section.

If the clinician is unable to remove the post, he or she will be faced with a decision of what to do. This decision is based on whether the post is being removed for restorative purposes or due to the persistence of disease. If the reason is for restorative purposes and the clinician can adequately restore the tooth with the existing post or post segment, then he or she should do so. If the tooth cannot be properly restored without removal of the post and placement of a new post, then extraction and replacement with an implant or fixed prosthesis will be needed. If the reason for post removal is the persistence of disease, the tooth should be treated surgically and restored as well as possible.

Regaining Access to the Apical Area

Once the coronal-radicular access is made and all posts and obstructing restorations have been removed, then the clinician must regain access to the apical area by removing the previous root-filling materials (Fig. 8-29). This part of nonsurgical retreatment is complicated by the large variety of types of root fillings used. Today, the majority of root fillings are performed using gutta-percha in various forms; however, many other materials have been and are still being used. Silver points were popular until the 1970s and various types of pastes are,

unfortunately, still in use. The authors have seen cases of definitive root filling with phenol-soaked paper points and sometimes no root filling at all. New materials, such as Resilon (Resilon Research LLC), a soft polyester material that is bonded into the canal space, are coming on the market all the time. Though all root-filling materials have their advocates and their critics, the only certainty is that all will have some incidence of persistent disease and will need retreatment.

During the diagnostic phase, it is important to ascertain the nature of the root filling to minimize surprises when attempting retreatment. Sometimes this is readily apparent, but, in other instances, this determination may require contacting the previous clinician to discover what type of root filling was used. Occasionally, this information cannot be determined until canal entry, so extreme caution should be used when performing access so as not to possibly remove parts of the root filling that may be useful in its removal, such as the core material in solid core obturators.

Gutta-Percha Removal

One of the great advantages of using gutta-percha for root filling is its relative ease of removal. When the canal contains gutta-percha and sealer or a chloropercha filling, it is relatively easy to remove this material using a combination of heat, solvents, and mechanical instrumentation.[69,187] Upon access, it is usually relatively easy to find the treated canal orifices with the visible pink gutta-percha material inside. Initial probing with an endodontic explorer into the material can help rule out the possibility that there is a solid core carrier. If there is a plastic carrier, then heat should not be used to remove the coronal gutta-percha (more on this later). If there is no carrier, heat is applied using an endodontic heat carrier that has been heated to a cherry red glow in a torch. Unfortunately, the carrier begins to cool upon removal from the flame, so many endodontists are now using a heat source, such as the Touch 'n Heat (SybronEndo, Orange, CA) (Fig. 8-30, *A*), to provide constant, consistent heat application to soften the gutta-percha in the coronal portion of the canal.[141] Care must be exercised, however, not to overheat the root, which can cause damage to the periodontal ligament.[132,193,194] Thus, the heat should be applied in a short burst to allow the instrument to penetrate the gutta-percha mass, followed by cooling, which will cause the material to adhere to the heat carrier facilitating its removal (Fig. 8-30, *B*). After removing as much gutta-percha as possible with the heated instrument, then remove any remaining coronal material with small Gates-Glidden drills, taking care not to over enlarge the cervical portion of the canal. However, because the previously treated tooth may have had an underprepared cervical third of the canals, these drills can also be used to flare the coronal aspect in an anticurvature direction to facilitate enhanced straight line access to the apical one third of the canal and to create a reservoir for potential solvent use.[149] Again, probe the canal, this time using a #10 or #15 K-file. It is sometimes possible to remove or bypass the existing cones of gutta-percha if the canal has been poorly obturated, thus eliminating the need for solvents.[223] If that is not possible, then a gutta-percha solvent must be used to remove the remaining material in the apical portion of the canal.

Several solvents have been recommended to dissolve and remove gutta-percha for retreatment (Fig. 8-31) including chloroform,[153] methylchloroform,[259] eucalyptol,[273] halothane,[105,127] rectified turpentine,[120] and xylene.[93] All of the solvents have some level of toxicity,[14,38] so their use should be avoided if possible; however, a solvent is usually needed to

FIG. 8-29 Accumulation of materials removed from retreated teeth in a 3-month period.

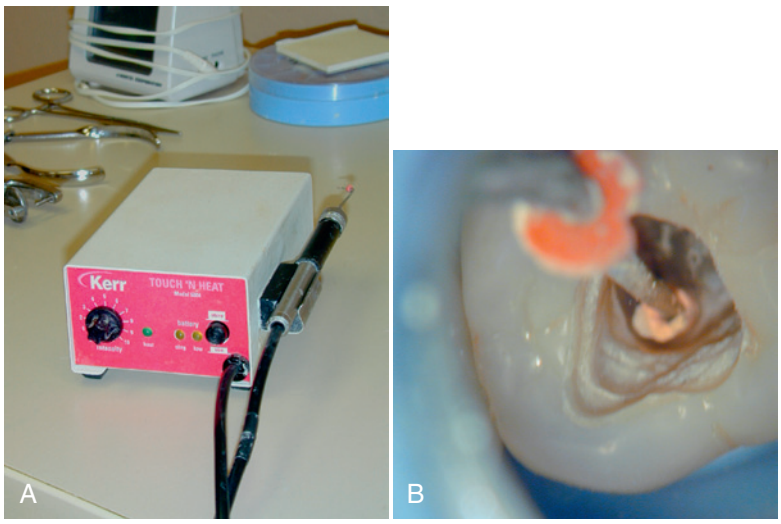

FIG. 8-30 A, Touch 'n Heat instrument. B, Gutta-percha adhering to the Touch 'n Heat tip as it cools.

FIG. 8-31 A, Chloroform. B, Eucalyptol. C, Halothane. D, Rectified turpentine. E, Xylenes.

remove well-condensed gutta-percha. The most popular solvent is chloroform because it dissolves the gutta-percha rapidly and has a long history of clinical use. In 1976, the U.S. Food and Drug Administration (FDA) banned the use of chloroform in drugs and cosmetics due to a report of suspected carcinogenicity.[251] There was no associated ban on its use in dentistry[153]; however, the report did result in the search for alternatives, some of which are listed earlier. When used carefully, chloroform is regarded as a safe and effective endodontic solvent.[38,153] All of the others generally have been reported to be less effective or have some other drawback that limits their use. Xylene and eucalyptol dissolve gutta-percha slowly and only approach the effectiveness of chloroform when heated.[269] Rectified turpentine has a higher level of toxicity than chloroform,[14] and it produces a very pungent odor in the operatory. Halothane has been shown to be as effective a solvent as chloroform in several studies,[105,127] but a more recent study indicated that the time for removal of the root filling was longer than when using chloroform.[263] The increased cost and volatility of halothane and the potential for idiosyncratic hepatic necrosis make it less desirable to use as a gutta-percha solvent.[38] Although methylchloroform is less toxic than chloroform, it is also less effective as a solvent for gutta-percha.[259] Both halothane and chloroform have been shown to affect the chemical composition of dentin[51,121] and may affect bonding strengths of adhesive cements to the altered dentin.[52] The clinical significance of these effects remains unknown, however. The evidence for the carcinogenicity of chloroform in humans is suspect,[153] but with careful use, its toxicity may be eliminated as a risk factor to both the patient[38] and the personnel in the operatory.[5] As such, its continued use as a gutta-percha solvent is recommended.

Using an irrigating syringe, the selected solvent is introduced into the coronal portions of the canals, which will then act as a reservoir for the solvent. Then, small hand files (sizes #15 and 20) are used to penetrate the remaining root filling and increase the surface area of the gutta-percha to enhance its dissolution. This procedure can be facilitated by using precurved, rigid files such as the C+ file (Dentsply Maillefer) (Fig. 8-32), which can penetrate the gutta-percha mass more efficiently than the more flexible types of K-files. The newly introduced C+ file is a stainless steel, end-cutting hand file that is twisted from a square blank. The secret to its stiffness is that the taper varies along the shaft, giving it the rigidity and strength to cut through well-condensed gutta-percha efficiently. The gutta-percha must be removed carefully, however, to avoid overextending the resultant mixture of gutta-percha and solvent beyond the confines of the canal to minimize the risk of severe postoperative pain.[141] Electronic apex locators are accurate in treatment and nonretreatment situations[123]; however, they seem

FIG. 8-32 C+ files. These rigid instruments remove gutta-percha more efficiently than more flexible types of K-files. (Courtesy Dentsply Maillefer, Ballaigues, Switzerland.)

to misread the working length frequently when gutta-percha is initially being removed. This clinical observation may be due to the file being covered with chloropercha that may affect its conductivity. It has been shown that apex locators may be less accurate in retreatment situations[258]; however, in this study the error was that readings indicated a working length that was too short. In a more recent study, an apex locator built into a rotary handpiece indicated working lengths that were too long in simulated retreatment situations.[252] It is recommended that a radiograph be made to gain a preliminary measurement when the estimated length is approached in order to avoid overextending root-filling materials into the periodontium. Well into the retreatment, after the root fillings have been thoroughly removed, the apex locator will regain its accuracy if a clean file is used. Once the working length is reached, progressively larger diameter hand files are rotated in a passive, nonbinding, clockwise reaming fashion to remove the bulk of the remaining gutta-percha until the files come out of the canal clean (i.e., with no pink material on them). The solvent should be replenished frequently, and when the last loose fitting instrument is removed clean, the canal is flooded with the solvent, which then acts as an irrigant. The solvent is then removed with paper points. The wicking action of the absorbent points[187] will remove much of the remaining film of gutta-percha and sealer that remains adhered to the canal walls and in the irregularities of the canal system.[265] Verification of the cleanliness of canals after gutta-percha removal is not improved by merely using a microscope[12]; however, using kinked small files, the clinician should probe the canal wall looking for irregularities that may harbor the last remnants of gutta-percha. These irregularities can usually be felt rather than seen and should be cleaned using this method.[141]

It should be noted that there exists a glass ionomer based endodontic sealer (Ketac-Endo, ESPE, Seefeld, Germany) that is used in conjunction with gutta-per cha.[180] This sealer is virtually insoluble in both chloroform and halothane,[261] and it must be retreated by removing the gutta-percha and then by using ultrasonics to debride the canal walls. Canal cleanliness can approach that of other gutta-percha retreatment cases, but it is difficult and time consuming to achieve this result.[67,159]

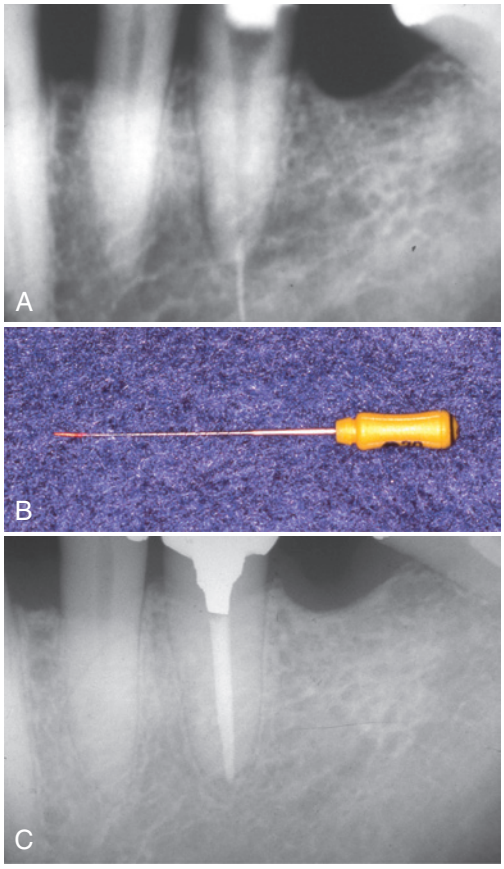

FIG. 8-33 Removal of overextended gutta-percha **A**, Preoperative radiograph showing overextended filling material. **B**, A small Hedstrom file pierces the overextended material and retrieves it. **C**, The 18-month reevaluation. The tooth is asymptomatic.

Overextended gutta-percha removal can be attempted by inserting a new Hedstrom file into the extruded apical fragment of root filling using a gentle clockwise rotation to a depth of 0.5 to 1 mm beyond the apical constriction, which may engage the overextended obturation. The file is then slowly and firmly withdrawn with no rotation, removing the overextended material[156] (Fig. 8-33). This technique works frequently, but care must be taken not to force the instrument apically, which furthers the extrusion of the gutta-percha; in addition, the file may separate. The overextended apical fragment should not be softened with solvent, as this application can decrease the likelihood of the Hedstrom file getting a solid purchase of the apical extrusion.[227]

Using rotary systems to remove gutta-percha in the canals has been advocated due to enhanced efficiency and effectiveness in removing gutta-percha from treated root canals.[187] This has generally been borne out in the literature. Several types of mechanical rotary systems are available for gutta-percha removal, including rotary file systems such as the ProFile (Dentsply) (Fig. 8-34), a mechanical push-pull, quarter-turn file system; the Canal Finder (Endo Technique Co., Tustin CA); and dedicated gutta-percha removal instruments, such as the GPX (Brasseler USA), the ProTaper Universal retreatment files (Dentsply) (Fig. 8-35), and the Mtwo R (Sweden and Martina, Due Carrare, Italy). These engine-driven instruments mechanically chop up the gutta-percha and sealer

FIG. 8-34 Nickel-titanium rotary Profile thermoplasticizing and removing gutta-percha. Optimum rotary speed is 1500 rpm.

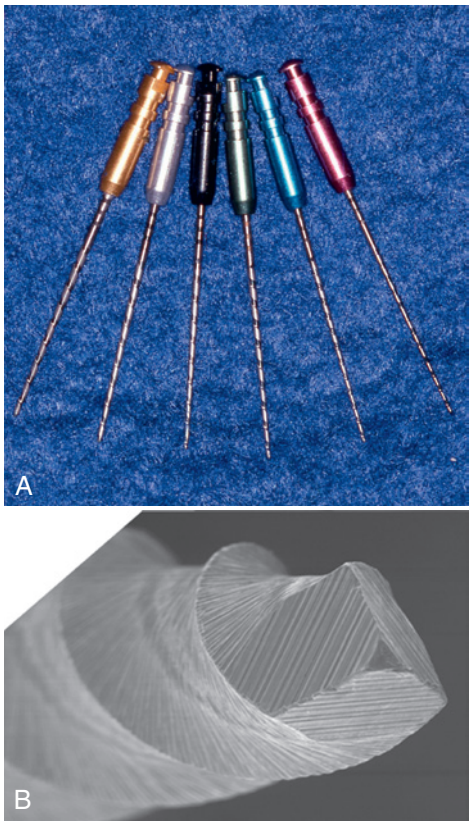

FIG. 8-35 Rotary gutta-percha removal instruments. A, Brasseler GPX instruments. B, ProTaper Universal retreatment file has a cutting tip for enhanced penetration of the root-filling materials.

while thermoplasticizing the root filling mass via frictional heat to aid in removal. A survey of Australian dentists showed that 54% of the respondents who perform endodontic retreatment used rotary instrumentation to remove gutta-percha either always (15%) or sometimes (39%) with an increased likelihood of rotary gutta-percha removal if the clinician had more experience with the use of these instruments.[173] In vitro studies have generally shown these systems to be efficient in that they typically need less time to remove the bulk of the gutta-percha filling material than is needed for hand removal,[13,59,77,103,104,190,242] although in two studies, they were slower to remove the root filling than hand filing[15,108] Assessments of canal cleanliness and extruded apical debris generally indicated that there were no overall differences between hand and mechanical gutta-percha removal.[15,59,103,104,109,168,190,218,242,277] In one study using Quantec SC instruments (Kyocera Tycom Corporation, Irvine, CA), however, it was found that hand files with solvent cleaned canals more effectively.[22] This finding has been repeated using the ProTaper retreatment files[92]; however, in another study in the same journal issue, ProTaper retreatment files were found to leave canals cleaner than hand files.[77] Clearly, this is an area where further research is warranted. It is recommended that after rotary gutta-percha removal, subsequent hand instrumentation is needed to remove the residual obturating materials completely from the canal. In several studies of mechanical gutta-percha removal, either the mechanical instruments or the tooth root fractured.[13,22,27,104,108,242] However, this result was reported to occur less frequently when the instrument rotary speed was increased from 350 to 1500 rpm,[27] and one study showed no separation or other canal defects when using dedicated retreatment files, the ProTaper Universal and Mtwo R instruments.[220] The dedicated retreatment files have end-cutting tips to enhance penetration and removal of the root filling mass, thus increasing their efficiency[239] (Fig. 8-35, *B*). This, in combination with flute design and techniques advocated, may be the reason for the potential reduced risk of separation. Although the mechanical gutta-percha removal systems may provide an enhanced efficiency, the increased risk of instrument separation, further complicating retreatment, may outweigh this benefit. Dedicated retreatment files may reduce this risk.

Engine-driven instruments can also help with the removal of residual root-filling materials after the bulk of the gutta-percha has been removed. A new instrument, the Self-Adjusting File (SAF; ReDent, Ra'anana, Israel), has been recommended for removal of the root-filling residue that remains after root-filling removal. When the Self-Adjusting Files were used after rotary retreatment files, reduction of canal residues were 66%, 68%, and 81% in the coronal, apical, and middle thirds of the canal, respectively, when compared with using ProTaper Universal retreatment files alone.[3] Another study compared the use of ProTaper Retreatment files followed by F1 and F2 ProTaper instruments with the use of a #25 .06 Profile followed by the Self-Adjusting File for their effectiveness in removing gutta-percha-based root fillings.[219] With the use of high-resolution micro-CT and an arbitrarily selected threshold of less than 0.5% residue remaining as effectively cleaned, none of the ProTaper group cases met this threshold, whereas 57% of the cases in the ProFile/SAF group met the threshold. Unfortunately, in both of the previously mentioned studies, none of the retreatment methods rendered all of the treated canals completely free of all root-filling residue.

FIG. 8-36 Epiphany Obturation System using Resilon material.

FIG. 8-37 Endosolv-E *(left)* and Endosolv-R *(right)*.

Use of the Nd:YAG laser to remove gutta-percha from root-filled teeth has been investigated in vitro.[254] The time taken to remove the gutta-percha was within the range of other studies of mechanical gutta-percha removal, and the addition of solvents did not improve the performance of the laser. As in most other studies, gutta-percha, in varying amounts, was left in the canals after laser removal. Root surface temperatures did increase, however, and without further investigation proving safety and efficacy, laser gutta-percha removal cannot be recommended at this time.

Resilon (Resilon Research LLC) (Fig. 8-36) is a thermoplastic polyester polymer that is bonded into the canal space using an unfilled resin bonding system (Epiphany, SybroEndo, Orange, CA). It is also marketed as RealSeal (SybroEndo). It has been advocated as a root canal obturating material to replace traditional gutta-percha and sealer due to its apparent, enhanced sealing ability[210] and potential to strengthen root resistance to fracture as a result of internal bonding.[241] Resin-bonded obturation systems have been advocated in the past[135]; however, the difficulty to retreat canals filled with this obturating material has prevented its widespread use. The Resilon polymer itself is reported by the manufacturer to be soluble in chloroform and may be removed by heat application, a behavior that is similar to gutta-percha. Studies have shown that the polycaprolactone polymer of the Resilon is removed easily and leaves canal walls cleaner than removal of gutta-percha and AH+ sealer,[44,46,56,199] although this finding has been disputed. Hassanloo and colleagues found that there was less residue on the walls when removing gutta-percha/AH+ if the materials were allowed to set for a longer period.[97] This finding has been corroborated[199,240] and indicates that there may be a temporal effect, which introduces a methodological bias in these studies. There may also be a problem with removal of the unfilled Epiphany resin sealer, especially because the sealer tags have been shown to penetrate deep into dentinal tubules[210] and presumably also into anatomic ramifications of the canal that need cleaning during retreatment. More research into this interesting material and technique is warranted, especially to determine the best technique for retreatment. After the Resilon core has been removed using heat and chloroform, the authors would recommend the use of a resin solvent such as Endosolv-R (Septodont, Paris, France) (Fig. 8-37) to attempt elimination of the unfilled resin sealer prior to instrumentation.

Managing Solid Core Obturators

Solid core canal obturation systems, such as Thermafil, Dens-Fil, and the GT Obturator (DENTSPLY Tulsa Dental Specialties, Tulsa, OK), have become popular since their introduction several years ago (see also Chapter 7). After cleaning and shaping procedures are completed, the clinician, using this technique, heats a solid core obturator (alpha-phase gutta-percha surrounding a core that is attached to a handle) in an oven and places the carrier in the canal. The solid core carries the gutta-percha down in the canal and condenses it while the material is cooling. This system provides a rapid and simple technique for warm gutta-percha endodontic obturation; however, as with any obturating material, retreatment will be necessary occasionally.

Retreatment of solid core materials is considered to be more complex and difficult than is the case with removal of gutta-percha alone due to the presence of the solid carrier within the mass of gutta-percha. The nature of the carrier will determine the method used and complexity of the retrieval. Three types of carriers are found in these systems: metal (stainless steel or titanium), plastic, and modified gutta-percha. The plastic carriers are smooth sided, as are some brands of metal carriers; however, most metal carriers are fluted and resemble endodontic hand files with a layer of gutta-percha on the outside. The fluted metal carriers present an exceptional challenge to the retreating clinician because many times they are improperly inserted and either wedged or screwed into the canal to make up for inadequate canal shaping or the lack of skilled use of the size verifying techniques available. This makes them especially difficult to remove. Once the carrier has been placed, it is cut off in the pulp chamber using a bur and the tooth is restored. The level at which the metal carrier is severed is important in its retrieval. If it is cut down to the level of the canal orifice, retrieval is difficult,[141] so the prudent clinician plans for retrievability by severing the handle from the carrier leaving 2 to 3 mm of carrier exposed in the access above the pulp chamber floor to allow easier removal if retreatment is ever needed. Unfortunately, this is not always the case. Some clinicians place a nick in the midcanal level of the carrier to

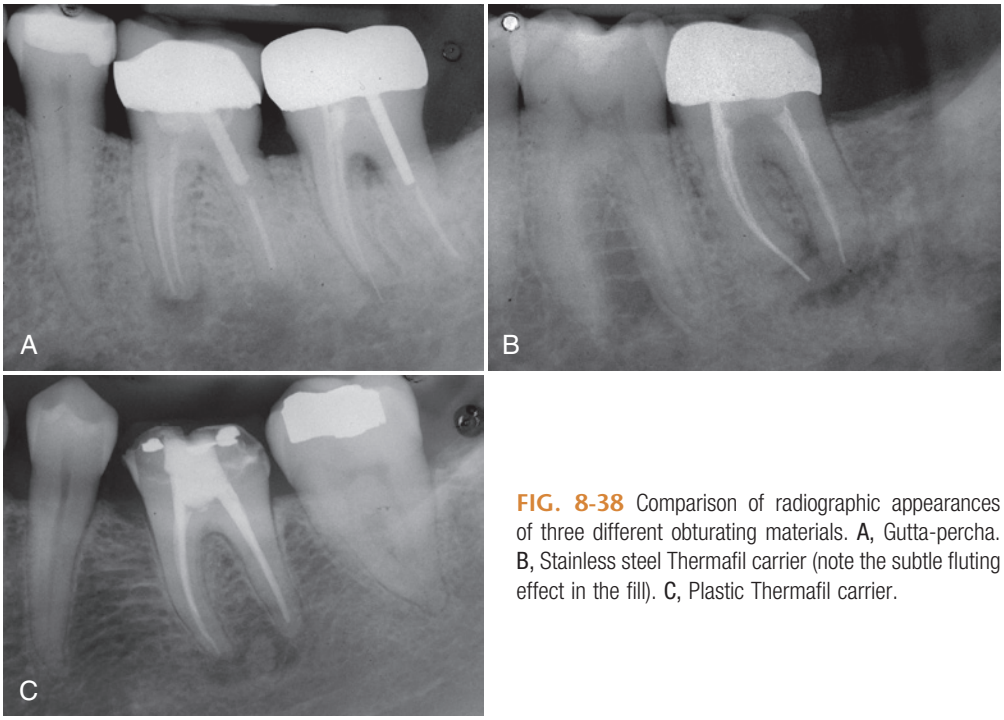

FIG. 8-38 Comparison of radiographic appearances of three different obturating materials. **A,** Gutta-percha. **B,** Stainless steel Thermafil carrier (note the subtle fluting effect in the fill). **C,** Plastic Thermafil carrier.

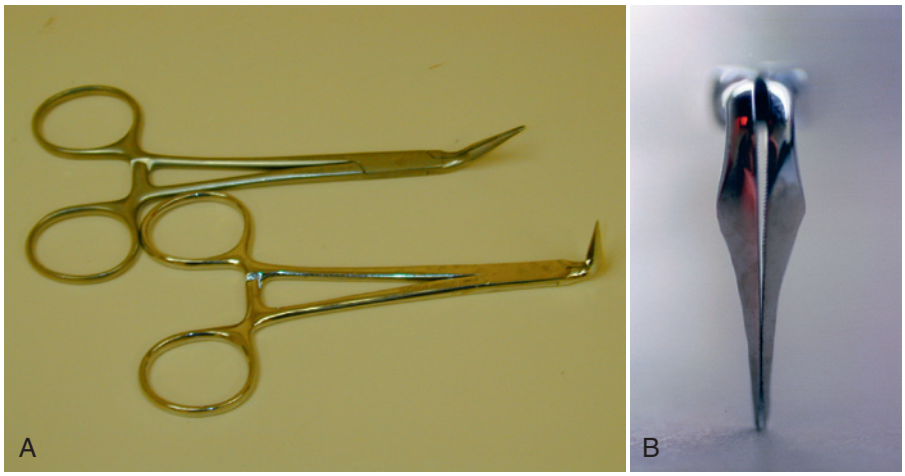

FIG. 8-39 **A,** Steiglitz forceps in 45- and 90-degree head angles. **B,** Tips of the Steiglitz forceps ground to a thinner contour to create the "modified" instrument. This allows deeper penetration into the tooth to enhance removal of obstructions. (*B,* Courtesy Dr. Daniel Erickson.)

allow the clinician to rotate the carrier handle and sever the obturator deep in the canal. This technique is used to allow creation of a post space; however, the rotational force used to create the "twist off" apical plug can engage the flutes of a metal carrier, increasing the complexity of removal if retreatment is needed.[276]

It is advantageous to determine prior to initiating treatment if there is a solid core obturation in the root-filled tooth. The preoperative radiograph may show this because the stainless steel carriers will exhibit a fluting effect on the radiograph (Fig. 8-38); however, the titanium carriers rarely are distinguishable from gutta-percha, and the plastic ones never are. Unfortunately, in most instances, the clinician finds that he or she is dealing with a carrier-based obturator after initial access to the pulp chamber. This is why, as stated in an earlier section, careful access and probing of the root-filling material is necessary when entering a canal. If there is a carrier, it will be detected as either a metallic structure embedded in the gutta-percha mass or a black or gray spot indicating a plastic or modified gutta-percha carrier. Occasionally, the carrier may be found embedded in the coronal core material, so careful excavation with small burs and straight, tapered ultrasonic tips may be necessary to preserve the carrier intact to help with removal.[141]

Removal of a metal carrier is accomplished with initial use of heat application to the carrier that can soften the gutta-percha surrounding it, facilitating its removal with Peet silver point forceps (Silvermans, New York, NY) or modified Steiglitz forceps (Union Broach, York, PA)[141,187,262,264] (Fig. 8-39). Often, there is not enough of the carrier remaining in the access to

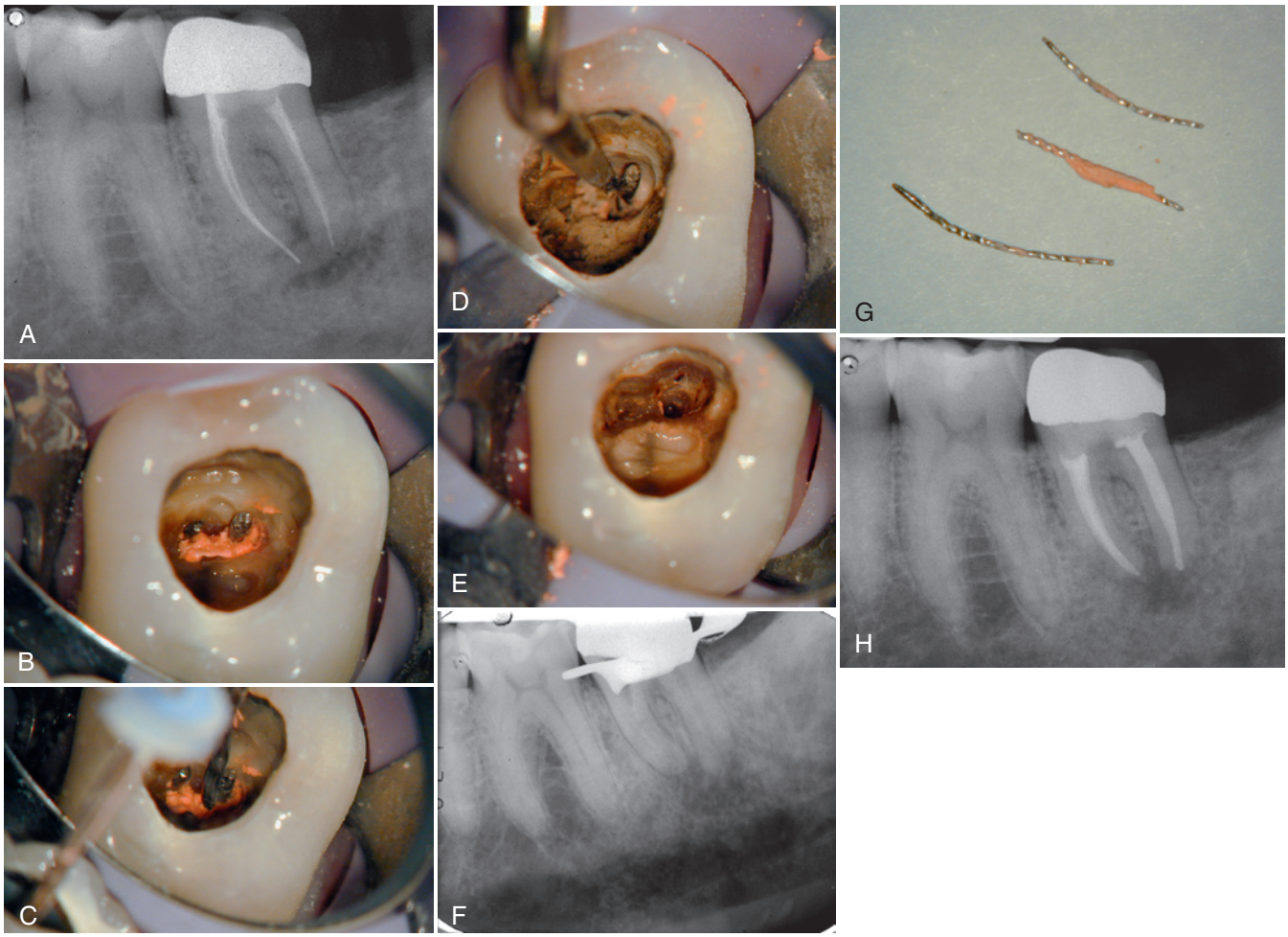

FIG. 8-40 Metal carrier retreatment **A,** Preoperative radiograph. **B,** Metal carriers exposed by careful excavation of gutta-percha. **C,** Use of the Touch 'n Heat instrument to heat the carriers and soften the gutta-percha. This allowed removal of one of the carriers using modified Steiglitz forceps. The other could not be removed using heat or solvents. **D,** Ultrasonic troughing around the carrier to facilitate grasping it with forceps. **E** and **F,** Carriers removed and confirmed with a radiograph. **G,** Metal carriers showing gutta-percha still adhering to them. **H,** Final obturation of the tooth.

grasp with forceps, so removal will require solvent application and removal of the surrounding coronal gutta-percha using small hand instruments, usually followed by ultrasonic excavation around the carrier and removing it like a separated instrument[141,187] (Fig. 8-40), as described in a later section of this chapter. Care should be exercised to avoid excessive heat generation during this procedure. This is also the case if the metal carrier has been sectioned for post space preparation. The metal carrier has been shown to be much more difficult to remove than plastic ones,[62,276] frequently resulting in nonretrieval. Fortunately, their use in endodontic therapy has been declining.

Removal of plastic carriers is similar to removal of gutta-percha root fillings, except that, in general, heat should be avoided to minimize the likelihood of damaging the carrier.[141] The older Thermafil plastic carriers were made of two different materials depending on their size. In the smaller sizes (up to size #40), the material used was Vectra, which is insoluble in available solvents, whereas the larger sizes used polysulfone, which is soluble in chloroform.[174] Solvents, on the other hand, seem not to affect the newer GT plastic carriers, so their use can be recommended[21] (Fig. 8-41). The access is flooded with a solvent, such as chloroform, and the gutta-percha surrounding the carrier is removed with hand files in a larger to smaller sequence (#25, 20, 15, etc.) each file progressively penetrating deeper around the carrier. The solvent should be replenished frequently, and when a #08 file can penetrate to the apical extent of the carrier and there is little remaining gutta-percha, a larger Hedstrom file is inserted into the canal alongside the plastic carrier and gently turned clockwise to engage the flutes. When the file is withdrawn, it invariably brings the carrier with it, and the rest of the gutta-percha and sealer removal proceeds as described earlier. Care must be taken to avoid overstressing the Hedstrom file. It should not be "screwed" into the canal, or the file or the carrier may separate.[21] Occasionally, grasping pliers will be needed to remove the carrier if it is accessible[110,187] after the gutta-percha has been removed. Another potential problem with retrieval is present if the carrier has been overextended beyond the apical foramen during the previous root canal treatment. This overextension may make it

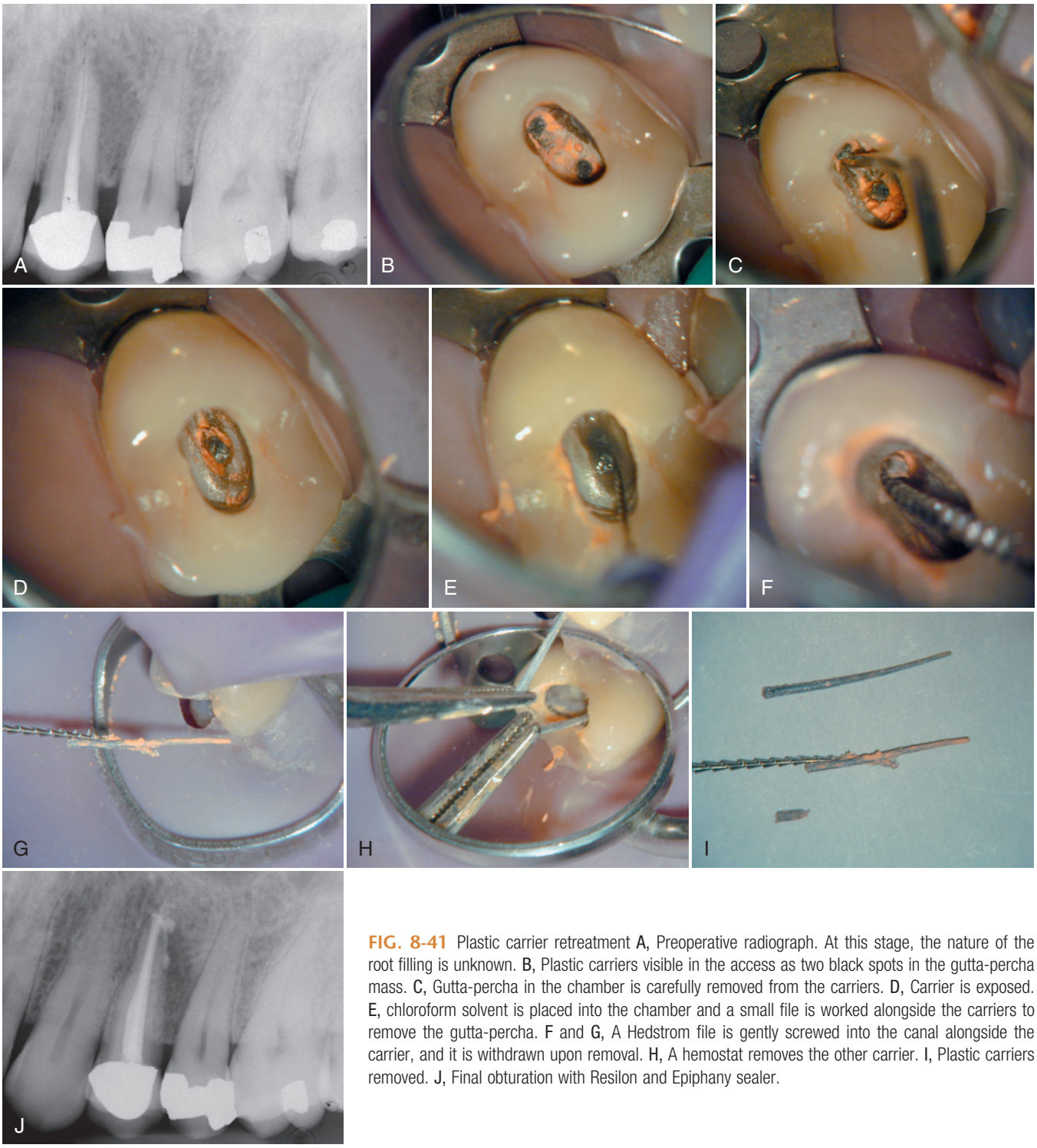

FIG. 8-41 Plastic carrier retreatment **A**, Preoperative radiograph. At this stage, the nature of the root filling is unknown. **B**, Plastic carriers visible in the access as two black spots in the gutta-percha mass. **C**, Gutta-percha in the chamber is carefully removed from the carriers. **D**, Carrier is exposed. **E**, chloroform solvent is placed into the chamber and a small file is worked alongside the carriers to remove the gutta-percha. **F** and **G**, A Hedstrom file is gently screwed into the canal alongside the carrier, and it is withdrawn upon removal. **H**, A hemostat removes the other carrier. **I**, Plastic carriers removed. **J**, Final obturation with Resilon and Epiphany sealer.

prone to separation and unable to be retrieved, potentially resulting in the need for apical surgery.[107]

A technique for plastic carrier removal has been described using a System B HeatSource (SybronEndo) to soften the gutta-percha surrounding the carrier without melting the carrier itself.[267] The temperature is set at 225° C, and the heat plugger is placed buccal and lingual to the carrier after which #50-55 Flex-R hand files are placed around the carrier and braided to engage the carrier and remove it. This technique has been shown to require significantly less time to remove the carrier compared with using solvent[267]; however, concerns regarding heat generation in the periradicular tissues have been raised,[139] with the authors concluding that caution should be exercised when using this technique. When other techniques have been unsuccessful and the plastic carrier has been sectioned apical to the orifice resulting in limited access, the clinician may attempt to retrieve the carrier by placing a heated System B tip directly into it. As apical pressure is maintained, the heat is

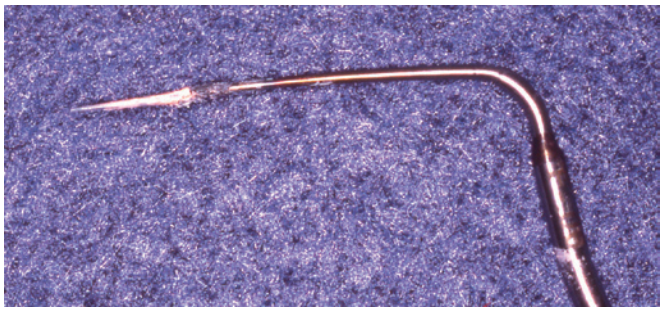

FIG. 8-42 Plastic Thermafil carrier adhering to a System B heat plugger.

turned off. This allows the plastic carrier to adhere to the tip while cooling and may result in its removal upon withdrawal of the heat tip (Fig. 8-42).

Rotary instruments have been advocated for use in removal of plastic carriers and gutta-percha, and one study showed that removal of plastic carriers was successful in all but one of the teeth obturated with them.[13] Unfortunately, root fracture occurred in the lone specimen that did not have a successful retrieval, and two instances of rotary instrument fracture also occurred in the study. As with gutta-percha removal, the clinician must carefully weigh the risks of rotary carrier removal against the perceived benefits.[186]

Modified gutta-percha has been introduced as a core material for solid core obturation. This obturation system is called GuttaCore (DENTSPLY Tulsa Dental Specialties) and, at first glance, it appears similar to plastic carrier based systems. With GuttaCore, however, the carrier is fabricated from cross-linked gutta-percha rather than plastic. The cross-linking connects the polymer chains, which changes the material and gives the carrier different properties than the plastic carriers. The gutta-percha surrounding the carrier is alpha-phase gutta-percha, which is identical to that which encompasses the plastic carrier based systems.

To understand how to effectively remove this type of filling material, one must understand how it is used. Obturation with GuttaCore is performed in a similar manner as obturation with Plastic Thermafil. The obturator is heated in a proprietary oven and then gently placed into the canal. Although it is flexible, the modified gutta-percha carrier is much more brittle than the plastic carrier and too much insertion force will cause the shank to collapse and the handle can break off prior to full insertion. Thus, it is difficult to wedge these obturators into underprepared canals and usually, in that instance, a short fill will result. This forces the operator either to accept a short fill or, ideally to enlarge the canal preparation prior to inserting the obturator, but in either case, retrieval of the root filling during retreatment is made easier by not having the carrier wedged into the canal.

While performing initial obturation, when it is time to section the carrier, the clinician will experience another difference from plastic carriers. Although the use of a heat-generating Prepi bur (DENTSPLY Tulsa Dental Specialties) or a System B Heat Source (SybronEndo) has been shown to produce predictably reliable results in terms of sectioning plastic carriers at the desired level, heat has virtually no effect on the GuttaCore carrier, and the authors found using heat to section the carrier to be unsuccessful. The modified gutta-percha core will not melt, even when probed with a Touch 'n Heat (SybronEndo) tip at full power.

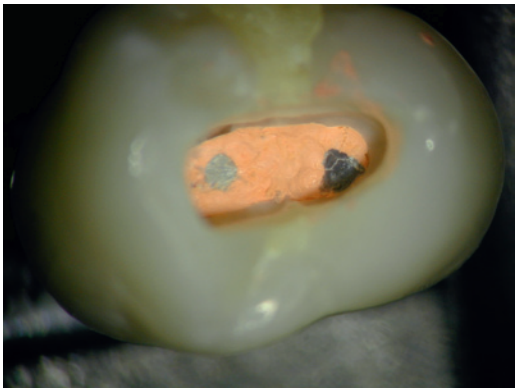

FIG. 8-43 Two different solid core carriers in an endodontic access preparation upon initial exposure. GuttaCore is on the left and a plastic carrier Thermafil Plus is on the right. Note the lighter gray color of the new modified gutta-percha core compared with the plastic carrier.

Because of the brittleness of the modified gutta-percha carrier, GuttaCore will separate by simply bending the carrier away from the access cavity wall. The material has little ability to resist even the smallest of lateral forces without separation, so simply pushing down laterally on the carrier handle will separate the carrier handle from the canal fill. This has implications for retreatment because this separation method generally results in the carrier being separated at or near the canal orifice level, leaving little or nothing to grasp with forceps when attempting to remove the carrier.

Removal of the GuttaCore obturators is directly affected by the properties of the modified gutta-percha carrier, so it is somewhat different from the approach taken to remove plastic carriers. For this reason, it is imperative that clinicians who discover a solid core, nonmetallic obturator while disassembling a tooth for nonsurgical retreatment can discern what type of core they are trying to remove. There are two ways to do this. First, the color of the plastic carrier is black, whereas the color of the modified gutta-percha carrier was originally gray (Fig. 8-43); however, the manufacturer recently changed the color of the carrier to pink. Second, because the GuttaCore carrier is heat resistant, the clinician can lightly touch the coronal extent of the unknown carrier to see if it melts (plastic carrier) or if it does not (modified gutta-percha carrier). Once the nature of the carrier is determined, then the clinician can proceed with removal. If it is plastic, then the methods of removal described previously can be used, but if it is a GuttaCore carrier, then a different approach will be needed.

The use of heat and solvents, which are two of the most common methods for endodontic retreatment, have no effect on the GuttaCore carrier. To date the authors are unaware of any known solvents or chair side heat sources that can soften the cross-linked gutta-percha carrier. Also, the use of hemostats or other grasping pliers have shown inconsistent results. When straight-line access can be achieved, and a few millimeters of the carrier is able to be grasped, removal may be accomplished; however, due to the brittleness of the GuttaCore carrier, often the carrier will separate at the apical extent of the grasping forceps. In spite of those concerns, the authors have found that GuttaCore can be removed easily with predictable results using a variety of hand, rotary, and retreatment instruments. One study found that when using ProTaper retreatment files, GuttaCore was more efficiently removed from moderately

curved canals than either thermoplasticized gutta-percha or plastic carrier obturation.[17] However, the authors' experience shows that upon removal of the segment of the carrier within the canal, any overextended tip of the carrier will separate, remaining in the periapical tissues. This potentially contaminated foreign material becomes difficult and many times impossible to remove. If this occurs, and postretreatment apical periodontitis persists or recurs after initial resolution, then apical surgery or extraction will be necessary.

Another approach is to treat the carrier like some hard paste fills that the clinician is unable to find a solvent for. Using this approach, the clinician will find that using ultrasonic instruments in a similar fashion as they are used to remove pastes to be helpful. Once a proper access preparation has been created, GuttaCore can be safely and easily removed with the use of ultrasonic instruments in the straight coronal portion of the canal. Upon reaching the canal curvature, the authors recommend using hand files or rotary files with safe noncutting tips to avoid perforation or excessive thinning of canal walls.

Once the carrier has been removed, the remaining gutta-percha and sealer must be removed from the canal, and like removal of root-filling materials described previously, there is no technique that completely removes all materials from the canal system. Canal cleaning may be even more difficult when removing carrier-based obturations because the more highly processed gutta-percha used with these carriers may be more difficult to remove than other forms of the material. Wilcox and Juhlin described a sticky film of gutta-percha and sealer adhering to the canal walls upon removal of metal carrier obturators and found more of it than if the canals had been obturated with lateral condensation.[264] Their findings have not been corroborated, however, and other studies have shown no difference in residual debris remaining after carrier-based removal.[62,110,276] It is important to remove as much of the residual gutta-percha and sealer from the pulpal anatomic spaces as possible, so flooding the canal with solvent and "wicking" it out with paper points is also recommended for carrier based retreatment.[187]

Paste Retreatment

Various pastes have been used as root canal obturating materials, especially outside of North America. Because of the wide variety of paste compounds used in endodontics, it is impossible to categorize them all. The individual clinician who is using it formulates most pastes, so the ultimate composition of a paste found in a tooth with persistent disease is generally indiscernible. Many of the pastes used, such as N2 or RC2B, contain formaldehyde and heavy metal oxides and so are toxic and potentially present a danger to the patient's health, both local and systemic, if overextended beyond the confines of the root canal system.[25,172] None has the potential to seal the canal effectively,[91] and many render a tooth impossible to retreat,[200] so their use is strongly discouraged. On radiographic examination, they can usually be discerned due to their lack of radiopacity, the presence of voids, and they usually show evidence of inadequate canal shaping and poor length control (Fig. 8-44). When a paste fill is suspected or found in a tooth, a telephone call to the previous treating dentist should be made, if possible, to find out the exact formulation of the paste because this information may help in its removal.

For purposes of retreatment, paste fills can be categorized as soft or hard, and all should be considered potentially toxic. Great care should be exercised when removing the paste to avoid overextension, potentially severe postoperative pain,[87] and possible paresthesia/dysesthesia from the paste's potential neurotoxicity.[28,207] Soft pastes are generally easy to remove using crown down instrumentation with copious sodium hypochlorite irrigation to minimize extrusion.[141] Greater difficulty arises when the paste is set hard.[200] Because the nature of the paste remains unknown, removing it becomes an empirical process. Following access preparation and coronal orifice exposure, the paste is probed with an endodontic explorer and files. If hard and impenetrable, then the coronal paste can be removed with burs[69] or a straight, tapered ultrasonic tip in the easily accessible straight portions of the canal using magnification and illumination.[187] Once the canal curvature is reached, further use of this method will result in damage to the canal walls and possible perforation. Precurved, small hand files are inserted to probe the apical area. Many times the density of the paste filling material decreases in the apical extent of the fill so that penetration to the apex may be possible.[187] If not, a solvent must be used to attempt to soften the remaining paste. The choice of solvent is usually made by trial and error starting with chloroform. If that does not soften the material in a reasonable amount of time and does not allow penetration with small files, then the chloroform is wicked out of the canal and another solvent is chosen. There are two frequently used solvents for paste fills: Endosolv-E and Endosolv-R (Septodont, Paris, France) (see Fig. 8-37). The Endosolv-E is selected if the paste contains zinc oxide and eugenol, and the Endosolv-R is chosen for resin-based pastes. The obvious problem is that the nature of the paste is usually unknown at the time of removal, so contacting the previous clinician, if possible, can help with this choice. Otherwise, the choice is just a guess. The chosen solvent should be placed in the access, and attempts should be made to penetrate the paste with hand or ultrasonic files; however, care must be taken to avoid creating a ledge or other defect in the canal that may preclude successful retreatment. The progress is frequently slow,[72] and the clinician may elect to leave some solvent in the canals between appointments to soften the paste.[187] Care should be taken in the choice of temporary restoration because the solvent left in the canal may also soften the temporary, potentially leading to breakdown of the interappointment seal.[158]

Ultrasonically activated files have been advocated for use in penetrating hard-set pastes in the curved apical segments of canals[117,126] (Fig. 8-45). The ultrasonic energy breaks up the paste, and the irrigation floats the fragments in a coronal direction until the apical terminus is reached.[69] This technique is reported to be time consuming, and care must be exercised to avoid instrument separation, perforations, or alteration of canal morphology. On occasion despite all best efforts, the paste cannot be removed from the tooth, so apical surgery or extraction should be considered in these cases.[87]

Biocalex 6.9 (currently known as Endocal 10) (Fig. 8-46) is a hard-setting calcium oxide paste that has been popular in Europe since the 1980s but is now being used in North America since its recent FDA approval.[83] The paste seems to seal well, but there is an unacceptably high incidence of root fracture due to the large amount of expansion on setting.[83] Retreatment will be complicated by the hard setting nature of this material; however, as it is a calcium oxide paste, ethylenediaminetetraacetic acid (EDTA) may soften it, facilitating its removal. Because EDTA also softens dentin, care must be taken not to

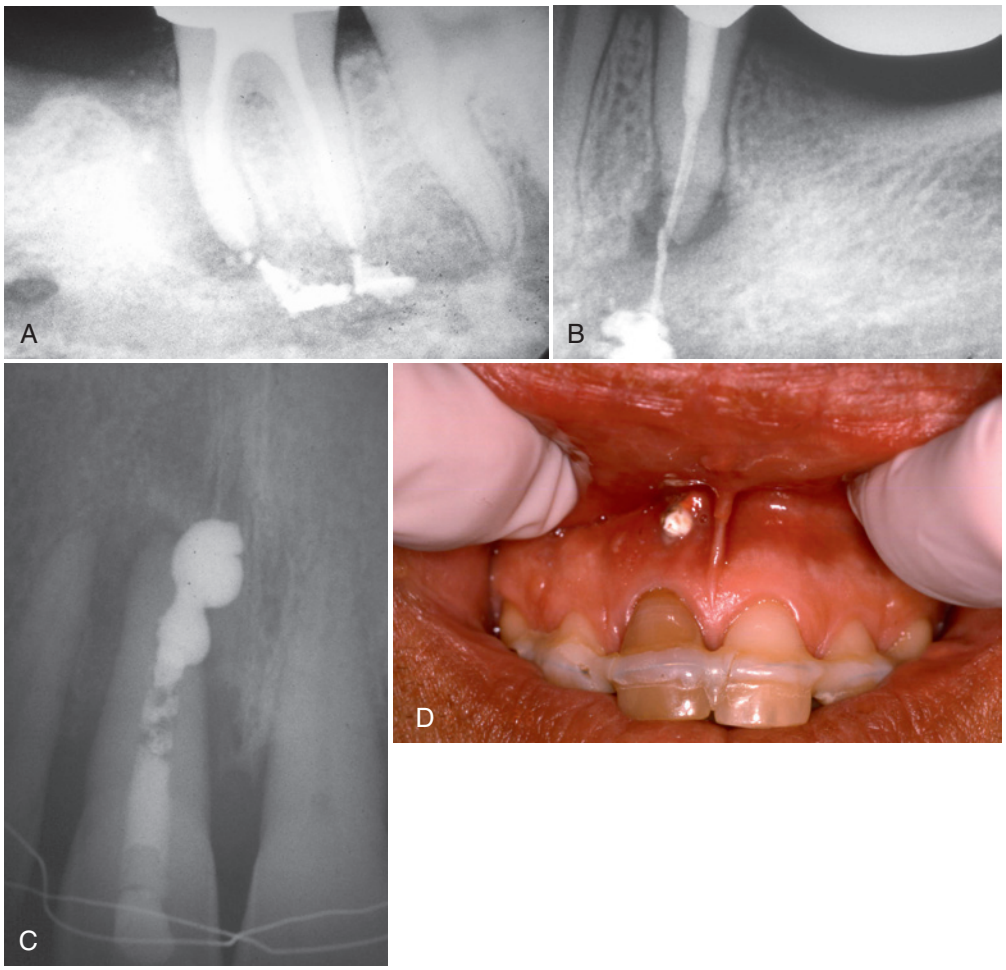

FIG. 8-44 Example of poor length control with paste root fillings **A,** Overextended paste filling into the inferior alveolar canal. **B,** Overextended paste filling into the mental foramen. **C,** Overextended paste filling extending through a perforation in an upper central incisor. **D,** Clinical appearance of the case in Fig. 8-41, *C*. Note the material extending out through a sinus tract.

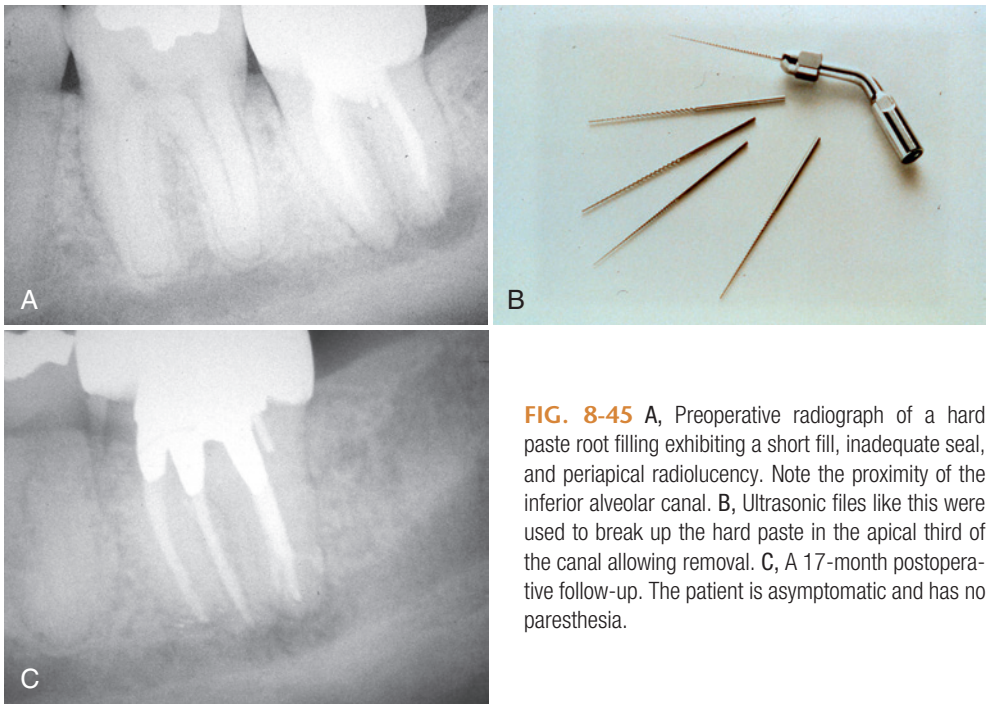

FIG. 8-45 **A,** Preoperative radiograph of a hard paste root filling exhibiting a short fill, inadequate seal, and periapical radiolucency. Note the proximity of the inferior alveolar canal. **B,** Ultrasonic files like this were used to break up the hard paste in the apical third of the canal allowing removal. **C,** A 17-month postoperative follow-up. The patient is asymptomatic and has no paresthesia.

FIG. 8-46 A, Endocal 10 (formerly known as Biocalyx). B, Split root in a case filled with Endocal 10. (Courtesy Dr. Rob Goldberg.)

gouge or ledge the canal walls during retreatment, and root fractures must be suspected as a potential complicating factor in the outcome.

Silver Point Removal

Historically, the use of silver points for endodontic therapy has been extremely popular and quite successful, because of their ease of handling and placement, ductility, radiopacity, and that silver appears to have some antibacterial activity.[202] However, the use of silver points has dramatically diminished, so presently they are considered a deviation from the standard of care.[274] The main reason for this change is because they corrode over time (Fig. 8-47), and the apical seal may be lost.[26] Also, silver points do not produce an acceptable three-dimensional seal of the canal system; rather, they simply produce a plug in the apical constriction while not sealing the accessory canals that are frequently present[143,198] (Fig. 8-48). The corrosion of silver points occurs when they come in contact with tissue fluids and certain chemicals used in endodontics, including sodium hypochlorite and some sealers.[88] This corrosion produces chemicals such as silver sulfide, silver sulfate, silver carbonate, and silver amine hydrate,[205] which have been shown to be cytotoxic in tissue culture.[202] Corrosion occurs mainly at the apical and coronal portions of the points indicating that leakage is responsible.[205] Gutta-percha root-filling techniques

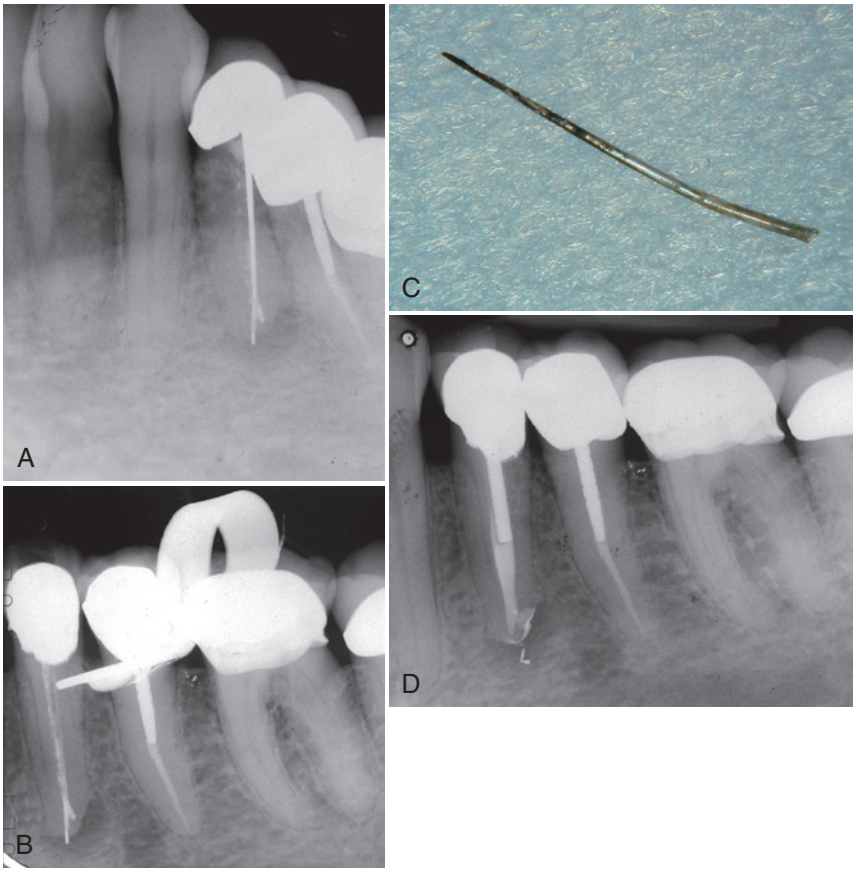

FIG. 8-47 A, Persistent disease in a silver point filled tooth. B, Silver point removed. Note the radiopaque material in the apical portion of the canal system. This represents corrosion products remaining in the canal and a possible separated apical segment of the cone. C, Removed silver point showing black corrosion products adhering to the apical one half. D, Crown-down instrumentation prevents extrusion of most of the corrosion products into the periradicular tissues.

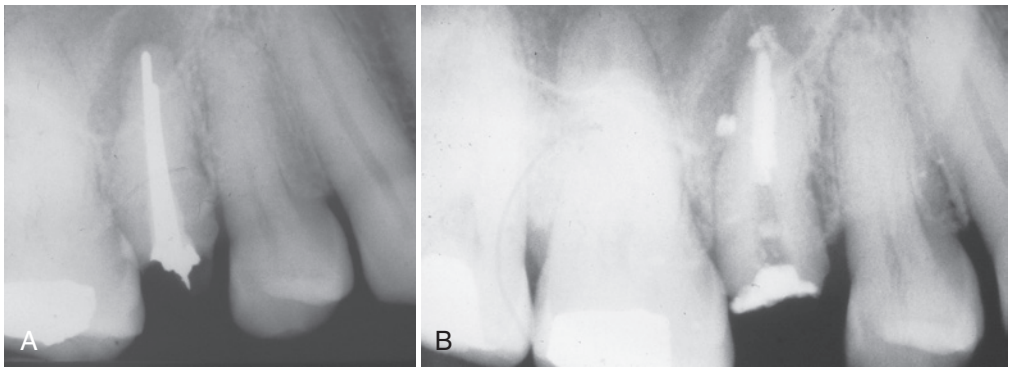

FIG. 8-48 A, Preoperative view of a silver point case with persistent disease. Note that the periapical radiolucency extends coronally on the distal aspect of the root end, indicating the presence of an unfilled lateral canal. B, Postobturation radiograph showing the cleaned and filled distal canal branch.

do not suffer from these disadvantages and have replaced the use of silver points in endodontics. Due to this decrease in use since the late 1980s, the quantity of cases the clinician will come across that will require silver point removal has also decreased. Nevertheless, there are still occasions when their removal will be necessary.

Many of the same techniques described for removing separated instruments in the following section apply to the removal of silver points. Silver points have a minimal taper and are smoothed sided, and corrosion may loosen the cone within the preparation. Therefore, the clinician should encounter a much easier time removing them than would be the case with separated instruments, which may be mechanically engaged into canals. Silver point canal preparation techniques produced a milled, round preparation in the apical 2 to 3 mm of the canal and, coronal to that, the clinician will frequently find space between the round silver point and the flared canal walls that can usually be negotiated with hand files facilitating point removal.[187]

The first step in removal of silver points is to establish proper access. Frequently, the coronal portion of the cone is embedded in the core material. This material must be carefully removed with burs and ultrasonics, taking care not to remove any of the silver point within the access cavity preparation. The more of the silver point the clinician has to work with, the more predictable will be its removal. Once proper access is established, the clinician should flood the access preparation with a solvent, such as chloroform, to soften or dissolve the cement, enabling easier removal. An endodontic explorer or small file may be used to carry the solvent down along the silver point to dissolve as much of the cement as possible. The chamber can be rinsed and dried, and this step may be repeated because fresh solvent enhances the efficiency of cement removal. At this point, the easiest technique, which is also predictable, is to grasp the exposed end of the silver point with Stieglitz pliers (Henry Schein) (see Fig. 8-39) or other appropriate forceps and gently pull it out of the access cavity preparation. If too much extraction force is needed, however, the point may separate, so slow force application is advised. The clinician will need a variety of sizes and angles of forceps available to deal with the variety of cases that will need to be treated. Occasionally, the forceps may not get a good purchase on the silver cone and will slip off. In these instances, gripping the cone with the forceps and then gripping the forceps in a hemostat or needle driver to increase the squeezing force of the forceps will allow removal of the cone[141] (Fig. 8-49). If the silver point is held tight by the frictional fit in the preparation, indirect ultrasonics may be employed to loosen it. The silver point is retained in a pair of forceps and ultrasonic energy is applied to the forceps, not the point (Fig. 8-50). This transmits energy down the cone and may loosen it. If there is not much of the silver point exposed in the chamber, the clinician can attempt to remove it using the Caufield silver point retrievers (Integra Miltex). This instrument is a spoon with a groove in the tip (Fig. 8-51) that can engage the exposed end of the silver point so it may be elevated from the canal or possibly elevated to the point where it may be grasped by forceps.[141] The

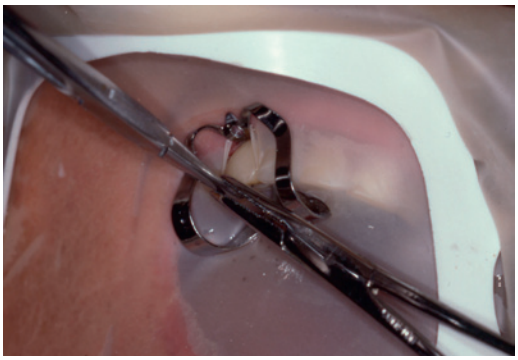

FIG. 8-49 Removal of a highly retentive silver point using a needle driver to squeeze the tips of the Steiglitz forceps. This applies increased gripping force to aid in removal.

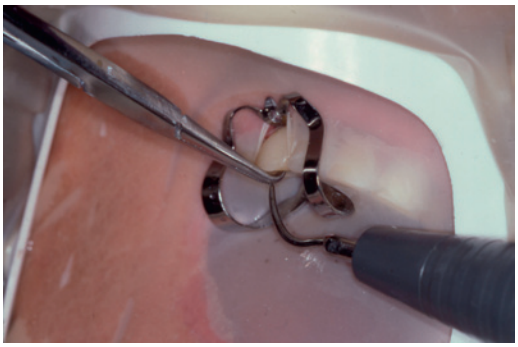

FIG. 8-50 Application of indirect ultrasonic energy to a silver point by placing the ultrasonic tip against forceps that are holding the silver point.

Caufield silver point retrievers are available in three sizes: 25, 35, and 50.

If the silver point cannot be dislodged by the forgoing techniques, the clinician should consider using Hedstrom files to remove the silver point. The Hedstrom file technique requires at least some coronal length of canal space around the silver point to be negotiated first.[140] The sealer is dissolved as previously mentioned, and then files are negotiated as far apically as possibly in two to three areas around the silver point. If only one space can be negotiated, this technique may still be effective. The spaces surrounding the silver point are carefully instrumented to size 15, and then small Hedstrom files are gently screwed in as far as possible apically. They should not be screwed in too tightly so as to prevent breakage. The flute design of Hedstrom file allows for much better engagement into the silver point compared with other file designs. The files are then twisted together and pulled out through the access (Fig. 8-52). If the first attempt fails, this technique may be repeated, possibly using larger Hedstrom files. If this technique does not completely remove the silver point from the canal, it may still be dislodged to the point where it can be grasped by forceps and removed.

If the clinician needs to expose more of the silver point to enable removal, the use of trephine burs and microtubes or ultrasonics may be necessary.[69] Trephine burs are used in the same manner as described for separated instrument removal; however, the clinician must be more careful when using ultrasonic instruments for retrieval of silver points. When using ultrasonics for post removal, or separated instrument removal, the tip of the ultrasonic instrument can be placed at the interface between the obstruction and the canal wall. Although applying the ultrasonic energy directly to a post or file may prove beneficial in vibrating them loose, silver points are much softer, and if ultrasonic instruments are applied directly to them, the portion in contact may be shredded leaving a smaller segment to work with because elemental silver rapidly erodes during mechanical manipulation.[187] The ultrasonic instrument is used on tooth structure circumferentially around the silver point. This is a delicate process requiring a microscope or other powerful source of magnification. The energy supplied by the careful use of the ultrasonic instrument can safely expose silver points as well as break up the cement around them.

In many cases, a silver point may have been sectioned deep in the canal to allow post space preparation. In these cases, where the most coronal portion of the silver point is well below the orifice, the use of Gates-Glidden burs to obtain straight-line access to the most coronal extent of the point may be necessary. The burs should be used in a brushlike manner, cutting on the outstroke while applying gentle pressure in an anticurvature direction to decrease the risk of root perforation. Following this step, techniques that involve the use of an end-cutting trephine bur to remove tooth structure around the point and then use of an extraction device to remove it may be employed (Fig. 8-53). There are many kits that use these principals with slight variations from one another, including the Endo Extractor (Brasseler USA),[226] the Masserann Kit (Medidenta International, Woodside, NY), and the Extractor System (Roydent, Johnson City, TN) (Fig. 8-54). Additional techniques, which are effective for removing silver points, include the S.I.R. (Separated Instrument Retrieval) System (Vista Dental Products, Racine, WI), the use of a dental injection needle with a 0.14-mm wire, the use of stainless-steel tubing with a Hedstrom file,[187] and the Instrument Removal System (Dentsply Tulsa Dental Specialties).

These kits are not only effective in the removal of silver points but also in the removal of separated instruments, and because of this common approach to removing both during endodontic retreatment, these techniques will be discussed

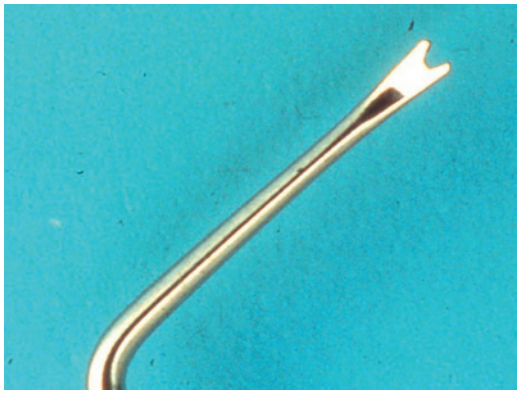

FIG. 8-51 Caufield elevator tip, useful for gripping and elevating silver points that are protruding a small amount into the pulp chamber.

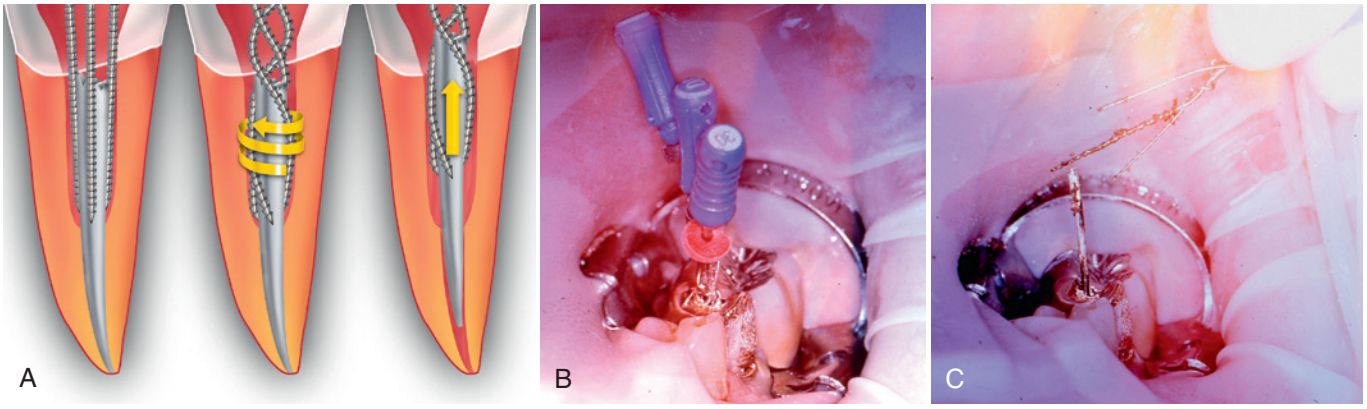

FIG. 8-52 A, Diagram illustrating the braiding of Hedstrom files around a silver point. By twisting the braided files, a gripping force is applied, which aids in removal of the obstruction. B, Small files being braided around a silver point. C, Pulling coronally with the braided files removes the silver point.

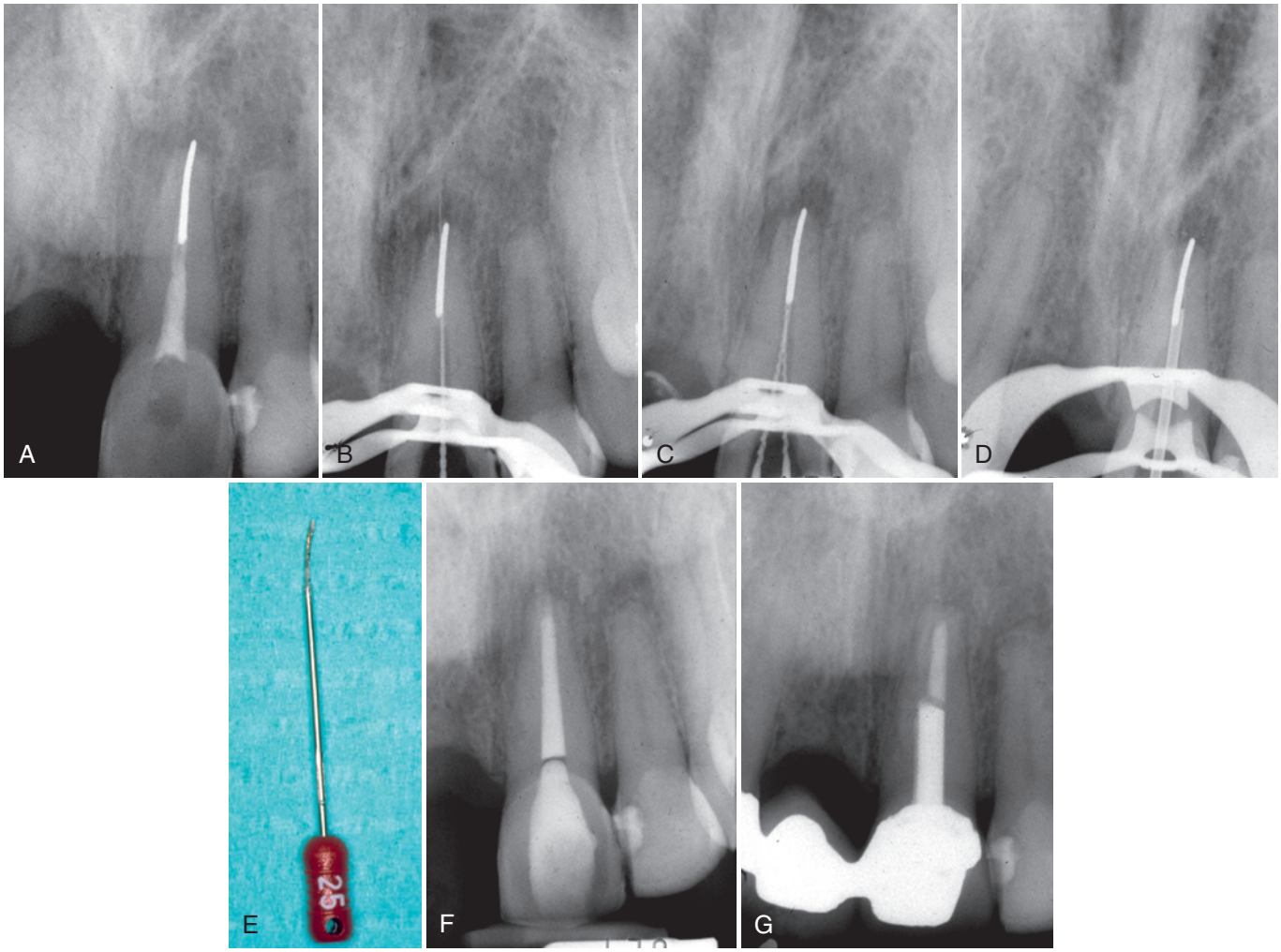

FIG. 8-53 Twist-off silver point case **A,** Preoperative radiograph showing apical periodontitis and a split silver cone ("twist-off) obturation technique. **B,** The cone was initially bypassed but could not be loosened. **C,** The braided Hedstrom file technique was attempted but was unsuccessful. **D,** A Brasseler Endo Extractor tube is cemented to the cone with cyanoacrylate cement. **E,** The silver point is removed. **F,** Immediate postobturation radiograph. **G,** The 1-year follow-up showing apical healing. (Reprinted with permission from Gutmann JL, Dumsha TC, Lovdahl PE, Hovland EJ, editors: *Problem solving in endodontics,* ed 3, St. Louis, 1997, Mosby, pp 180-81.)

in detail in the following section on separated instrument removal.

After the silver point is removed, it is important that subsequent instrumentation procedures be performed in a crown-down manner to minimize extrusion of the silver corrosion products into the periradicular tissues to decrease the occurrence of painful acute flare-ups. This goal is complicated by the fact that ledges are frequently encountered at the level of the apical extent of the silver point due to the type of milled preparation that was frequently used in this technique. Managing ledges will be discussed in a following section of this chapter.

Occasionally, the apical portion of a silver point will separate upon the removal attempt. If it cannot be bypassed or removed, then the case should be completed and followed carefully (Fig. 8-55). Apical surgery or extraction could be necessary in the future (Fig. 8-56).

Removal of Separated Instruments

Causes of Instrument Separation

Occasionally during nonsurgical root canal therapy, an instrument will separate in a canal system blocking access to the apical canal terminus. This instrument is usually some type of file or reamer but can include Gates-Glidden or Peeso drills, lentulo spiral paste fillers, thermomechanical gutta-percha compactors, or the tips of hand instruments, such as explorers or gutta-percha spreaders. During retreatment, it may be obvious after completing the diagnostic phase that there is a separated instrument in the canal system or it may only become apparent after removal of the root-filling materials (Fig. 8-57). It is useful, therefore, to expose a check radiograph after removal of the root filling to see if there is any metallic obstruction in the canal. Regardless of which type of instruments the clinician uses, whether stainless steel or nickel-titanium, and

FIG. 8-54 A, Brasseler Endo Extractor Kit. B, Masserann Kit. C, Roydent Extractor System. D, Separated Instrument Retrieval System (SIR). E, Instrument Removal System (IRS). (*B*, Courtesy Dr. Daniel Erickson.)

how they are used, by hand or engine driven, the potential for separation exists. The incidence of hand instrument separation has been reported to be 0.25%,[114] and for rotary instruments it ranges from 1.68% to 2.4%.[114,268] The most common causes for file separation are improper use, limitations in physical properties, inadequate access, root canal anatomy, and possibly manufacturing defects.

A common cause for instrument separation is improper use. Included in this category are overuse and not discarding an instrument and replacing it with a new one when needed. The following is a list of guidelines for when to discard and replace instruments[79]:

1. Flaws, such as shiny areas or unwinding, are detected on the flutes.
2. Excessive use has caused instrument bending or crimping (common with smaller-sized instruments). A major concern with nickel-titanium instruments is that they tend to fracture without warning; as a result, constant monitoring of usage is critical.
3. Excessive bending or precurving has been necessary.
4. Accidental bending occurs during file use.
5. The file kinks instead of curving.
6. Corrosion is noted on the instrument.
7. Compacting instruments have defective tips or have been excessively heated.[79]

Another type of improper use is to apply too much apical pressure during instrumentation,[247] especially when using rotary nickel-titanium files. This pressure can lead to deflection of the instrument within the canal system or increased frictional binding against the canal walls that can overstress the metal, resulting in separation. Regardless of which type of files the clinician uses, they should never be used in a dry canal, as attempting to instrument a dry canal will cause excessive frictional stresses on an instrument.[247] Continual lubrication of the canal with either irrigating solutions or lubricants is required,[79] as this will reduce the frictional resistance as well as increase the efficiency of the instrument. All files have flutes that have the ability to build up with dentin shavings, which will decrease the efficiency of the instrument leading to greater frictional forces and ultimately separation. Therefore, files should be periodically removed and cleaned during the instrumentation process.

Inadequate access cavity preparations can lead to many problems, one of which is excessive or unnecessary force

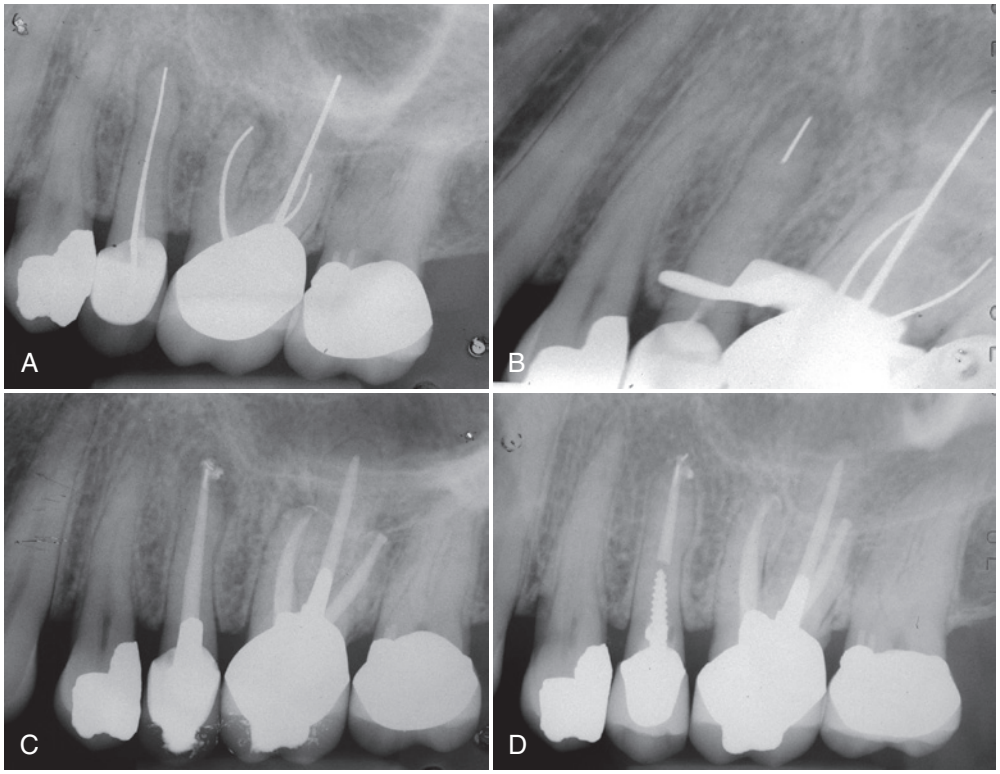

FIG. 8-55 Case illustrating healing despite inability to remove a separated silver point **A**, Preoperative radiograph showing persistent disease in an upper premolar and molar. **B**, Working film showing the separated cone that could not be retrieved despite extensive clinical efforts. **C**, Final obturation after a two-appointment procedure using a calcium hydroxide interappointment medicament. **D**, The 4-year follow-up showing apical healing.

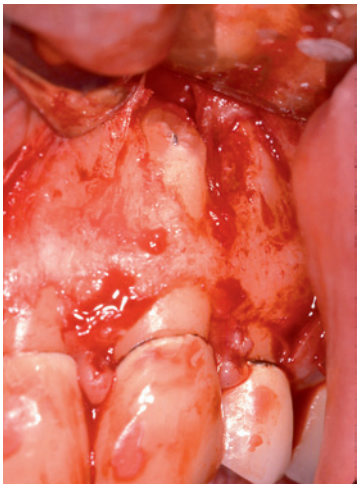

FIG. 8-56 If silver point retreatment is unsuccessful, then apical surgery may be needed. Note the silver point visible on the resected root end. If the point cannot be pulled out in a retrograde direction, then ultrasonic root-end preparation may be complicated by its presence, and root-end preparation using rotary burs may be necessary.

applied to the instrument if it is not allowed to enter the canal freely without interference from the access cavity walls. If the file is in contact with the access cavity wall during instrumentation, the chance for separation is greatly increased. Inadequately enlarged access preparations also increase the number and severity of curvatures that the file must negotiate. This underprepared access can lead to the creation of an iatrogenic "S" curve that can overstress the instrument. This situation is especially hazardous when using rotary instrumentation because traversing an "S" curve greatly stresses the rotating file, leading to separation (Fig. 8-58).

Anatomy, such as abrupt curvatures or anatomic ledges, increases the likelihood of instrument fracture. When the file's progress is hindered, it is natural to try to force it further. This approach will rarely result in the file advancing along the naturally occurring path and indeed may result in file separation, perforation, or ledge creation. Some clinicians would like to blame instrument separation on manufacturing defects; however, this has never been shown to be of clinical relevance and is quite rare.[247]

The best treatment for the separated instrument is prevention. If proper techniques for cleaning and shaping of the root canal system are followed, file separation should be an infrequent occurrence. Nevertheless, an occasional event may take place. When instrument separation occurs, a radiograph should be taken immediately.[247] This radiograph will not only confirm the separation, it will give the clinician information that may aid in removal, such as location, size of the file segment, root canal anatomy, and, ultimately, the possibility of removal. The patient should be advised of the accident as well as its effect on the prognosis.[41] In addition, when a file separates, as with other procedural accidents, detailed documentation is necessary for medical-legal considerations,[247] and the remaining segment of the file should not be discarded but, rather, placed in a coin envelope and kept in the patients record.[41,274]

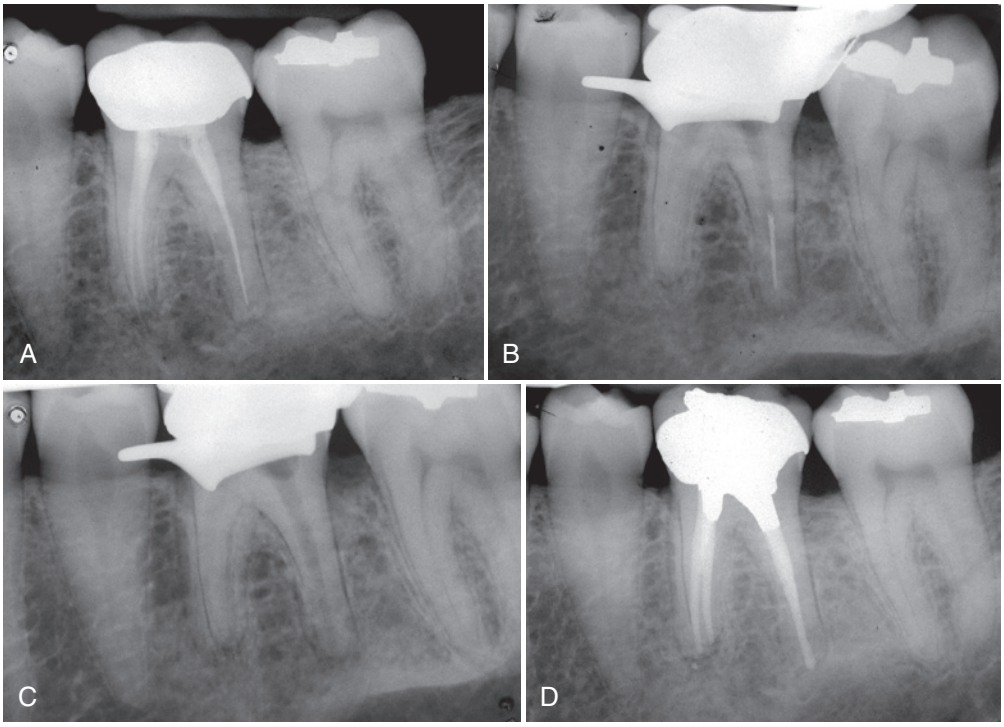

FIG. 8-57 A, Preoperative radiograph of a tooth with symptomatic posttreatment disease. B, Although not readily apparent on the preoperative film, there is a separated nickel-titanium instrument in the distal canal. C, Check film showing that ultrasonics has removed the separated file. D, A 13-month recall film. The patient was asymptomatic.

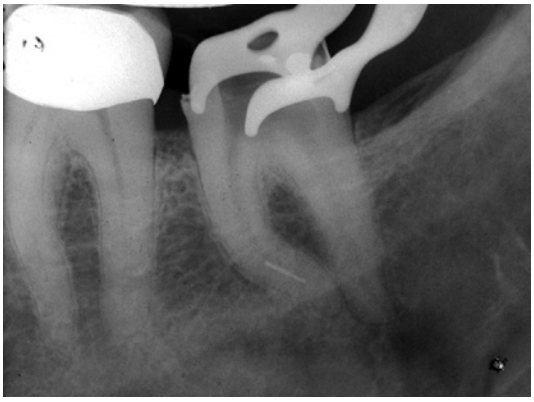

FIG. 8-58 Complicated canal anatomy can increase stress on rotary instruments, leading to separation such as in this "S"-shaped canal.

Prognosis

A separated instrument does not necessarily mean surgery or loss of the tooth. Actually, the prognosis may not be reduced at all depending on what stage of instrumentation the separation occurs, the preoperative status of the pulp and periradicular tissues, and whether or not the file can be removed or bypassed.[225] The presence of a separated instrument in the canal in itself does not predispose the case to posttreatment disease. Rather, it is the presence of any necrotic, infected pulp tissue that remains in the apical canal space that determines the prognosis. The outcome is better if the canal was instrumented to the later stages of preparation when the separation occurs.[247] If the preoperative pulp was vital and noninfected (irreversible pulpitis, for example), and there was no apical periodontitis, the presence of the separated instrument should not affect the prognosis.[43] If the file can be removed without excessive over enlargement of the canal or causing an additional iatrogenic mishap, such as a perforation, the prognosis will not be affected. Bypassing the instrument and incorporating it into the obturation should also have no effect on the prognosis. However, if the instrument cannot be removed or bypassed in a tooth with a necrotic, infected pulp and apical periodontitis, the prognosis will be uncertain. These cases should be followed closely and, if symptoms persist, apical surgery or extraction should be considered.[247,271]

The potential to remove a separated instrument depends on many factors that should be considered during the diagnostic workup. The location of the separated instrument is critical. If the separated instrument extends into the straight, coronal portion of the canal, retrieval is likely. If, however, the instrument has separated deep in the canal and the entire broken segment is apical to the canal curvature, then orthograde removal will not be possible and attempts to do so could lead to a much higher rate of iatrogenic complication.[209,223] If there is persistent disease and the file cannot be bypassed safely, either apical surgery or extraction will be necessary. Because of the need to enlarge the coronal radicular access, root curvatures, external root concavities, and root thickness all will be important factors to consider when deciding which treatment option will provide the best chance of long-term success. Teeth with thin roots and deep external root concavities have a greater likelihood of being perforated during the coronal radicular access, so surgery should be considered as an alternative to orthograde instrument retrieval. The type of material the separated instrument is made of will affect the chances of removal. Nickel-titanium files tend to shatter when ultrasonic

FIG. 8-59 The surgical operating microscope is not only invaluable in helping to remove separated instruments, but it is in fact a necessary tool for these procedures.

energy is applied to them, hindering removal, whereas stainless steel instruments are more robust and more easily removed with ultrasonics.[187]

Removal Techniques

Many different instruments and techniques will be discussed in this section, all of which are important to include in the armamentarium for separated instrument removal. None, however, is more important than the operating microscope (Fig. 8-59). This instrument will not only increase visibility by the use of magnification and light, but it will also increase the efficiency and safety of almost all of the techniques to be discussed. The use of a headlamp and magnifying loupes will help with the removal of many canal impediments. However, the use of the operating microscope has caused a quantum leap in visualization due to the enhanced lighting and magnification that it offers,[124] and many of the techniques to be described should not even be attempted without the use of this valuable tool.[234]

Once the patient has been advised of the treatment options and the decision has been made to attempt removal, the clinician's first choice in treatment will be based on the location of the instrument. If the file is clinically visible in the coronal access and can be grasped with an instrument, such as a hemostat or Stieglitz Pliers (Sullivan-Schein, Port Washington, NY) (see Fig. 8-39), then these should be used to obtain a firm hold of the file and extract it out through the access cavity preparation. Many sizes and angles of forceps are available, and almost all are necessary in order to have the ability to remove obstructions from the many different angles and levels of accessibility presented to the clinician. These will work well if the object is loose fitting within the canal and if the clinician has good access. However, establishing a firm purchase can sometimes be difficult without removing excessive tooth structure. Once a purchase onto the file has been achieved, it is best to pull it from the canal with a slight counterclockwise action. This action will unscrew the flutes that are engaged in the dentin as the file is being removed. This is the easiest technique for removal of a separated file; however, unfortunately, many files separate at a point where these forceps cannot be used.

Frequently, a file will separate at a point deeper in the canal where visibility is difficult. To remove separated root canal instruments predictably, the clinician must create straight-line coronal radicular access. Either removing the crown or creating a large access cavity preparation establishes adequate coronal access to allow the use of the appropriate instruments. Straight-line radicular access can be created with the use of modified Gates-Glidden drills. These drills may be ground down or sectioned with a bur at their maximum cross-sectional diameter. This process will create a circumferential staging platform to facilitate ultrasonic use (Fig. 8-60).[187] One study showed that use of similarly modified Lightspeed nickel-titanium rotary instruments (Lightspeed Technology Inc, San Antonio, TX) created a staging platform that was more centered in curved canals than the Gates-Glidden drills.[115]

Ultrasonic instruments have been shown to be very effective for the removal of canal obstructions.[36,161,187] The ultrasonic tip is placed on the staging platform between the exposed end of the file and the canal wall and is vibrated around the obstruction in a counterclockwise direction that applies an unscrewing force to the file as it is being vibrated. This technique will help with removing instruments that have a clockwise cutting action. If the file had a counterclockwise cutting action (such as the hand GT files), then a clockwise rotation will be needed. The energy applied will aid in loosening the file, and occasionally, the file will appear to jump out of the canal. It is prudent to cover the orifices of the adjacent open canals with cotton or paper points to prevent the removed file fragment from falling into them, causing further case complication[187] (Fig. 8-61). Many sizes and angles of ultrasonic tips are available for this purpose, but in general, the deeper in the canal the obstruction is, the longer and thinner an ultrasonic tip must be. It should be remembered that long, thin tips must be used on very low power settings to prevent tip breakage (Fig. 8-62). Occasionally, if the separated instrument can be bypassed, the use of ultrasonic files can loosen it. Care must be taken, however, to avoid ultrasonic file separation or root perforation.[102] As mentioned previously, nickel-titanium instruments often break into fragments when subject to the energy supplied by an ultrasonic instrument. Clinicians may be tempted to use this information to their advantage by applying the tip of the ultrasonic directly onto nickel-titanium files. Occasionally, this method may work; however, the chance of pushing the separated file further into the canal or beyond the apical foramen may increase the risk of this technique.

If the direct application of ultrasonic energy does not loosen the separated instrument sufficiently to remove it, the fragment must be grabbed and retrieved. This is accomplished with a variety of techniques most using some variant of a microtube. The staging platform is further reduced by ultrasonics until enough of the separated instrument is exposed to retrieve (about 2 to 3 mm).[187] This reduction must be done carefully to avoid root perforation. One relatively simple microtube technique is to use a short piece of stainless-steel tubing that is pushed over the exposed end of the object. A small Hedstrom file is then pushed between the tube and the end of the object using a clockwise turning motion that produces a good mechanical lock between the separated instrument, the tube, and the Hedstrom file. The three connected objects can then be removed by pulling them in a coronal direction[233] (Fig. 8-63).

Another technique is to use a 25-gauge dental injection needle along with a 0.14-mm-diameter steel ligature wire. The needle is cut to remove the beveled end as well as the opposite

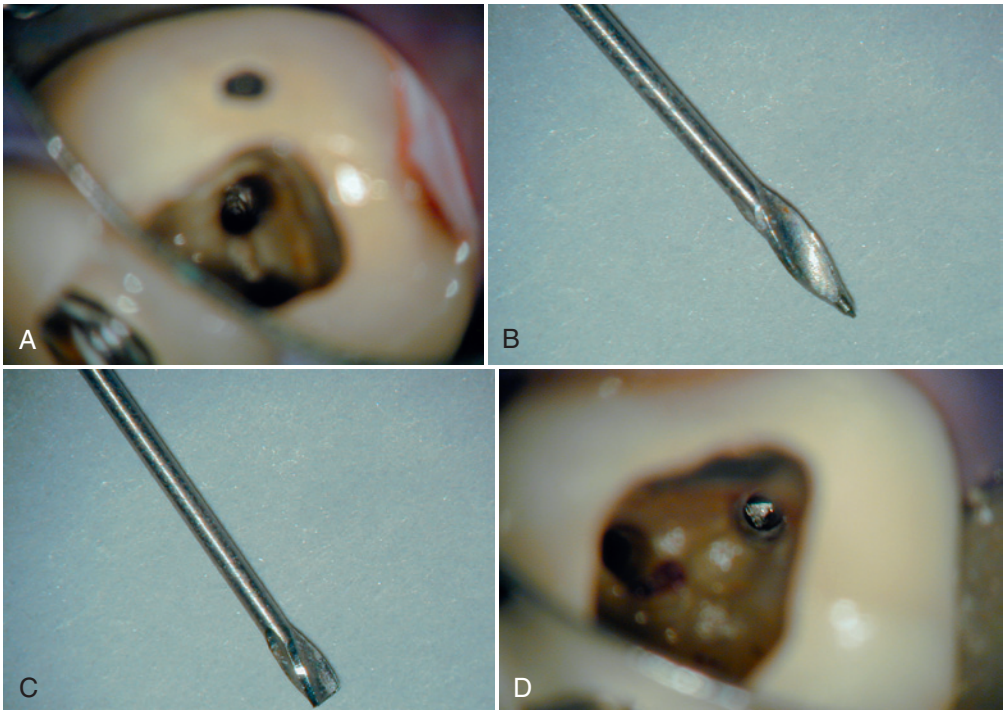

FIG. 8-60 A, Separated instrument in the mesiobuccal canal of a molar. B, Unmodified Gates-Glidden drill. C, Modified instrument. The tip has been ground off to the maximum diameter of the cutting head. D, Staging platform created in the straight coronal section of the canal. Note the enhanced visibility and the triangular cross section of this rotary instrument.

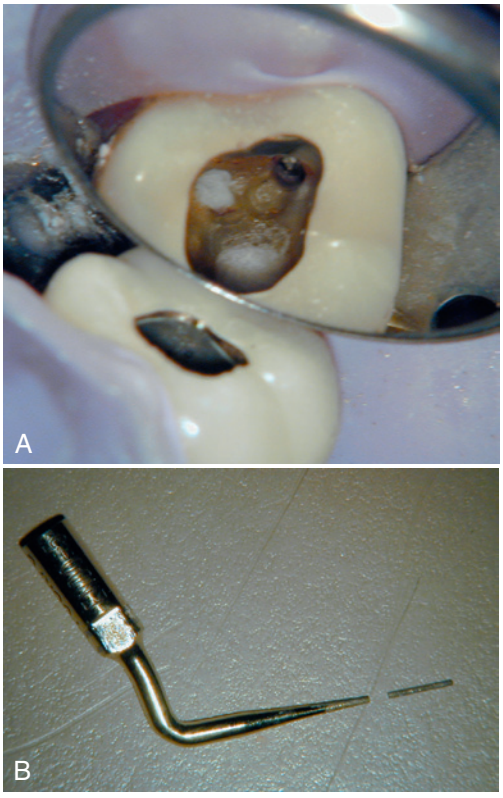

FIG. 8-61 A, Cotton pellets protecting the orifices of the other canals when ultrasonic obstruction removal is needed. B, Ultrasonic tip separated during excavation around separated instrument. If the adjacent canals were not protected, further unnecessary complication could result.

end so it no longer extends beyond the hub. Both ends of the wire are then passed through the needle from the injection end until they slide out of the hub end, creating a wire loop that extends from the injection end of the needle. Once the loop has passed around the object to be retrieved, a small hemostat is used to pull the wire loop up and tighten it around the obstruction and then the complete assembly is withdrawn from the canal.[185] Occasionally, a larger diameter tube and thinner (0.11-mm) ligature wire will facilitate assembly of this extractor (Fig. 8-64).

Another effective technique, especially in cases where access or obtaining an adequate purchase on the file is difficult, is to use an end-cutting trephine bur to remove tooth structure around the file and then use an extraction device to remove it. There are many kits that use these principals with slight variations from one another, including the Endo Extractor (Brasseler USA), the Masserann Kit (Medidenta International), and the Extractor System (Roydent) (see Fig. 8-54).

The Endo Extractor kit includes a cyanoacrylate adhesive, which is used to bond a hollow tube to the exposed end of the file for removal. This kit also includes four sizes of trephine burs and extractors. The most important factor in using this kit is the snugness of fit between the extractor tube and the obstruction. It has been shown that, even with only 1 millimeter of overlap between the extractor tube and the obstruction, if there is a snug fit, the bond created with the cyanoacrylate may be strong enough to remove many obstructions. However, the recommended amount of overlap between the tube and the obstruction is 2 millimeters. The time needed for the adhesive to set to ensure adequate bond strength for removal is 5 minutes for a snug fit and 10

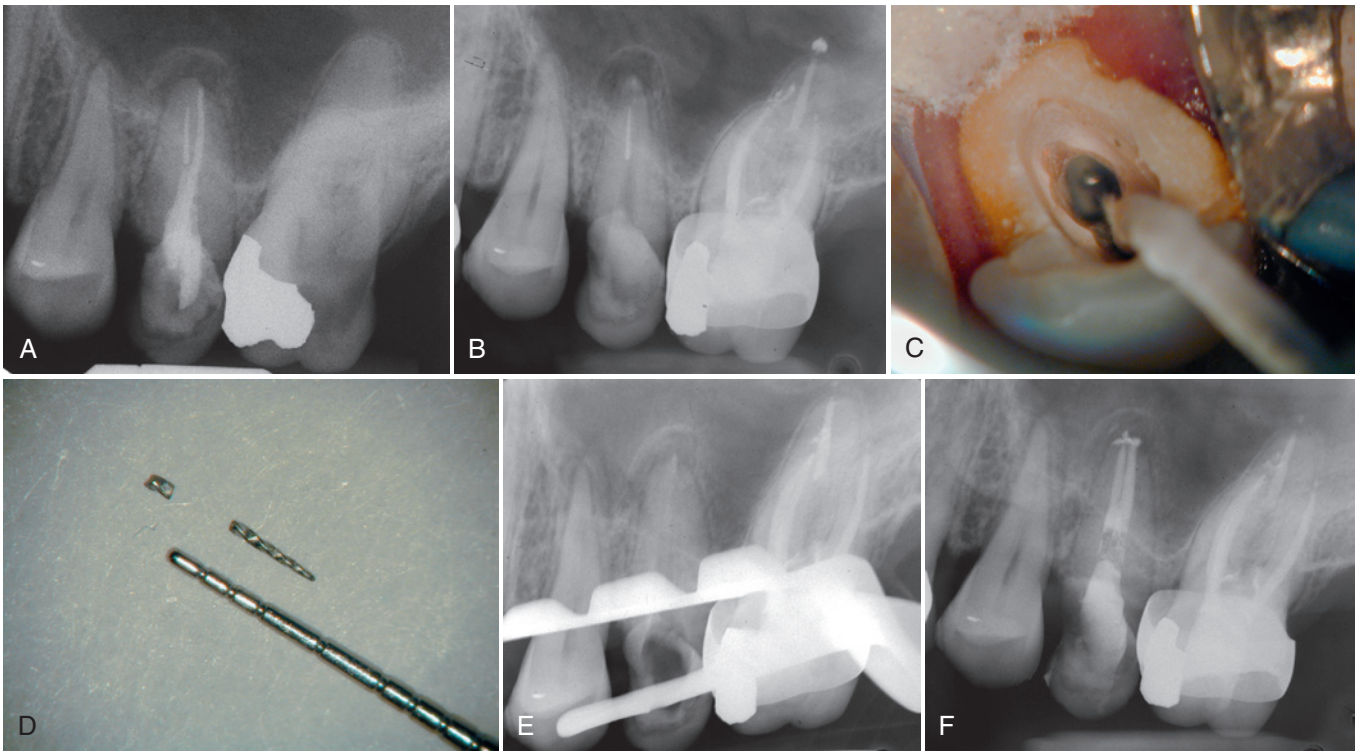

FIG. 8-62 A, Preoperative radiograph showing a separated file in the palatal canal, potential coronal leakage, and apical periodontitis. B, Check film showing the separated instrument after gutta-percha removal. C, Photograph showing the separated instrument in the palatal canal and a paper point in the buccal canal to protect it. D, Separated nickel-titanium file removed. Note that it is in two pieces, a result typical when applying ultrasonic energy to nickel-titanium. E, Check film showing that the file has been completely removed. F, Final canal obturation.

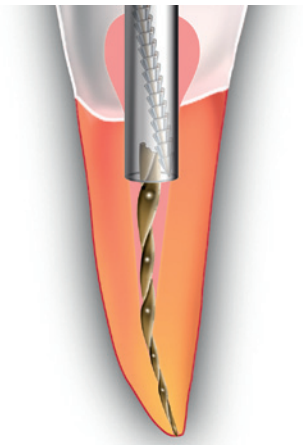

FIG. 8-63 Tube and Hedstrom file removal technique. The tube is slipped over the obstruction and a Hedstrom file is gently screwed into the space between the tube and the obstruction. Pulling the tube and Hedstrom file together can withdraw the obstruction. (Diagram courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, OK.)

minutes for a loose fit.[76,226] One disadvantage of this instrument is that the trephine burs are much larger than their International Standards Organization ISO equivalents, so the manufacturer has also added a separate smaller trephine bur, which is sold separately from the kit. This bur corresponds better with the smaller extractors and removes less dentin that can decrease the likelihood of weakening the root leading to fracture.[61] Another disadvantage is that the burs cut aggressively when new but dull rather quickly. When new, this aggressive cutting may lead to perforation, or even separation of the obstruction. Therefore, great care is needed when using this instrument (Fig. 8-65). Once the separated instrument has been removed, the extractors may be reused, either by using the debonding agent that is included in the kit to remove the embedded instrument from the extractor tube or by simply cutting the extraction device with a bur beyond the extent that the separated instrument has penetrated.

The Masserann technique has also been recommended for the removal of separated instruments.[151] This technique is similar to the Endo Extractor in that it uses trephine burs and a specific extraction device. This kit comes with a convenient gauge that aids in predicting the size of the bur and the extractor to be used, and it contains many sizes of trephine burs. In addition, the trephine burs with this kit cut in a counterclockwise direction that provides an unscrewing force on separated files. The extraction mandrels have an internal stylus that wedges the file against the internal wall of the mandrel allowing the obstruction to be removed. Although effective, this technique may require removal of an excessive amount of radicular dentin,[69] leading to root weakening and the risk of perforation[272]; therefore, this instrument must be used with caution.

The Extractor System from Roydent comes with only one bur and three extraction devices. The bur is very conservative

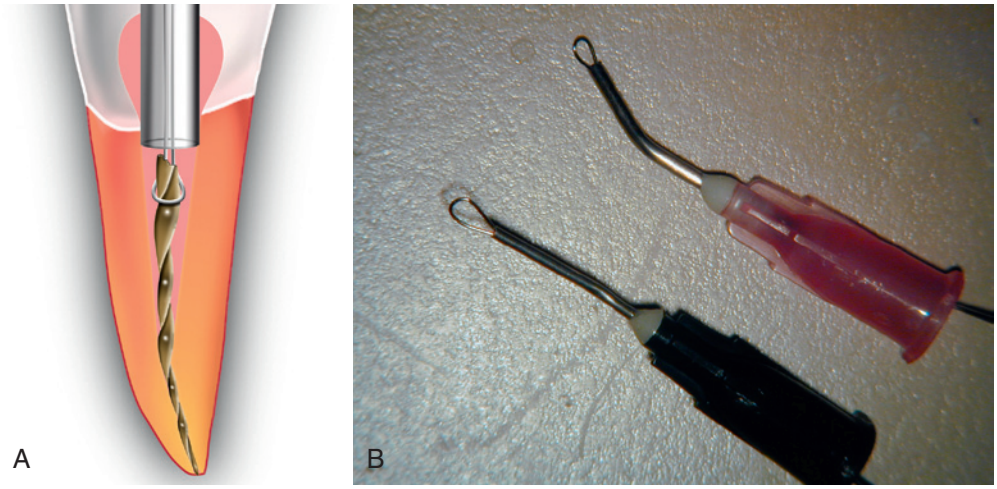

FIG. 8-64 A, Diagram illustrating the wire loop and tube method of obstruction removal The wire loop is carefully placed around the obstruction, tightened, and then removed. B, Larger diameter tubes and smaller diameter (0.11-mm) ligature wire enhances the efficiency of this technique. (*A,* Diagram courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, OK.)

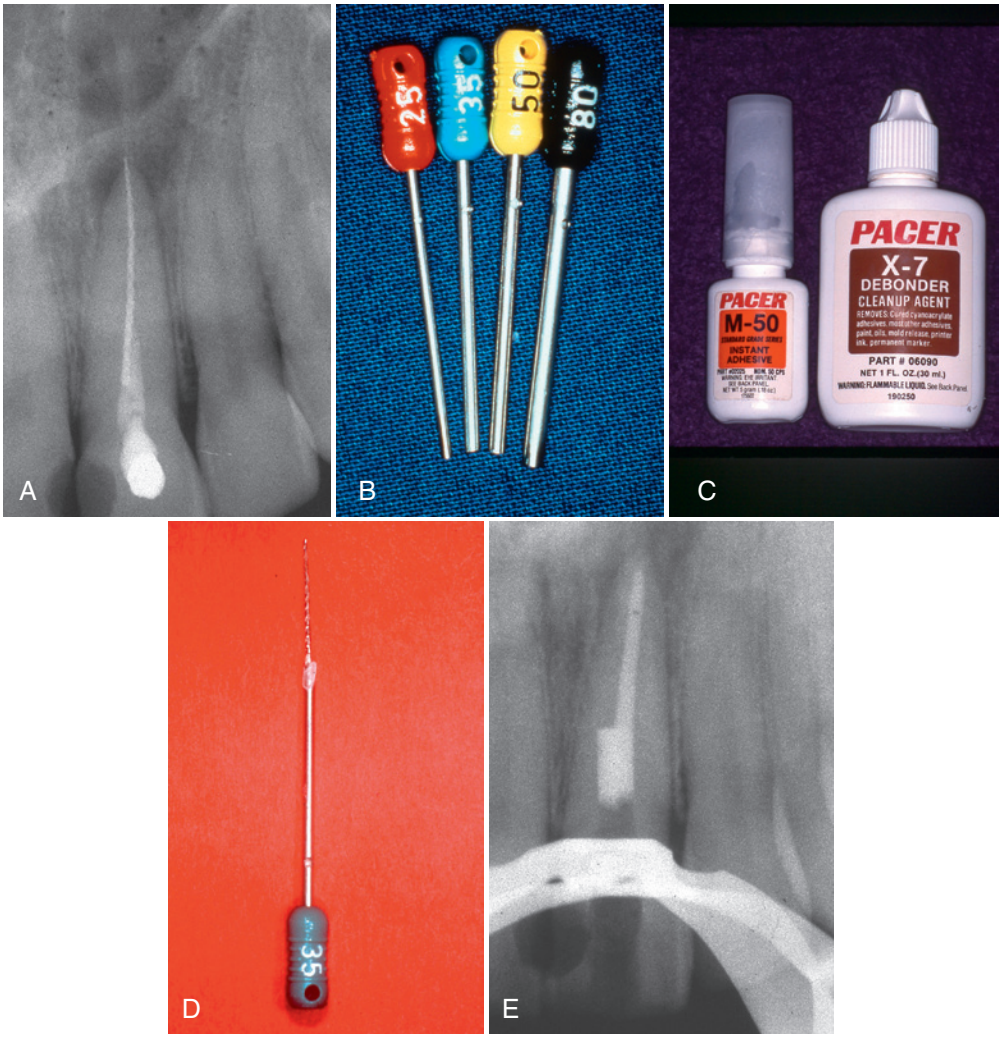

FIG. 8-65 A, Separated file wedged into an upper incisor. B, Brasseler Endo Extractor tubes. C, Cyanoacrylate cement and debonding agent. D, Separated file pulled out by the bonded tube. E, Final obturation. Note the excessive amount of tooth structure removal by the trephine bur that was needed to bond the tube.

and removes a minimal amount of tooth structure, enabling access to the obstruction. The extractor tubes are also quite small and, therefore, will only work for the removal of small obstructions. The extractor surrounds the obstruction with six prongs that can be tightened onto the object, enabling removal. This works in the same way a drill chuck tightens onto a drill bit (Fig. 8-66). The disadvantages of this kit are the lack of variety of instruments, the possibility of separating the obstruction with the bur, and the potential problem of breakage of the prongs in the extractor if they are submitted to bending rather than applying strict tensile force during removal.

Two techniques have been designed specifically for removing instruments in conjunction with the operating microscope:

FIG. 8-66 Close-up view of the Roydent Extractor tip. The tip is placed over the separated instrument and tightened to grasp the obstruction.

the Cancellier instrument and the Mounce extractor (Sybron-Endo) (Fig. 8-67). The Cancellier instrument works in a similar manner to the Brasseler Endo Extractor in that it is used in conjunction with cyanoacrylate adhesive to bond onto the separated end of the instrument. Unlike the Brasseler extractors, the Cancellier extractors are attached to a handle that enables them to be used without blocking visibility when using the operating microscope. The Brasseler extractors are finger instruments that interfere with the line of sight the microscope requires. There are no trephine burs in the Cancellier kit; rather, it is used in conjunction with ultrasonic exposure of the separated instrument. Four sizes of extractor tubes are available, each of which corresponds to specific file sizes. The Mounce extractors are also hand instruments that enable use with the operating microscope. These instruments are similar to a ball burnisher with slots cut into the ball end. These slots are cut at various angles and are designed to slide onto the broken end of the file. Cyanoacrylate is used to bond the extractor to the file allowing removal. This instrument can be used when the separated file is lying against the canal wall; however, the ball tip is relatively large and is only useful in retrieving instruments that are in the most accessible coronal portion of the canal.

Another device designed specifically for the purpose of separated file removal is the Instrument Removal System (DENTSPLY Tulsa Dental Specialties) (see Fig. 8-54). This kit consists of two different sizes of extraction devices that are tubes with a 45-degree bevel on the end and a side cutout window. Each tube has a corresponding internal stylus or screw wedge. Prior to use of this instrument, 2 to 3 mm of the obstruction is exposed by troughing around it with an ultrasonic instrument. Once the file is exposed, the appropriate size microtube is selected and slid into place over the obstruction. Once in place, the screw wedge is turned counterclockwise to engage and displace the head of the obstruction through the

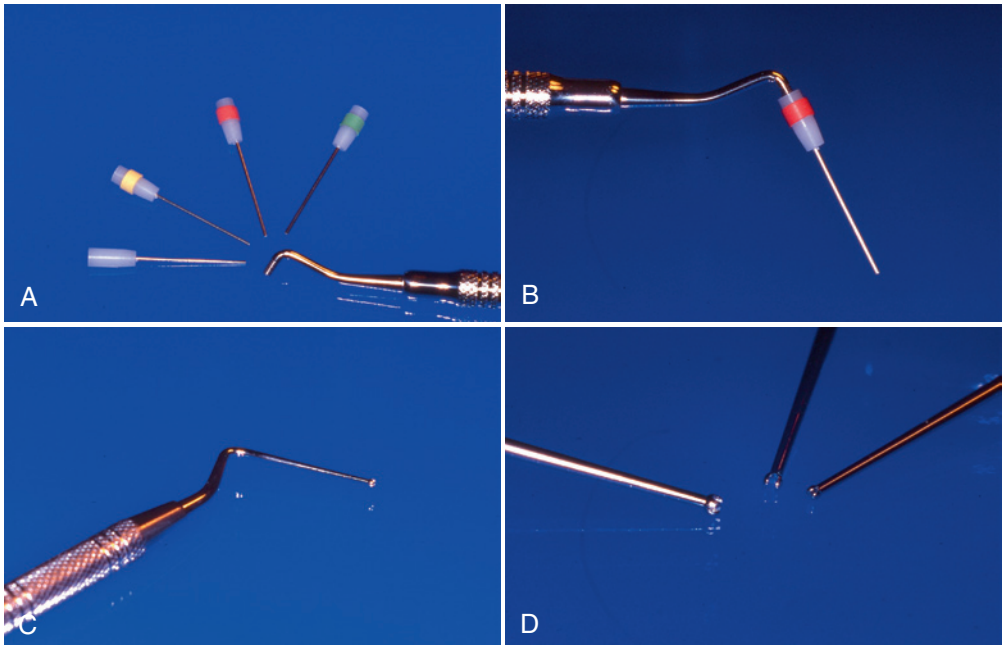

FIG. 8-67 A, The Cancellier Kit with four tube sizes available. B, The Cancellier instrument is used with super glue to bond the obstruction but its design allows for greater visibility during use. C, The Mounce instrument. D, Varying tip sizes for the Mounce instrument.

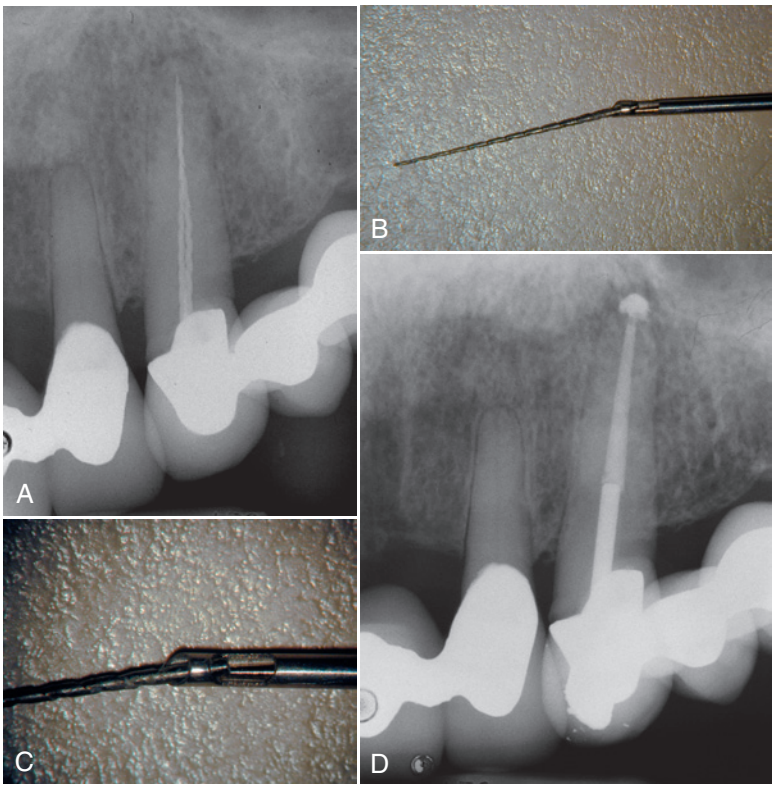

FIG. 8-68 A, Preoperative radiograph showing two separated instruments in one tooth. B, IRS instrument with a removed file. C, Note that large size files are difficult to push through the cutout window. D, Postoperative radiograph.

side window. The assembly is then removed.[187] This instrument is useful in the straight portion of the canal, but it is difficult to force large-diameter separated files through the cutout window, hampering their removal (Fig. 8-68).

The S.I.R. (Separated Instrument Retrieval) System (Vista Dental Products) (see Fig. 8-54) is another microtube method of separated instrument retrieval. Like the Cancellier instrument, it utilizes extractor tubes bonded onto an obstruction, enabling removal. Once the obstruction is exposed using ultrasonics or the trephine burs from one of the other kits described, the bendable dead-soft tubes are bonded onto it. Once the adhesive is set, the obstruction is removed through the access cavity preparation. Included in this kit are the necessary bonding agent, a bottle of accelerator, five different sizes of tubes, assorted fulcrum props, and a hemostat. The accelerator causes the bonding agent to set almost instantaneously. The ability to bend these tubes allows for access in most areas of the mouth. A hemostat allows the clinician to establish a firm purchase onto the tube, creating the ability to lever the bonded obstruction out of the canal. A vinyl, autoclavable instrument prop provides protection for the next most anterior tooth, which is to be used as a fulcrum; however, if the clinician has access to grasp the extractor with his or her fingers to remove the extractor/obstruction unit, the hemostat may not be necessary.

Heat Generation During Retreatment Procedures

There are many procedures in endodontic therapy that can generate heat, but perhaps the area with the greatest risk of heat related tissue damage occurs during nonsurgical retreatment. Use of heat to soften canal filling materials to aid in their removal[132,139] and use of ultrasonics to dislodge posts[192] and separated instruments[95,146] can potentially generate enough heat to raise the temperature of the external root surface by 10° C or more. Temperature elevations of the periodontal ligament in excess of 10° C can cause damage to the attachment apparatus.[11,53,194,195]

The greatest danger of heat-related damage occurs with the use of ultrasonic energy to dislodge foreign objects in the canal space in order to gain access to the apical portion. As described earlier, this has recently become an extremely important and useful part of the clinician's armamentarium due to the ability of ultrasonic energy application to conserve tooth structure while removing the obstruction. These instruments have allowed clinicians to perform predictable retreatment when surgery would have been previously indicated. As with most instruments used in dentistry, however, these devices must be used with caution as in vitro research and clinical case reports imply that they have the potential to damage the tooth attachment apparatus due to the heat generated during use.[30,78,201] One in vitro study has showed that ultrasonic vibration for post removal without coolant can cause root surface temperature increases approaching 10° C in as little as 15 seconds.[48] Thermal damage to the periradicular tissues may be so serious as to result in both tooth loss and permanent bone loss (see Fig. 8-28). This is not to say that ultrasound energy should be avoided for the removal of canal obstructions because it is many times the only way to reach the apical area of the canal.

The factors that may contribute to a heat-induced injury are the length of the post, post diameter, post material, and type

of luting cement. Studies need to be done to establish heat reduction protocols based on these variables. Some have proposed that the dentin thickness between the outer surface of the post and the root surface may affect root surface temperature rise,[81,82] and one study showed this.[192] However, a more recent study has shown that the dentin thickness is statistically insignificant as a factor in root surface temperature rise.[101] One possible mitigating factor would be the effect of the periradicular blood supply, which could act as a heat sink dissipating the generated thermal energy and thus helping to prevent injury. This may be why the effect of seemingly similar conditions of ultrasonic application can have such differing results on different patients. Clearly, more in vivo research is needed in this area.

It has become accepted that the heat-induced damage to periradicular tissues during the usage of ultrasound energy for post removal is time dependent.[30,106] Studies have advised cooling of the ultrasonic tips can greatly reduce heat buildup[30,106] despite reducing the efficiency of debonding resin bonded posts.[48,73] The amount of time the clinician can use these instruments safely is difficult to determine, as specific protocols that are evidence based using in vivo research have yet to be established. Therefore, the authors feel any specific recommendations regarding rest intervals between usage of ultrasound energy, ways of monitoring heat buildup, or duration of consistent activation cannot be made based on the available research at the time of publication. However, the authors do feel strongly with regard to several recommendations for the use of ultrasound energy during the removal of canal obstructions, and they are as follows:

- Use ultrasonic tips with water ports whenever possible.
- If your ultrasound device does not have tips with water ports, have your assistant use a continuous coolant air/water spray during usage.[55]
- Take frequent breaks to let the tooth cool down.
- Avoid using the ultrasound on the high power setting.[55]

Prudent clinicians must take extreme care when applying ultrasound energy to a canal obstruction, as it has been shown that even with the use of water coolant, the temperature of root surfaces can increase rapidly.[48,192] Thus, until evidence-based heat reduction protocols are developed, caution is required when using instruments that can generate heat in the periodontal ligament.

Management of Canal Impediments

Following removal of all root-filling materials, further progress to the apical constriction may be prevented by the presence of a block or a ledge in the apical portion of the canal. Most of these impediments are iatrogenic mishaps resulting from vigorous instrumentation short of the appropriate working length and failure to confirm apical patency regularly during instrumentation. A blocked canal contains residual pulp tissue (sometimes necrotic, often fibrosed or calcified) and packed dentinal "mud" in the apical several millimeters of the canal system.[187] This debris is frequently infected, resulting in persistent disease, and must be removed if possible. A ledge is the result of placing non-precurved, end-cutting instruments into curved canals and filing with too much apical pressure.[79,116] It is a type of canal transportation that results in a canal irregularity on the outside of the canal curvature that is difficult or impossible to bypass. The canal space apical to the ledge is not thoroughly cleaned and sealed, so ledges frequently result in posttreatment disease. The best treatment for blocked and ledged canals, as with all iatrogenic problems, is prevention. If the clinician is careful and attentive during the instrumentation process, the chance for an impediment to develop is minimized. When the clinician becomes careless or hurried, problems occur. The strategies for preventing blocks and ledges are found in Chapter 19.

During the treatment-planning phase, blocks and ledges may be detectible on radiographs as a root filling short of the ideal working length, and the patient should be warned that they might prove impenetrable and require future apical surgery or extraction.[68] This should not deter the clinician from choosing nonsurgical retreatment, however. In one study, 74% of teeth showing short root fillings were successfully negotiated to adequate length, with the authors stating that presence of a short fill should not be considered a technical contraindication to retreatment.[57] The clinical encounter usually occurs after removal of the previous root-filling material when apical advancement of small files is impeded. At this point, the clinician may be unaware of which type of impediment exists, but a common approach to management is helpful in the early stages of the process. The coronal portion of the canal should be enlarged to enhance tactile sensation and remove cervical and middle third obstructions in the canal space. The canal should be flooded with irrigant, and instrumentation to the level of the impediment should be accomplished using non-end-cutting rotary files, such as the Lightspeed (Lightspeed Endodontics, San Antonio, TX), the Profile or GT instruments (Dentsply, York, PA), or the K-3 instrument (Sybron Endodontics, Orange, CA), in a crown-down manner. This procedure will enlarge and flare the canal space coronal to the impediment while minimizing the likelihood of worsening any ledge present.

At this point, the impediment should be gently probed with a precurved #8 or #10 file to determine if there are any "sticky" spots that could be the entrance to a blocked canal. A directional rubber stop should be used so that the clinician knows in which direction the tip of the instrument is pointing, which helps in visualizing the three-dimensional layout of the canal system. Frequently, evacuating the irrigant and using a lubricant, such as RC Prep (Premier, Plymouth Meeting, PA) or Pro-Lube (Dentsply), will enhance the ability to place the small file into the apical canal segment. If repeated, gentle apical pressure or "pecking" of the hand file against the blockage results in some resistance when withdrawing the instrument on the outstroke ("stickiness"), then the clinician should continue to peck at the "sticky" spot until further apical advancement is accomplished.[187] This is frequently a slow and tedious process. The endeavor can be made more efficient by using precurved stiff files such as the C+ file (Maillefer, Baillagues, Switzerland), but there is a risk of deviating from the original canal path, creating a ledge, and ultimately a false canal leading to zip perforation.[79] It is prudent to make a working radiograph when some apical progress has been made to confirm the placement of the instrument into the suspected apical extent of the canal. The clinician should resist the urge to rotate the file excessively. If the tip of a small instrument is tightly bound in the blocked segment of the canal and the tip has been worked by pecking, it is prone to fracture in the apical area further complicating the case.[187] The separated file tip is frequently irretrievable, and surgery or extraction may be the result. Dropping down to the next smaller size file and using a gentle reciprocal rotational motion ("twiddling") will aid in

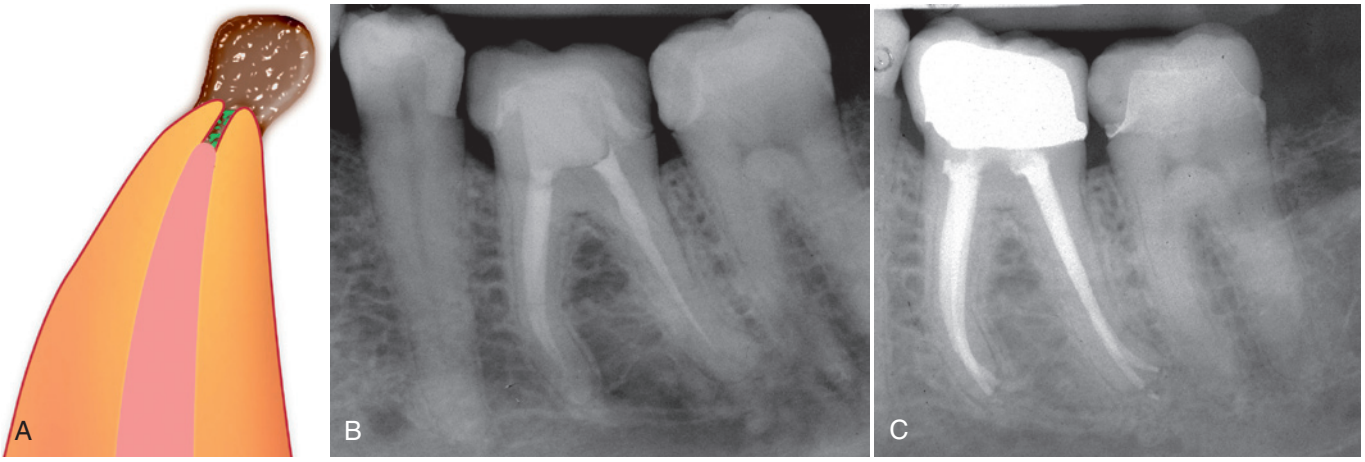

FIG. 8-69 A, Diagrammatic representation of a canal block. Fibrotic or calcified pulp and debris that is potentially infected remains in the apical segment of the canal when the canal is instrumented short of the apical constriction. B, Preoperative radiograph showing obturation short of ideal length. The patient was symptomatic and the canals were blocked. C, The 3-month posttreatment. Treatment took a total of 3.5 hours over three appointments due to the time-consuming nature of bypassing blocked canals. (*A,* Diagram courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, OK.)

advancement through the blocked canal. Frequently, as apical progress is being made, the clinician will be using an electronic apex locator to gauge the proximity of the apical constriction. Unfortunately, the apex locator is sometimes not able to give an accurate reading in a blocked canal, and because of the continued "sticky" feel that occurs even as the instrument bypasses the foramen and penetrates apical tissues, an overextension may result. To prevent this complication with its attendant risk of a painful postoperative flare-up, when the estimated working length is reached, a working length radiograph is necessary.[149] Once apical working length is achieved, apical patency should be confirmed, and gentle, short amplitude 1- to 2-mm push pull strokes should be made until the file can pass freely to the apical constriction (Fig. 8-69).

If, after a reasonable amount of time, no sticky spot can be found, the clinician must consider the possible presence of a ledge despite possibly not detecting it on the preoperative radiograph. The main problem with ledges is that instruments will invariably find their way to the ledge while finding the original canal is many times impossible. They feel like a hard brick wall, short of length when encountered, and care must be used to prevent worsening the ledge by indiscriminately burrowing into it.[255] To manage a ledge, the tip of a small #08 or #10 file has a small bend placed in it 1 to 2 mm from the end,[116,247] so the tip of the file forms an approximately 45-degree angle with the shaft of the instrument. The directional stop is oriented to the bend, and the file is carefully negotiated to the level of the ledge. Because ledges form mainly on the outside of curvatures, the directional stop (and thus the bent tip of the file) is turned in the direction of the suspected apical curvature away from the ledge (Fig. 8-70). The file tip is slowly scraped along the internal wall of the canal curve slightly coronal to the level of the ledge[247] in an effort to find another sticky spot. This spot will be the entrance to the apical canal segment, and gentle reciprocal rotation will usually allow the file to be negotiated to the canal terminus. Confirm with a radiograph. Once the ledge has been bypassed, short amplitude push-pull and rotational forces keeping the file tip apical to the ledge will be needed to clean and enlarge the apical canal space. When the

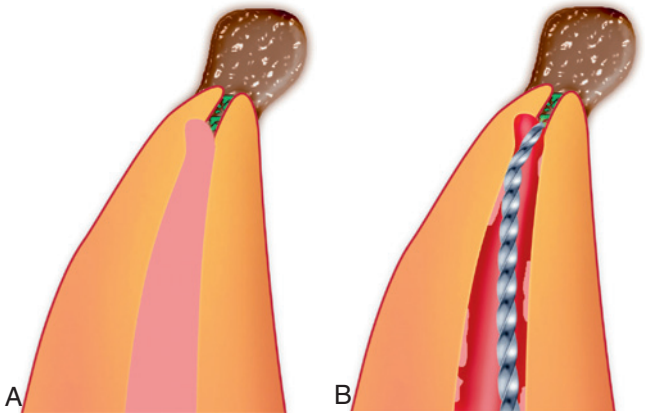

FIG. 8-70 A, Diagrammatic representation of a ledged canal. Potentially infected debris remaining in the apical segment can result in posttreatment disease. B, Attempting to bypass the ledge with a small file having a 45-degree bend in the tip. Note that the opening to the apical canal segment is on the inside of the canal curvature and coronal to the level of the ledge. (Diagrams courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, OK.)

file can be easily negotiated around the ledge, anticurvature filing will enable the clinician to blend the ledge into the canal preparation (Fig. 8-71). Many times this cannot be completely accomplished,[255] but as long as the apical segment can be cleaned and obturated, the prognosis should not be adversely affected.

The use of Greater Taper (GT) NiTi hand files (DENTSPLY Tulsa Dental Specialties) for the blending of ledges has been advocated.[187] The advantage these instruments have is that they are non-end cutting, and their rate of taper is two to six times that of conventional 0.02 tapered files, so they can do the work of multiple 0.02 tapered hand files. Once the ledge has been bypassed and the canal can be negotiated with a conventional size #15 or 20 K-file, a GT hand file is selected. The K-file creates a pilot hole so that the tip of the GT file can passively follow this glide path beyond the ledge. The GT file must have

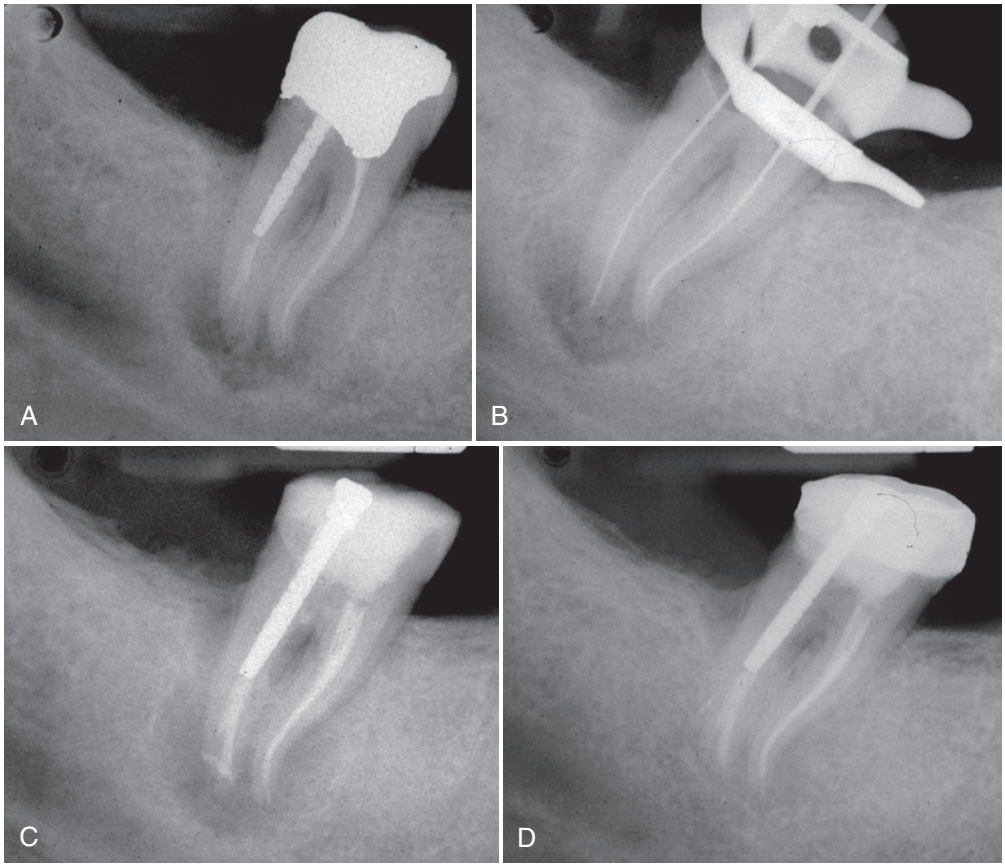

FIG. 8-71 **A,** Preoperative radiograph showing a distal canal ledge with a small amount of sealer that has entered the apical segment. The ledge prevented proper cleaning and sealing of the canal system resulting in posttreatment disease. **B,** The ledge has been bypassed. The attempt is made to blend the ledge into the contour of the prepared canal wall. **C,** Final obturation showing the filled ledge and apical segment. **D,** A 13-month recall showing healing. The patient was then directed to have a definitive coronal restoration placed.

a tip diameter of 0.2 mm (#20) and a taper that will vary depending on the requirements of the preparation. The largest taper that will enter the apical segment is used; however, these instruments must be precurved, which presents a challenge because they are made from nickel-titanium alloy. To precurve this superelastic shape memory alloy, a file-bending tool, such as the Endo Bender Pliers (SybronEndo), is needed. The pliers grasp the tip of the instrument, and the file is overcurved between 180 and 270 degrees to plastically deform the alloy. At this time, the appropriate tapered GT file is then carried into the canal, and the rubber stop is oriented so that the instruments precurved, working end can bypass and move apical to the ledge. The GT file is then worked to length, and the ledge is either reduced or eliminated (Fig. 8-72).

If the canal blockage or ledge cannot be negotiated, then the canal space coronal to the impediment should be cleaned, shaped, obturated, and coronally sealed. The patient must be informed of this complication, the guarded prognosis, and the need for regular reevaluation (Fig. 8-73). If symptoms of posttreatment disease arise subsequently, apical surgery or extraction will be needed.[187,247]

Finishing the Retreatment

After regaining the apical extent of the canal system, routine endodontic procedures are instituted to complete the retreatment. Any missed canals must be found using magnification, micro-excavation techniques and, most important, the knowledge of canal anatomy that is discussed in another section of this text (Fig. 8-74). One cannot find a canal unless one suspects it is there. Cleaning and shaping procedures must focus on a crown-down approach to minimize extrusion of irritants into the periradicular tissues and also must emphasize enlargement of the apical portion of the preparation to ensure complete removal of apical debris. These aims are best accomplished using technique hybridization during instrumentation and keeping the goals of the retreatment procedure in mind. These matters are covered in detail in Chapter 6. Canal disinfection procedures are, however, paramount after the completion of cleaning and shaping. Because the primary cause of posttreatment disease is usually microbial[64,163] and these microbes (i.e., *Enterococcus faecalis*) are frequently resistant to traditional canal disinfection regimens,[16] every effort must be made to eliminate these organisms from the canal system. This effort is complicated by the fact that no instrumentation regimen can predictably remove the entire previous root filling from the canal space after retreatment.[12] This leaves areas where microbes can reside underneath fragments of root-filling materials and remain protected from standard antimicrobial canal irrigants such, as sodium hypochlorite. Whether the canal space can be adequately disinfected when completing treatment in one visit or whether an interappointment medicament such as calcium hydroxide is needed is still a matter of debate, and the reader

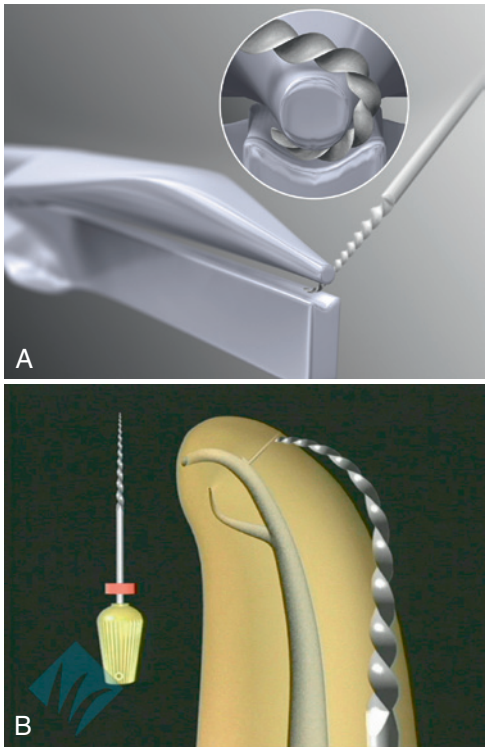

FIG. 8-72 A, Endobender Pliers (SybronEndo) used to overbend a nickel-titanium Hand GT file. B, GT hand file can hold a bend to allow it to bypass ledges. (Images courtesy Dr. Steve Buchanan.)

is directed to the appropriate chapter of this text for details of the problem. It is important to keep in mind, however, that teeth that require retreatment also require the highest level of disinfection possible to ensure the most favorable outcome.

REPAIR OF PERFORATIONS

Occasionally posttreatment endodontic disease will be the result of root perforation.[112] Root perforations are created pathologically by resorption and caries, and iatrogenically during root canal therapy (zip, strip, and furcation perforations) or its aftermath (e.g., post preparation perforation)[184] (Fig. 8-75). When they are present, perforations may usually be found during the diagnostic phase as areas where the root-filling materials or possibly restorative materials such as posts are found to radiographically leave the confines of the presumed canal space and approach or cross the radiographic interface between the dentin and the periodontal ligament. Angled radiographs are of paramount importance in determining whether a perforation exists and locating which surface or surfaces of the root have been perforated. This information is necessary when deciding upon treatment options. Frequently, cervical and occasionally midroot perforations are associated with epithelial downgrowth and subsequent periodontal defects, so thorough periodontal assessment is required[148,206] (Fig. 8-76). If there were no evidence of posttreatment disease associated with the defect or tooth, then no treatment would be indicated. If, however, there is evidence of periradicular periodontitis, repair may be instituted in one of two ways,

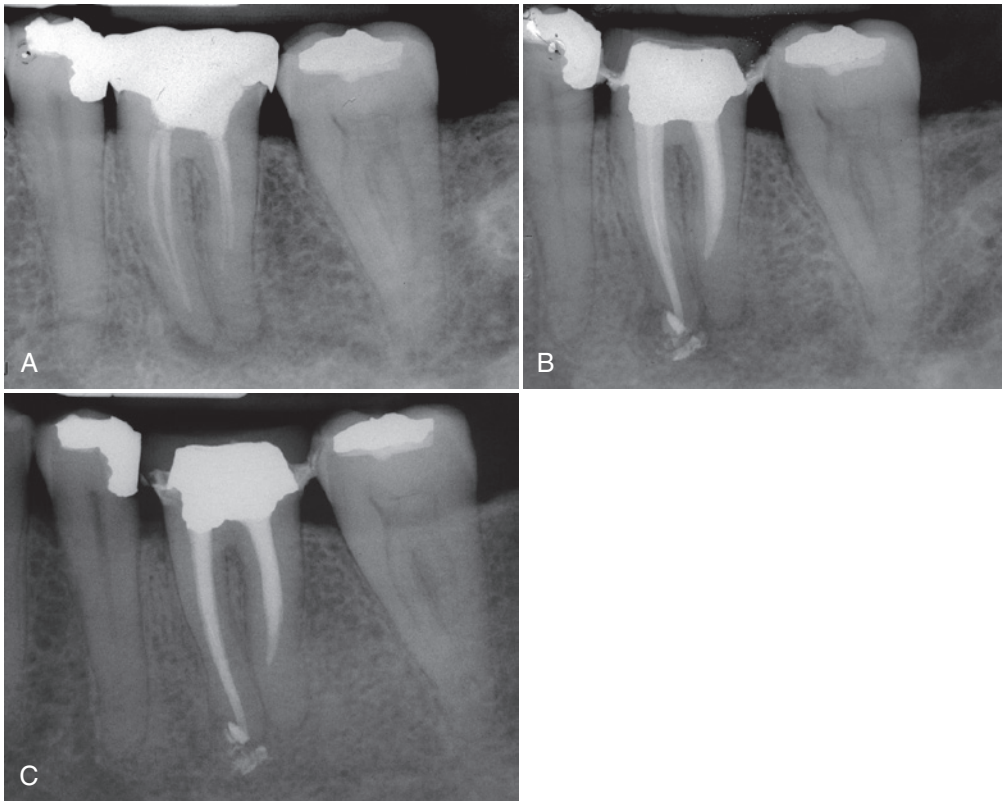

FIG. 8-73 A, Preoperative radiograph showing mesial canal blockages and potential distal root ledging with accompanying posttreatment disease. B, Final film showing mesial blockages bypassed but inability to negotiate beyond the distal ledge. The patient elected to pursue no further treatment at this time. C, One-year recall. Despite not achieving all of the aims of conventional endodontic therapy, periradicular healing is apparent. The patient is asymptomatic and will now begin the final restoration with the knowledge that apical surgery may be needed in the future.

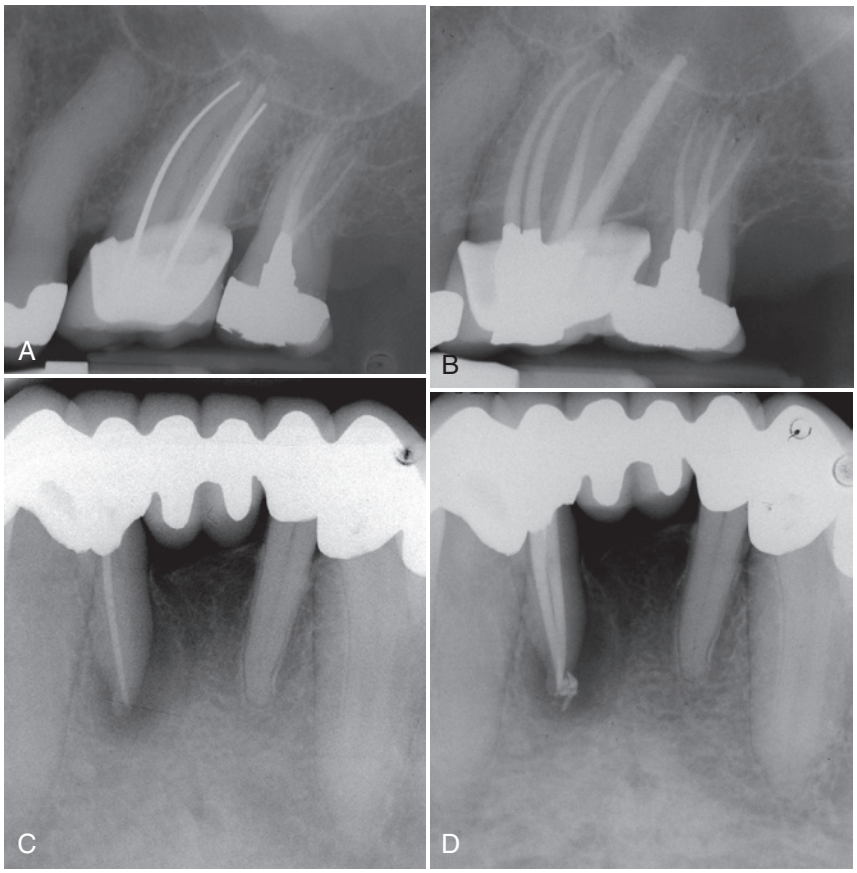

FIG. 8-74 Frequently missed canals that may result in posttreatment disease **A**, Missed second mesiobuccal canal (MB2) in an upper molar. **B**, Final obturation showing cleaned, shaped, and filled MB2 canal. **C**, Missed lingual canal on a lower incisor results in posttreatment disease. **D**, Immediate postoperative film showing management of the missed canal.

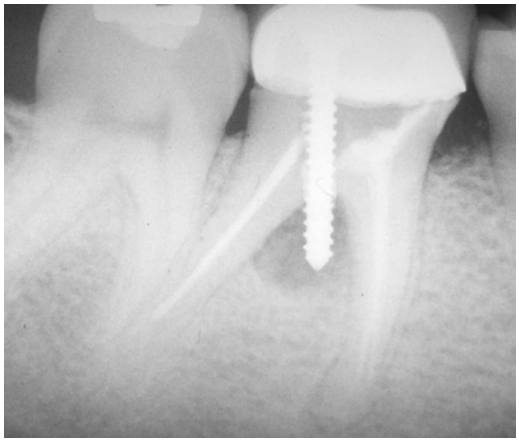

FIG. 8-75 Furcal post perforation resulting in persistent infection and furcal bone loss.

either nonsurgically by approaching the defect internally through the tooth or surgically by using an external approach through the periradicular tissues.[184] In general, if all other factors are considered equal, internal nonsurgical perforation repair will be the preferred method, as it is usually less invasive, produces less destruction of periradicular tissues via the surgical access wound needed, and usually enhances isolation from microbes and disinfection. If, however, the defect is readily accessible surgically and disassembly of the existing restorations would impose an unacceptably high cost and long treatment time to the patient, surgical repair should be selected. If a longstanding defect has a periodontal lesion that has formed around it, surgery perhaps with guided tissue regeneration will usually be needed. However, in most of these cases, nonsurgical retreatment and internal perforation repair prior to surgery will be beneficial to the treatment outcome. A multidisciplinary approach will be required, usually in consultation with the restorative dentist, a periodontist, and perhaps an orthodontist.[187,260]

Factors that affect the prognosis of perforation repair include location of perforation, time delay before perforation repair, ability to seal the defect, and previous contamination with microorganisms.[129,206,213] In general, the more apical the perforation site, the more favorable is the prognosis; however, the converse is true for the repair procedure itself. The difficulty of the repair will be determined by the level at which the perforation occurred. If the defect is in the furcal floor of a multirooted tooth or in the coronal one third of a straight canal (access perforation), it is considered to be easily accessible. If it is in the middle one third of the canal (strip or post perforations), difficulty increases, and in the apical one third of the canal (instrumentation errors), predictable repair is most challenging and, frequently, apical surgery will be needed.

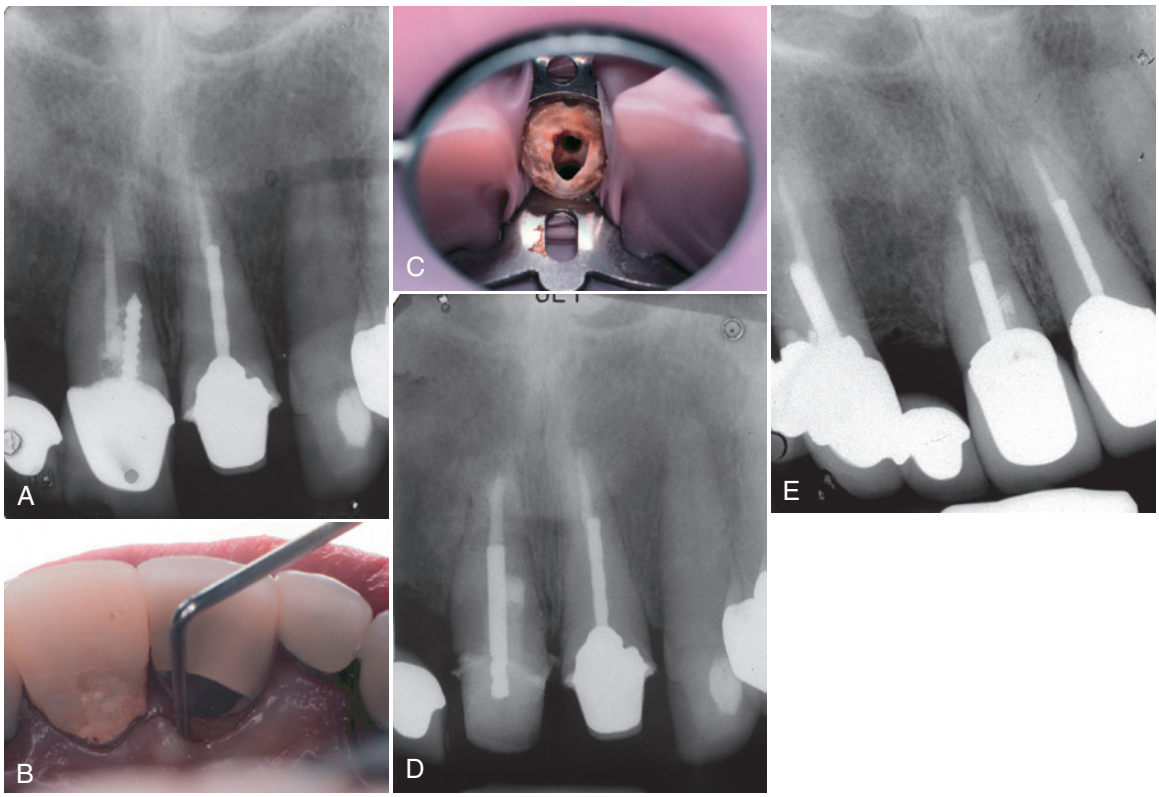

FIG. 8-76 A, Mesially angulated preoperative radiograph showing a palatally oriented post perforation in an upper incisor. B, An 8-mm narrow-based probing defect on the mesiopalatal corner of the tooth. C, Following coronal disassembly, the true canal can be seen lying in a facial direction relative to the palatal post preparation. D, The perforation was repaired with an external matrix of Colla-Cote and MTA. Subsequently, in conjunction with a periodontist, periodontal flap surgery was used to remove periodontal disease etiology from the longstanding pocket, and guided tissue regeneration procedures were instituted. E, The 3-year reevaluation. The tooth is asymptomatic, and the mesiopalatal probing depth is 4 mm.

Immediate repair is better than delayed repair, as delay can cause breakdown of the periodontium, resulting in endo-perio lesions that are difficult to manage,[118,212] and elimination of microbial contamination of the defect and sealing it properly is critical to success. Many materials have been advocated for the repair of perforations in the past; however, none provided predictable healing after treatment. Commonly used materials include amalgam, Super EBA cement (Bosworth, Skokie, IL), various bonded composite materials, and, more recently, mineral trioxide aggregate (Pro-Root MTA, DENTSPLY Tulsa Dental Specialties)[187] (Fig. 8-77).

Since the recent introduction of mineral trioxide aggregate for perforation repair, the choice of which repair material to use is more clear.[148,184] MTA has many advantages over other restoratives when being used for perforation repair. This material seals well,[167,270] even when the cavity preparation is contaminated with blood.[243] It is very biocompatible,[171,179,244,246] rarely eliciting any response from the periradicular tissues, and a cementum-like material has been consistently shown to grow directly on the material after placement.[100,179] MTA has also been shown to have a high degree of clinically favorable long-term outcomes when used as a perforation repair material.[148,184] The main disadvantage of MTA is the long time required for setting,[245] which makes this material inappropriate for transgingival defects such as those associated with cervical

FIG. 8-77 ProRoot MTA is a medical grade of Portland cement that has had the arsenic removed so that it can be used in the human body. It is the material of choice for endodontic perforation repair. (Courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, OK.)

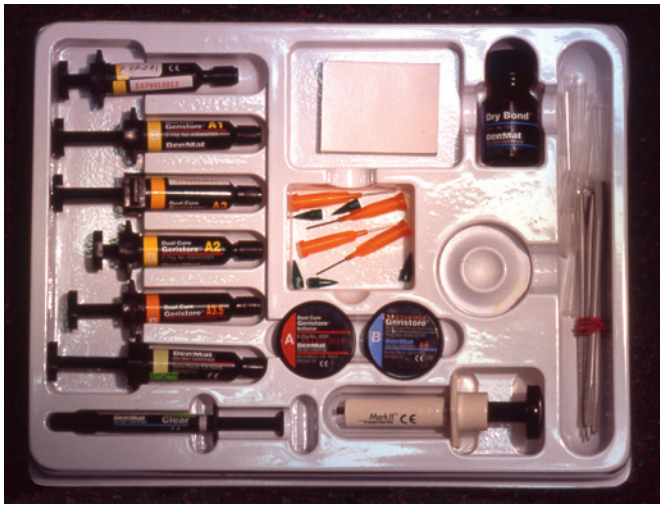

FIG. 8-78 Geristore Kit. This and other resin-ionomers have been advocated for cervical perforation repair due to good biocompatibility and the shorter more controlled setting times that make them useful for transgingival root fillings.

resorption. If the material is in contact with oral fluids, it will wash out of the defect prior to setting, so a more rapid setting resin-ionomer such as Geristore (Den-Mat, Lompoc, CA) is recommended for lesions that cross the gingival margin[18,49,197] (Fig. 8-78). MTA is available in the original gray-colored formulation and a newer, more aesthetic off-white color for treatments in the aesthetic zone of the mouth, although there is little research on the differences between the two formulations. Their sealing ability seems comparable,[60] but questions remain as to whether white MTA exhibits the same biocompatibility[177] and will have the same long-term success as the older variety.

If the perforation is to be repaired nonsurgically through the tooth, coronal-radicular access to the defect is prepared as stated previously (Fig. 8-79). First, the root canals are located and preliminarily instrumented to create enough coronal shape to allow them to be protected from blockage by the repair material. The defect is cleaned and sometimes enlarged with the use of ultrasonics or appropriate rotary drills such as the Gates-Glidden to remove any potentially contaminated dentin surrounding the perforation. Use of a disinfectant irrigating solution such as sodium hypochlorite should be considered if the perforation is not so large as to allow the irrigant to significantly damage the periradicular tissues. If the perforation is large, then sterile saline should be used as an irrigant, and disinfection of the margins of the defect is performed using mechanical dentin removal. Arens and Torabinejad have advocated the use of copious flushing of the defect with 2.5% hypochlorite,[10] but in light of the potential severe complications of hypochlorite overextension through a perforation,[74] extreme care should be used. After the defect has been cleaned, vigorous bleeding may result. Hemostasis should be undertaken using collagen (Colla-Cote, Integra Life Sciences, Plainsboro, NJ) (Fig. 8-79, *B*), calcium sulfate (Capset, Lifecore Biomedical, Chaska, MN), or calcium hydroxide[187]; however, astringents such as ferric sulfate should be avoided, as the coagulum they leave behind may promote bacterial growth and may compromise the seal of the repair.[134]

When the bleeding has been controlled, some easily removable material should be placed over the entrances to the deeper portion of the canals to prevent the repair material from blocking reaccess to the apical terminus. The canals may be protected with cotton, gutta-percha cones, paper points, or shredded collagen. The use of severed files is not recommended, as removal of the files after placement of the repair material is difficult because the material tends to lock into the instrument's flutes (Fig. 8-79, *F*). After protecting the canals, the perforation site is inspected to determine if an external matrix is needed to ensure a proper contour of the restoration.[133] If the surrounding bone is closely adapted to the defect margins, minimal to no matrix material will be necessary; however, if the perforation is associated with a large osseous defect, this must be filled with an external matrix to minimize overcontouring of the repair restoration. The matrix material should be a biocompatible, usually absorbable material such as collagen, freeze-dried demineralized bone allograft (FDDB), hydroxyapatite, Gelfoam, or calcium sulfate.[184,187] Care must be exercised so that the external matrix material is not condensed so forcefully that it damages adjacent vital structures, such as the mental nerve or the floor of the sinus.

Following the preparation of the defect, the repair material is placed. It may be carried in a small syringe or amalgam carrier, and it is condensed with pluggers or microspatulas. In the case of MTA in an accessible defect, the butt end of paper points make an excellent condenser because they can wick some of the water out of the material giving it a firmer consistency, aiding condensation. When the MTA has been positioned, a moist cotton pellet is placed over it to hydrate the material and the tooth is sealed to allow the MTA to set. Upon reentry, the material should be set hard and well retained in the perforation site[217] (see Fig. 8-79, *F*). If there is an overextension of the material beyond the normal external root contour, it seems not to affect the prognosis of the repair.[10,184]

If the perforation is deeper in the canal, the objectives and principles of repair as just outlined all apply, except that access to the defect is more complicated (Fig. 8-80). Protecting the canal from blockage is somewhat more difficult, and placement of the repair material requires the enhanced vision that is provided by the surgical operating microscope. Ideally, the canal should be fully shaped prior to the repair attempt,[187] and a canal patency protector should be placed apical to the defect. In some of these instances, the canal can be protected by using a severed file, notwithstanding the previous warning, because not only can it protect the canal from blockage, but it can also be used as an indirect carrier for transmitting ultrasonic energy to the MTA, causing it to "slump" into the defect when direct condensation is impossible. The file is placed into the canal to a level well below the defect, and the MTA is carried to place. Once condensation has been performed as well as possible, the coronal extent of the file is touched with an ultrasonic tip to vibrate the MTA into the defect. After this is accomplished, the file must be vigorously instrumented in a short 1- to 2-mm amplitude push-pull motion to free it from the placed MTA so that it can be easily removed after the material is set[187] (Fig. 8-81). There is some evidence that ultrasonic placement of MTA may enhance the seal against bacteria in an apexification model,[130] although other researchers have not agreed with this conclusion and found poorer canal wall adaptation when filling the apical extent of canals using ultrasonic condensation compared with hand filling.[8] Further study of ultrasonic MTA placement is warranted, but clinical observation suggests that this method has merit.

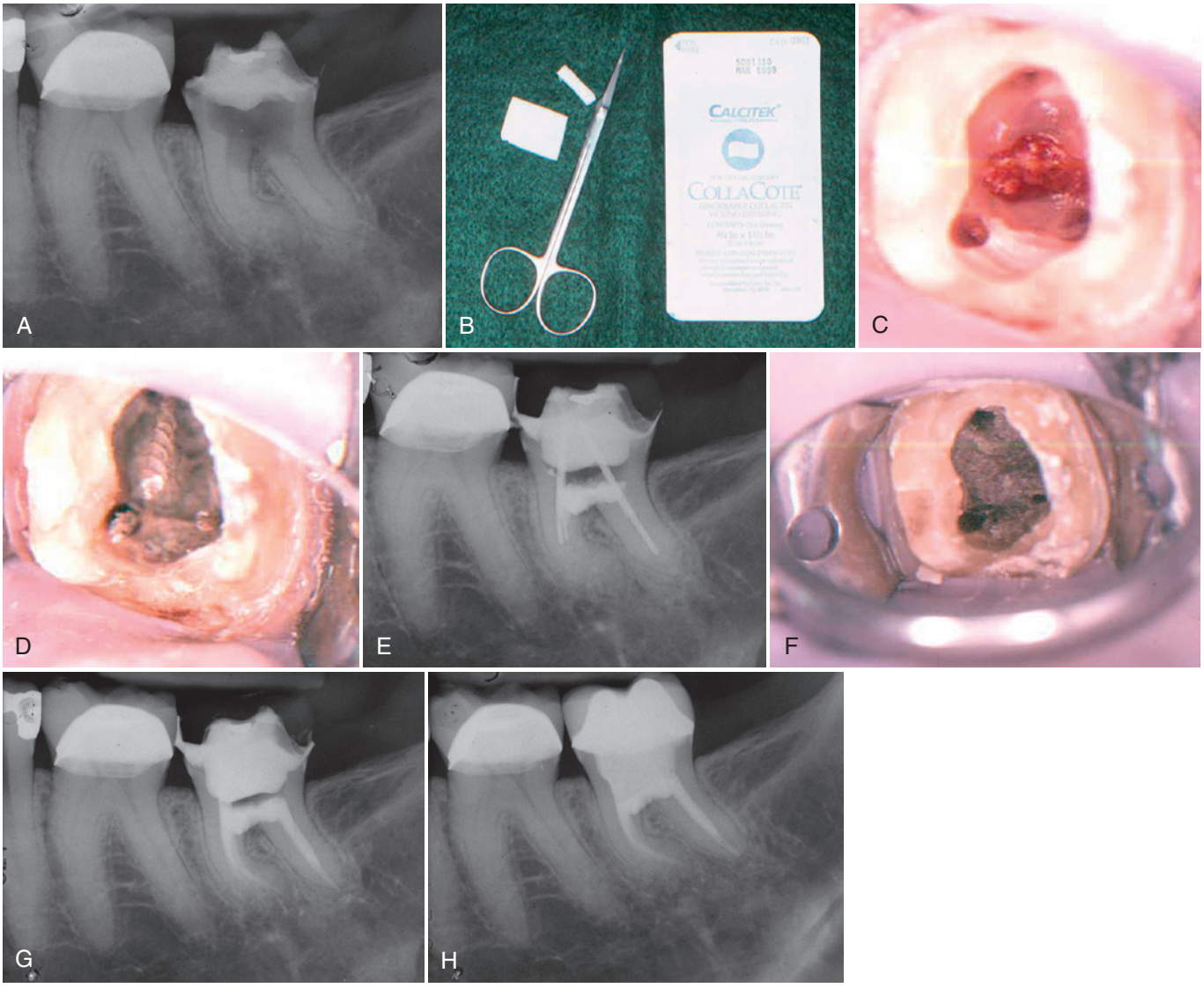

FIG. 8-79 A, Large furcal perforation created during an attempt at endodontic access. B, Colla-Cote (Integra Life Sciences, Plainsboro, NJ) to be used as an external matrix material to recreate the external root contour. C, Canals have been found, preliminarily instrumented, and the external matrix has been placed. D, Canals are protected from being blocked using large endodontic files cut off above the orifice. MTA has been placed into the defect. E, Radiograph showing the initial repair with MTA recreating the vault of the furcation. F, On the second appointment, the blocking files are removed with difficulty because the MTA has flowed into the flutes and set. At this time, endodontic therapy is completed as normal. G, Postobturation radiograph. Note the radiolucency in the furcal vault, which represents the Colla-Cote external matrix material. H, The 19-month reevaluation. The patient is asymptomatic, and there is evidence of healing in the furcal vault area.

If the perforation is in the apical portion of the canal, it is usually due to a procedural accident during instrumentation of a curved canal and is invariably accompanied by a block or ledge. This type of perforation is the most difficult to repair, because repair not only involves cleaning and sealing the defect but also finding, cleaning, and filling the apical canal segment. All of the aforementioned techniques for managing blocks and ledges are required to find and clean the apical canal segment. When this has been accomplished, the decision is made as to whether the canal should be filled with MTA or with gutta-percha and sealer. The MTA is undoubtedly more effective in sealing the canal (especially if it cannot be dried) and is much more biocompatible, but carrying it predictably to the apical extent of a curved canal is problematic. If a holding file is placed in the apical canal segment to anticipate eventual gutta-percha placement after the MTA repair material is set, then the presence of the file precludes consistent extension of the MTA into the apical end of the defect, even when ultrasonically vibrated. If a holding file is not placed, the MTA will also flow into the prepared apical segment, which may not be completely three-dimensionally obturated. In general, whichever choice is made, the outcome will be unpredictable, so the patient must be advised that regular reevaluation is necessary and apical surgery or extraction may ultimately be needed.

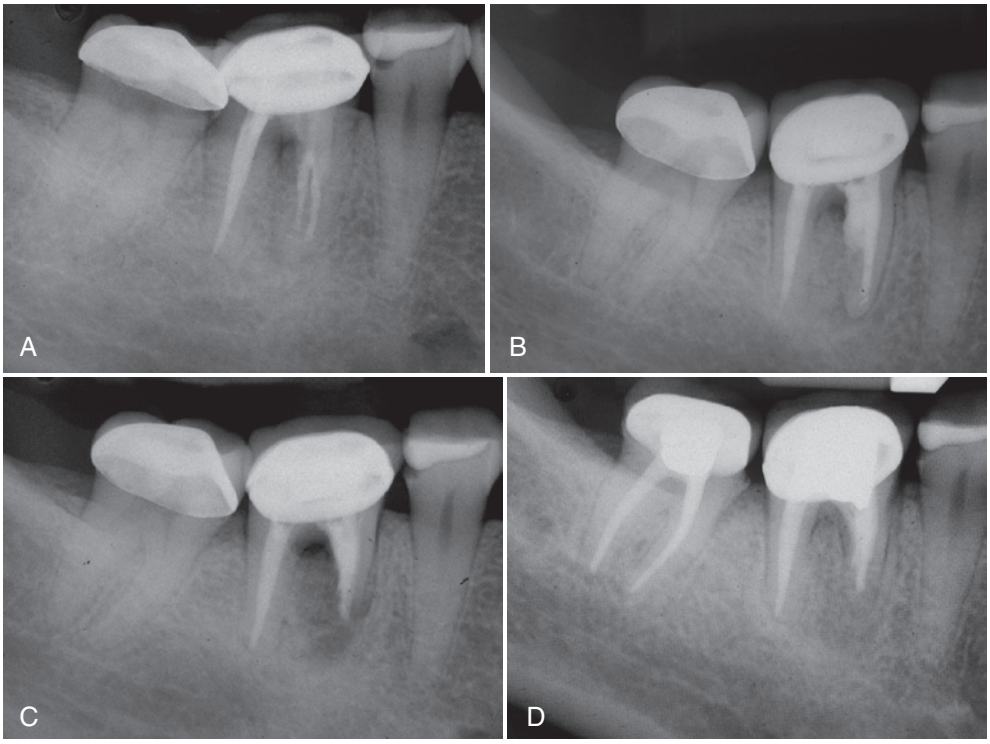

FIG. 8-80 Midroot level perforation repair. **A,** Preoperative film showing mesial strip perforation with bone loss. **B,** Nonsurgical internal repair with inability to negotiate canals to the apical terminus and MTA overextension into the furca. **C,** Apical and perforation repair surgery performed. **D,** he 1-year follow-up showing complete healing.

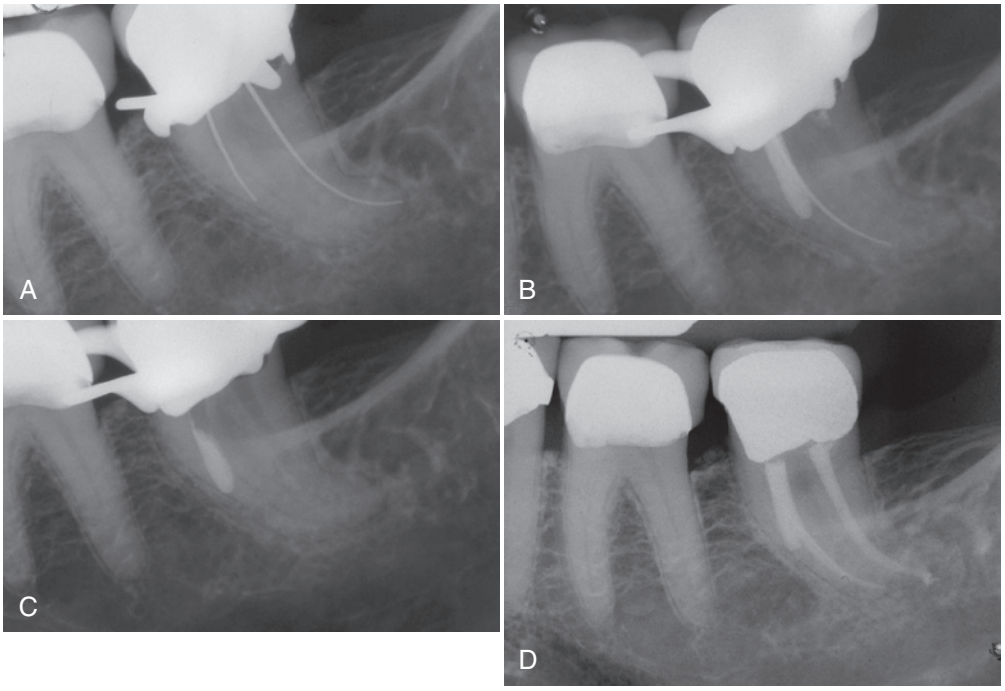

FIG. 8-81 **A,** This patient was in extreme pain following initial endodontic instrumentation by her dentist. Midroot level perforation found on access. **B,** The original canal was found and protected with an endodontic file. MTA was vibrated using ultrasonic energy applied to the file, and it flowed into the defect. Then the file was moved in a push-pull manner to dislodge it from the MTA prior to closure. Note that the defect was intentionally enlarged to allow for more predictable application of the MTA. **C,** On the second appointment, the file was withdrawn easily because it was detached from the MTA repair material. The endodontic therapy could then be concluded as normal. The patient has been asymptomatic since the end of the first appointment. **D,** The 27-month follow-up showing complete healing.

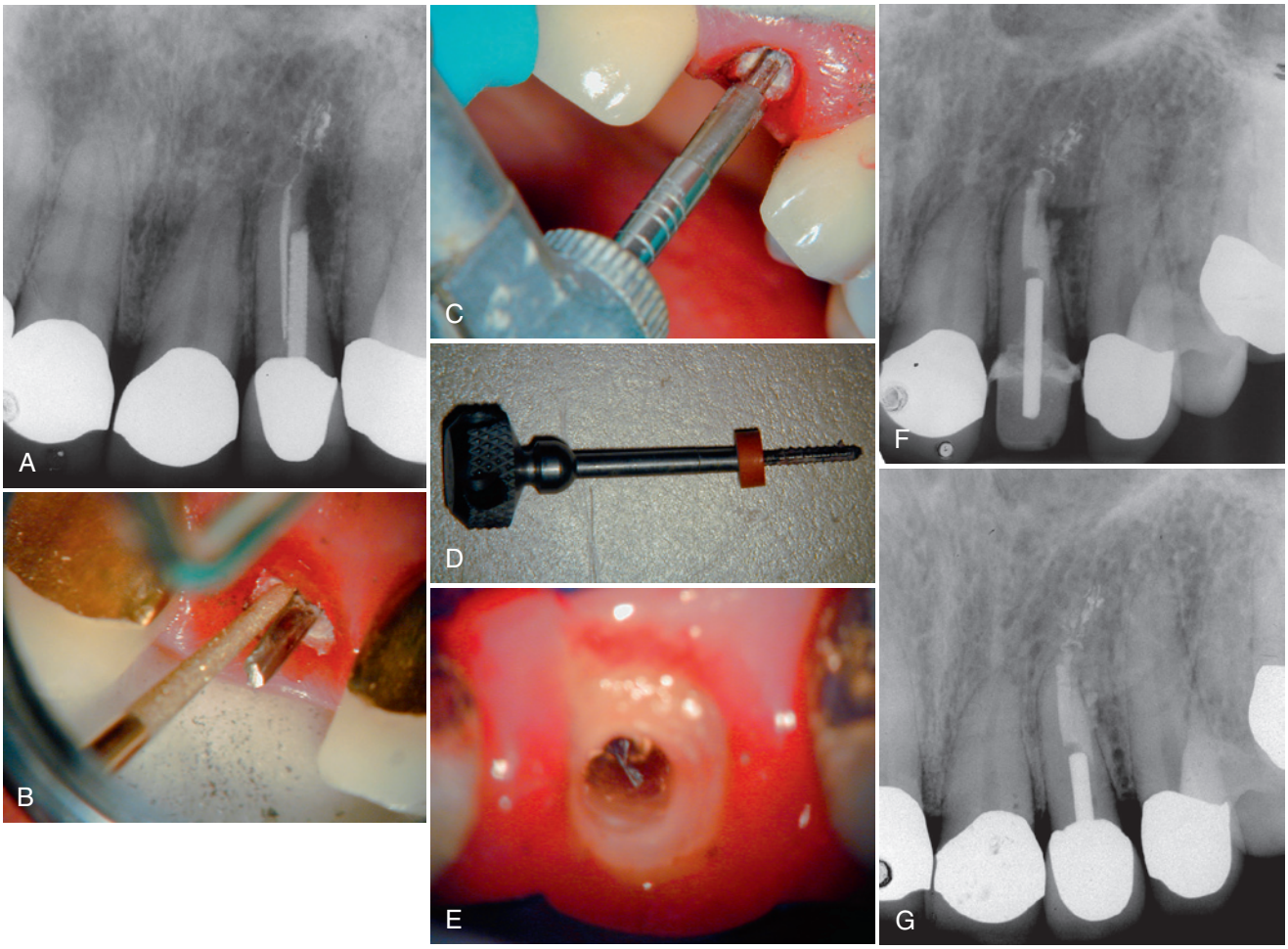

FIG. 8-82 A, Preoperative radiograph showing mid-root level post perforation and associated periradicular periodontitis. B, The crown was removed and ultrasonic energy was applied to the post. C, Using the trephine bur from the Ruddle Post Removal kit to mill the endo of the post. D, The screw post is removed using the wrench from the Ruddle kit. E, After removal of the post and gutta-percha, a plastic solid core carrier is found in the canal and removed using the techniques described in this chapter. F, Postoperative radiograph showing MTA perforation repair, canal seal with gutta-percha, and post and core fabrication. G, The13-month recall showing healing around the perforation repair site.

PROGNOSIS OF RETREATMENT

When the proper diagnosis has been made and all of the technical aspects of retreatment are carefully performed, orthograde retreatment can be highly successful (Fig. 8-82). The prognosis depends to a large extent on whether apical periodontitis exists prior to retreatment.[169] In a systematic review of outcomes studies,[66] Friedman and Mor reported that in the absence of prior apical periodontitis, the incidence of healed cases after both initial treatment and orthograde retreatment ranges from 92% to 98% up to 10 years after treatment. When prior apical periodontitis is present, the incidence of healing drops to 74% to 86% regardless of whether initial treatment or orthograde retreatment was performed. The authors stated that this "similar potential to heal after initial treatment and orthograde retreatment challenges the historic perception of the latter having a poorer prognosis than the former."[66]

Unfortunately, these numbers mean that the desired outcome will not occur in potentially one quarter of retreatment cases. Many techniques and devices for endodontic retreatment have been mentioned here to aid the clinician. However, none of this will guarantee success. Even when strict endodontic principles and fundamentals are followed, the result may be persistent posttreatment disease. When healing does not occur, the clinician is faced with the decision of what to do next. The choice is between four treatment options: observation, endodontic surgery, extraction-replantation, or extraction.

Many times a tooth that has persistent apical periodontitis may remain in asymptomatic function for an extended period of time, a state that has been referred to as functional retention of the tooth.[66] If the patient's goal of treatment is not necessarily complete healing of the tooth, but simply to retain it in function and without pain, then regular evaluation by the clinician is warranted. If signs and symptoms of worsening infection such as progressive enlargement of a periapical

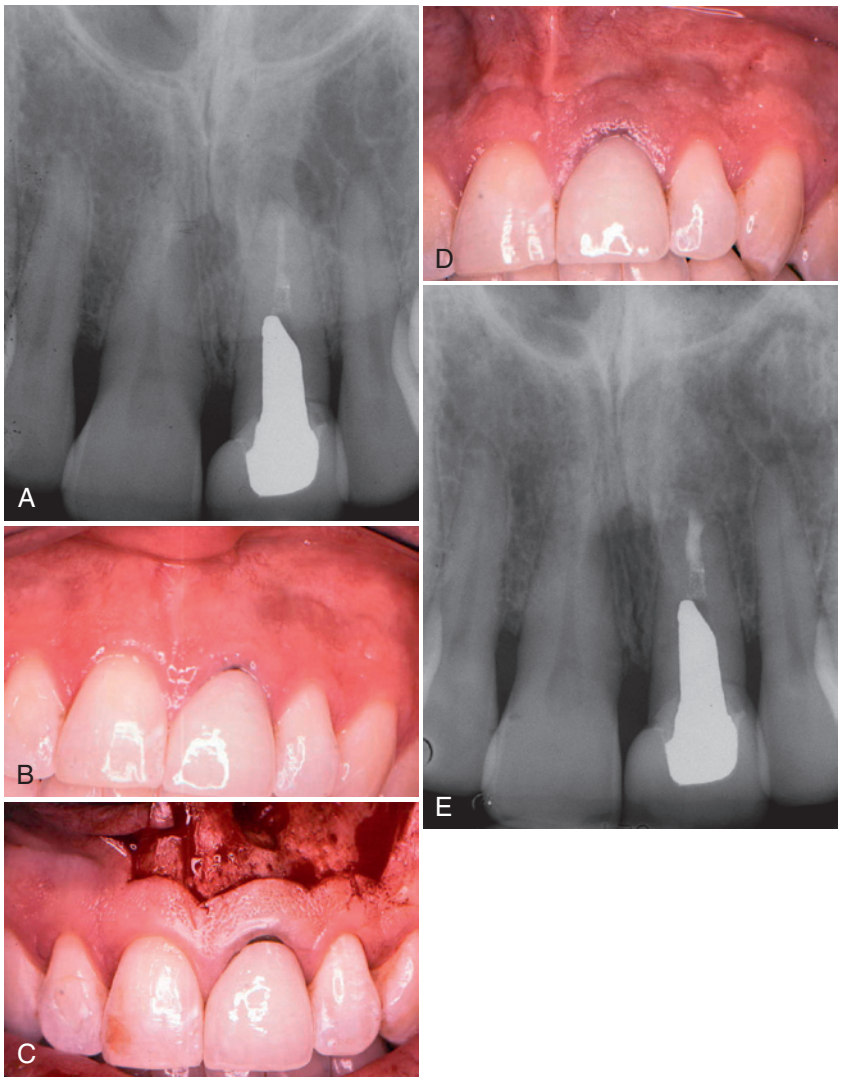

FIG. 8-83 A and B, Preoperative images showing posttreatment disease in the upper left central incisor and a large custom cast post. The patient elected to leave the post and perform surgery rather than risk damage to his new crown. C, Submarginal rectangular flap design. D, The 3-week follow-up showing excellent soft tissue healing. E, The 18-month follow-up showing excellent healing of the periradicular tissues.

radiolucency, pain, periodontal pocket formation, or sinus tract eruption occur, then further treatment may be needed. However, many teeth classified early on as uncertain healing may indeed be retained for many years.[157]

Endodontic surgery (Fig. 8-83) is a very predictable procedure[66,89] that can be performed on most teeth; however, there are some anatomic and medical concerns regarding treatment planning for this procedure, which are covered in detail in another chapter. Extraction-replantation (Fig. 8-84), also referred to as intentional replantation,[170] is another treatment option. This involves extraction of the tooth and performing the apicoectomy and root-end filling while the tooth is out of the patient's mouth, followed by replantation and splinting if indicated. This procedure is also discussed in detail in Chapter 9. Extraction and replacement should be the treatment of last resort to be selected only when the tooth has been shown to be nonrepairable. If the decision is made to extract the tooth, usually replacement will be necessary to prevent shifting of the dentition with its attendant problems. Replacement can be with an implant, a fixed partial denture, or a removable partial denture.

SUMMARY

Posttreatment endodontic disease does not preclude saving the involved tooth. In fact, the majority of these teeth can be returned to health and long-term function by current retreatment procedures. In most instances the retreatment option provides the greatest advantage to the patient because there is no replacement that functions as well as a natural tooth. Armed with the information in the preceding section, appropriate armamentaria, and the desire to do what is best for the patient, the clinician will provide the foundation for long-term restorative success.

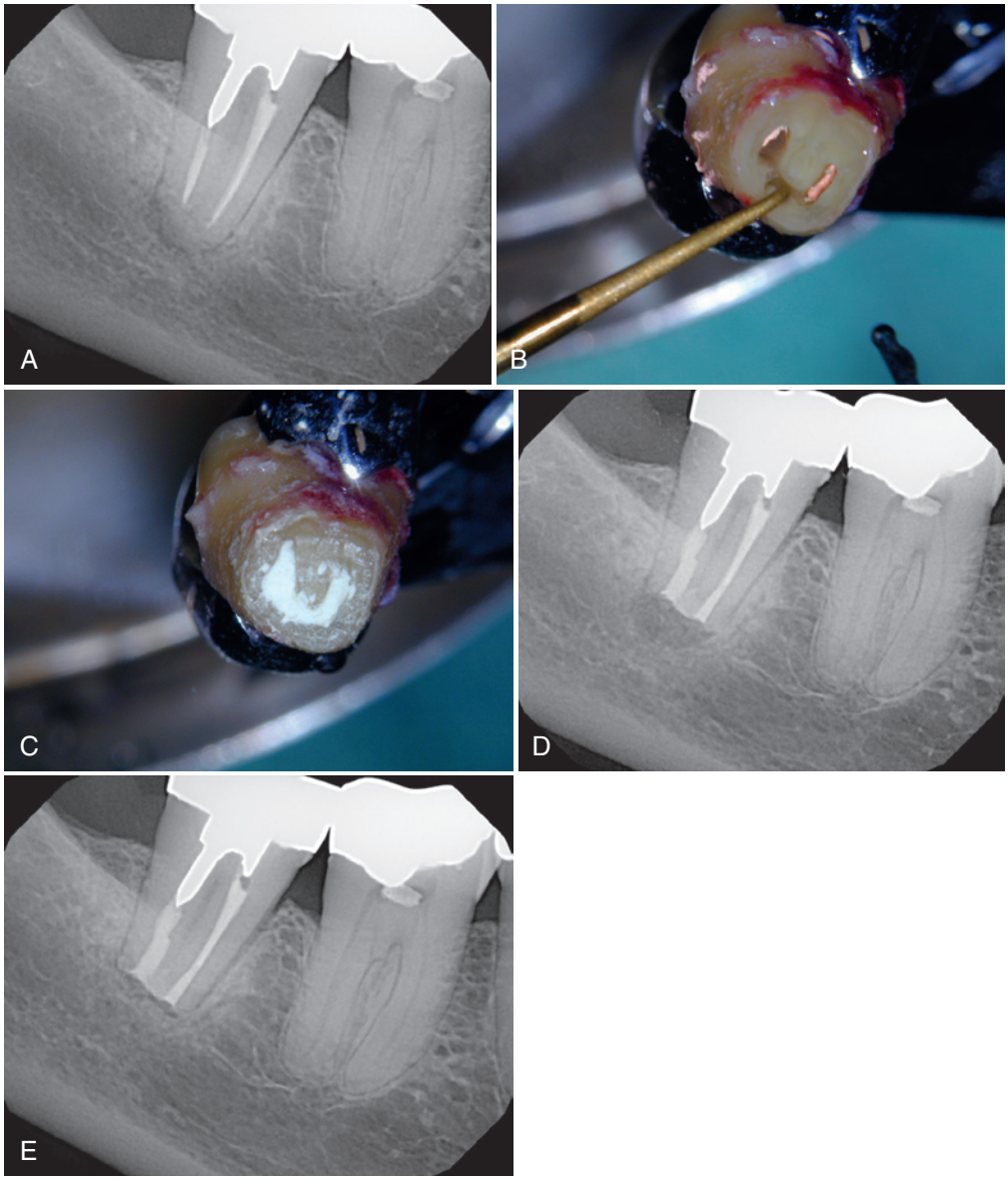

FIG. 8-84 **A,** This lower second molar became symptomatic many years after nonsurgical treatment. Nonsurgical retreatment had a guarded prognosis due to the large multiroot cast custom post and core. Surgery was precluded by the poor access and proximity of the inferior alveolar canal. **B,** Ultrasonic root end preparation is made in the extracted tooth. **C,** A white MTA retrograde filling was placed. Note the C-shape. **D,** Immediate postimplantation radiograph. **E,** The 7-month reevaluation showing apical healing. The patient was asymptomatic.

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CHAPTER 9

Periradicular Surgery

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CHAPTER OUTLINE

Indications for Periradicular Surgery
Etiology of Persistent Periradicular Disease
Rationale for Surgical Treatment
Clinical Decision Making
General Biologic Principles of Wound Healing
Soft-Tissue Wound Healing
Inflammatory Phase
Proliferative Phase
Maturation Phase
Hard-Tissue Healing: Excisional Dentoalveolar Wound
Osteoblasts: Osteogenesis
Cementoblasts: Cementogenesis
Systemic Medications and Wound Healing
Bisphosphonates
Glucocorticoids
Nonsteroidal Anti-inflammatory Drugs (NSAIDs)
Cyclooxygenase-2 (COX-2) Inhibitors
Preoperative Evaluation of Medically Complex Patients
Anatomic Considerations
Posterior Mandible
Posterior Maxilla
Anterior Maxilla and Mandible
Cone-Beam Computed Tomography
Differences Between CT and CBCT Imaging
Potential Applications of CBCT in the Management of Endodontic Posttreatment Disease
Patient Preparation for Surgery
Informed-Consent Issues Specific to Surgery
Premedication: NSAIDs, Antibiotics, Chlorhexidine, and Conscious Sedation
Instruments and Operatory Setup
Local Anesthesia for Surgery
Surgical Access
Soft-Tissue Access
Vertical Incision
Horizontal Incision
Flap Design
Tissue Reflection
Tissue Retraction
Hard-Tissue Access
Periradicular Curettage and Biopsy
Localized Hemostasis
Preoperative Considerations
Local Hemostatic Agents
Cautery/Electrosurgery
Management of the Root End
Determination of the Need for Root-End Resection and Filling
Root-End Resection
Angle of Resection
Root-End Surface Preparation
Resected Root-End Surface Topography
Root-End Conditioning
Root-End Cavity Preparation
Ultrasonic Root-End Preparation and Apical Fractures
Significance of Ultrasonic Tip Design
Temperature Changes Induced by Ultrasonic Instruments
Bonded Root-End Fillings
Root-End Filling Materials
Zinc Oxide–Eugenol (ZOE) Cements
Glass-Ionomer Cements
Diaket
Composite Resins and Resin-Ionomer Hybrids
Mineral Trioxide Aggregate (MTA)
Bioceramics
Overview of Root-End Filling Materials
Closure of the Surgical Site and Selection of Suture Material
Closure of the Surgical Site
Selection of Suture Material
Guided Tissue Regeneration and Endodontic Surgery
Ridge Preservation
Intentional Replantation
Postoperative Care
Management of Surgical Complications
Summary

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Although nonsurgical endodontic treatment is a highly predictable option in most cases, surgery may be indicated for teeth with persistent periradicular pathoses that have not responded to nonsurgical approaches. Surgical root canal therapy, including root-end resection, has been practiced since at least the mid-1800s.[211] In 1906, Schamberg[473] described using radiographs to assist diagnosis and the use of surgical burs to perform a rapid osteotomy and root-end "ablation."

Perhaps the single most important development in dental practice in the early 20th century was the introduction of safe, effective local anesthesia, which allowed for more meticulous and comfortable surgical treatment. Formal recognition of endodontics as a specialty in 1963 ushered in a new era of basic and clinical research focused on the prevention and treatment of pulpal and periradicular pathosis.

Since the 1990s, periradicular surgery has continued to evolve into a precise, biologically based adjunct to nonsurgical root canal therapy. The parallel development of new instruments and materials, along with a better understanding of the biology of wound healing, has made surgical treatment a viable alternative to extraction and tooth replacement, rather than a treatment of last resort.

Periradicular surgery, when indicated, should be considered an extension of nonsurgical treatment, because the underlying etiology of the disease process and the objectives of treatment are the same: prevention or elimination of apical periodontitis. Surgical root canal treatment should not be considered as somehow separate from nonsurgical treatment, although the instruments and techniques are obviously quite different. Surgical treatment accounts for about 3% to 10% of the typical endodontic specialty practice.[1,75,374] A web-based survey found that 91% of active endodontists perform some type of root-end surgery, and almost all are using a dental operating microscope and ultrasonics.[115] Endodontists perform almost 78% of surgical root canal treatments; general dentists and other specialists perform 15.5% and 6.6%, respectively.[242] Although appropriately trained general dentists and other dental specialists may perform periradicular surgery, individuals with advanced training in endodontics have developed most of the current periradicular surgery techniques and science presented in this chapter. We believe that endodontists must continue to include periradicular surgery as a routine part of clinical endodontic practice and not abrogate this treatment option to others, who may not possess the same background, skills, or values.[343,424,439] When patient preferences and quality of life measures are considered, patients can be expected to place high value on endodontic treatment and the retention of a natural tooth.[153,180]

INDICATIONS FOR PERIRADICULAR SURGERY

Etiology of Persistent Periradicular Disease

The first and arguably most important step in treatment decision making is attempting to determine the cause of persistent periradicular disease. Treatment then is directed at eliminating the etiology, which most often is the presence of bacteria and other microbial irritants in the root canal space.[492] Nonsurgical retreatment, when possible, often is the first choice for attempting to correct obvious deficiencies in the previous treatment (see Chapter 8).[564] However, microorganisms can survive even in apparently well-treated teeth in dentinal tubules, canal irregularities, deltas, and isthmus areas.[275,307,568] If residual microorganisms remain completely entombed in the root canal system, periradicular healing should occur. Sealing off all potential routes of microbial escape from the root canal system is the goal of both nonsurgical and surgical treatment. When microorganisms of sufficient pathogenicity and number gain access to the periradicular tissues, pathosis develops.

Enterococcus faecalis is commonly isolated from failing root canal–treated teeth and is known to be especially difficult to eliminate with standard instrumentation and irrigation techniques.[510] Unlike primary endodontic infections, which predominantly are associated with mixed anaerobic microbiota, it has been commonly accepted that treatment failures are more frequently associated with one or two microorganisms.[492] However, research using more sophisticated techniques (16S ribosomal RNA gene clone library analysis) has determined that multiple, previously uncultivated phylotypes can be identified in the majority of teeth with persistent apical periodontitis.[464] Fungi and viruses have also emerged as potential causes of root canal failure and may play either a primary or secondary role in persistent periradicular pathosis.[369,399,462,575]

Established extraradicular colonies of microorganisms also may be a reason for failure of some teeth to respond to nonsurgical treatment. When microorganisms are able to arrange in an extraradicular biofilm, they may be particularly resistant to elimination by host defense mechanisms and antimicrobial agents.[490] Persistent extraradicular root surface colonization cannot be diagnosed by noninvasive methods but may be suspected in well-treated cases that are refractory to nonsurgical treatment.[166,218,465,495,511] Although the presence of extraradicular microbial colonies has been somewhat controversial, studies using DNA-DNA hybridization techniques have confirmed the persistence of microorganisms in the periradicular tissues of some root canal–treated teeth.[187,509]

Overextended filling materials may contribute to treatment failure, presumably as a result of a chronic inflammatory response.[369] Although this is possible and even likely with certain toxic materials (e.g., pastes containing formaldehyde[376]), the role of relatively inert materials such as gutta-percha and set sealer is less clear, and these materials probably become a significant contributing factor only if microorganisms are present. If the root apex is close to the buccal cortical plate, apical fenestration may occur, leading to persistent symptoms, especially tenderness to palpation over the root apex.[72] Some have suggested that overextension of filling materials may contribute to endodontic failure because certain dental materials may induce periodontal ligament (PDL) cell apoptosis.[467] This specific interaction between filling materials and periradicular tissues is not fully understood and deserves further research. The poorer prognosis often reported with overextended root canal fillings also may be related simply to lack of an adequate apical seal and subsequent egress of microorganisms from the root canal space. Regardless, minor overextension of filling material is rarely a sole indication for surgery except when symptoms or periradicular pathosis develops. Significant overextension of filling material, especially when important anatomic areas and possibly toxic materials are involved, is an indication for referral to an endodontist or oral surgeon for evaluation and, possibly, treatment.

The presence of periradicular cholesterol crystals may interfere with healing after nonsurgical root canal treatment.[365]

Although relatively uncommon, true periradicular cysts (completely enclosed, epithelium-lined cavities) may not be expected to resolve after nonsurgical treatment.[364] As with other extraradicular causes of failure, surgery is indicated, because definitive diagnosis and treatment require an excisional biopsy and removal of the periradicular tissue.

Vertical root fractures are a significant cause of failure and may be difficult to diagnose in the early stages.[90] Exploratory surgery often is required to confirm a root fracture. Cone-beam computed tomography (CBCT) is a promising tool for noninvasive diagnosis of root fractures[155,165] and is discussed in greater detail later in this chapter and in Chapter 2. Although several promising approaches to the management of vertical root fractures have been proposed,[226,268,506] the prognosis generally is believed to be poor. Extraction usually is the treatment of choice, especially if appropriate tooth replacement options are available (see Chapter 21). Root amputation or hemisection may be considered in a multirooted tooth if the remaining tooth structure is uninvolved and sufficient periodontal support is present.

The relationship between systemic disease and periradicular healing is not completely understood. The possible influence of certain systemic medications on wound healing is discussed later in the chapter. Compromised host healing capacity may be a contributing factor in delayed healing and failure of some root canal–treated teeth. For example, complete healing after nonsurgical root canal treatment is less likely in diabetic patients with preoperative periradicular pathosis.[78,175] Patients undergoing immunosuppressive therapy may be at greater risk for delayed healing, treatment failure, or acute exacerbation of a subclinical infection, although two studies involving subgroups of immunocompromised patients (bone marrow transplant and acquired immunodeficiency syndrome [AIDS]) did not find these patients to be at higher risk for complications related to endodontic treatment.[197,406]

Rationale for Surgical Treatment

Although nonsurgical retreatment generally is believed to be the preferred first approach in the management of persistent apical periodontitis,[77,492,510,564] periradicular surgery is indicated when nonsurgical retreatment is impractical or unlikely to improve on the previous result (see Chapter 8). In particular, a surgical approach may be the first choice for managing teeth with long posts or irretrievable separated instruments, nonnegotiable ledges and canal blockages or transportation, hard cement filling materials, failure of previous nonsurgical retreatment, and suspected vertical root fracture, or when a biopsy is indicated (Figs. 9-1 to 9-4). Even when surgical treatment is the likely definitive approach, nonsurgical therapy before the procedure may be recommended to help reduce the number of microorganisms in the root canal system and ensure a more favorable long-term prognosis.[230] On the other hand, surgery may be the first choice even if a tooth can be treated nonsurgically if the risks and costs of retreatment are considered excessive. For example, disassembling a recently restored bridge abutment tooth to allow endodontic retreatment may be technically possible but not economically feasible. One study found that endodontic microsurgery may be the most cost-effective option for management of persistent periapical disease, compared to nonsurgical retreatment, extraction and placement of a fixed partial denture, and extraction and placement of a single tooth implant.[284] Case-specific variables and clinical judgment are key elements in the decision-making process, as current evidence supports the contention that the prognosis for surgical treatment is approximately the same as that for nonsurgical retreatment.[127,128,135,293,468,577]

FIG. 9-1 Previously treated maxillary lateral incisor with persistent periradicular disease. Nonsurgical retreatment is possible but would involve disassembly of an otherwise adequate coronal restoration. Periradicular surgery is a reasonable option.

Clinical Decision Making

Clinical decision making is a process that combines the best available evidence, clinical judgment, and patient preferences. Treatment choices are always made under conditions of at least some uncertainty. Surgical root canal treatment rarely is the only possible choice. Clinicians and patients must weigh the relative benefits, risks, and costs of two or more acceptable alternatives. Patients and clinicians can be expected to hold different attitudes about the value of potential treatment outcomes.[269] In a prognosis study of single-tooth dental implants, Gibbard and Zarb[193] reported that factors considered significant by health care professionals may not be important to patients. Even among groups of dentists and dental specialists, the threshold for treatment varies widely and treatment recommendations may depend more on personal values and experience than objective analysis of treatment costs, prognosis, risks, and alternatives.[56,343,439]

Shared decision making, in which the patient and clinician consider outcome probabilities and patient preferences and agree on the appropriate treatment, is preferable to a clinician-directed treatment decision.[46,179] Such a two-way exchange allows the clinician to provide the best available current evidence and case-specific clinical judgment while encouraging a decision that considers the patient's personal values and preferences. This decision-making model has been shown to increase patient knowledge about and satisfaction with the treatment choice.[262,386,387] In general, most patients prefer to be actively involved in the decision-making process but want to leave the specifics of the treatment to the clinician.[179] That is, the risks,

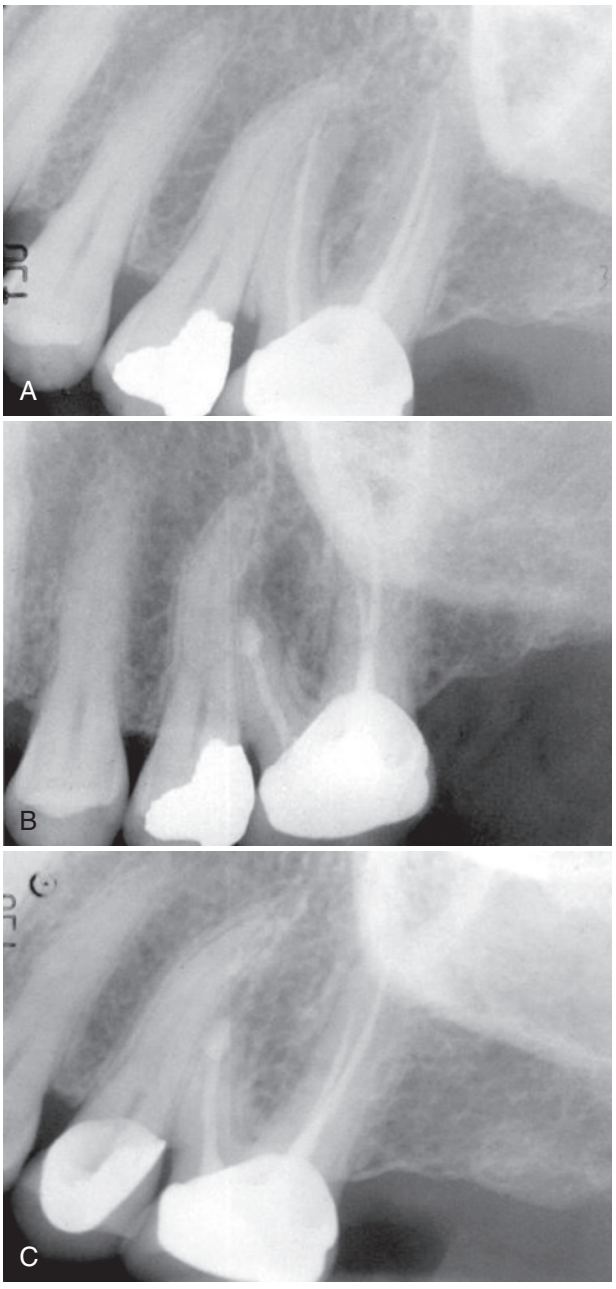

FIG. 9-2 A, Previously treated maxillary first molar with persistent periradicular disease and obvious transportation of the main mesiobuccal (MB-1) canal. Nonsurgical retreatment is unlikely to correct this iatrogenic condition, and surgery is the preferred choice. B, Immediate postsurgery radiograph. The root-end preparation and fill was extended from the MB-1 canal in a palatal direction to include the isthmus area and the second (MB-2) canal. C, One-year follow-up examination: the tooth is asymptomatic, and periradicular healing is apparent radiographically. Although surgery was the first-choice treatment in this case and the outcome was favorable, a good argument could be made for nonsurgical retreatment before surgery to ensure disinfection of the canal and to attempt to locate a MB-2 canal.

benefits, and costs of treatment alternatives are important to patients, but the details of the procedure usually are not. Shared decision making is particularly relevant in light of the current trend in some areas to recommend implants as an alternative to root canal treatment.

Comparative probabilities for a successful outcome after surgical root canal treatment, nonsurgical retreatment, or extraction and replacement with a fixed prosthesis or implant are difficult to project. Many treatment variables are complex and not easily quantified, such as the affected location in the mouth, bone quality, practitioner's skill, possible influence of systemic disease on healing, periodontal support, bulk of the remaining tooth structure and resistance to fracture, quality of the coronal restoration, patient susceptibility to recurrent caries, materials used, and other factors. In addition, the definition of success varies considerably and is inconsistent from study to study.

Boioli and colleagues[66] performed a meta-analysis of implant studies and reported a 93% survival rate at 5 years. The 5- to 10-year success rate for implant retention routinely is reported to be 90% to 97%, depending on the location in the mouth and other variables.[302,309] Surgical root canal treatment was previously reported to have a lower success rate than implant placement.[230,546] However, most of these older studies would be considered weak by today's evidence-based standards and, more important, do not reflect the use of many of the newer surgical materials and techniques. With careful case selection, surgical skill, and the use of materials and techniques described later in this chapter, many studies have demonstrated success rates over 90% for surgical root canal treatment.[326,448,500,501,546,609] von Arx reported that the 5-year success for teeth receiving endodontic microsurgery was 8% less than observed at 1 year.[566] A similar study found an 85% success rate after 10 years.[500] A systematic review and meta-analysis comparing traditional root-end surgery to modern endodontic microsurgery (ultrasonic root-end preparation; root-end filling with IRM, Super EBA, or MTA; and high power [> ×10] magnification and illumination) found that the outcome for endodontic microsurgery using current materials and techniques was 94%, compared to 59% success with traditional root-end surgery.[483]

In broad terms and under ideal conditions, the prognoses for nonsurgical retreatment, surgical treatment, and implant placement should be roughly equal. The choice of treatment should be based on the best available evidence, case-specific clinical judgment, and the patient's preferences. As a stronger evidence base continues to develop, we predict that algorithms encompassing multiple patient and treatment variables will be devised to assist clinical decision making.

GENERAL BIOLOGIC PRINCIPLES OF WOUND HEALING

Wound healing varies from region to region in the body and depends on several factors, including the type of tissue, the type of wound, and the type of healing. In periradicular surgery, the tissues include free and attached gingiva, the alveolar mucosa, periosteum, bone, the periodontal ligament, and cementum. The wound may be intentional surgical trauma, which includes incision, blunt dissection, and excision (surgical), or pathologic or traumatic wounds. Healing occurs by primary or secondary intention.[212]

An incisional blunt dissectional wound, for example, can be considered to heal typically by primary intention, whereas a dissectional wound involving the resected root surface and osseous crypt heals by secondary intention. An important concept of the wound healing process in general is the difference between regeneration and repair. The goal of all surgical

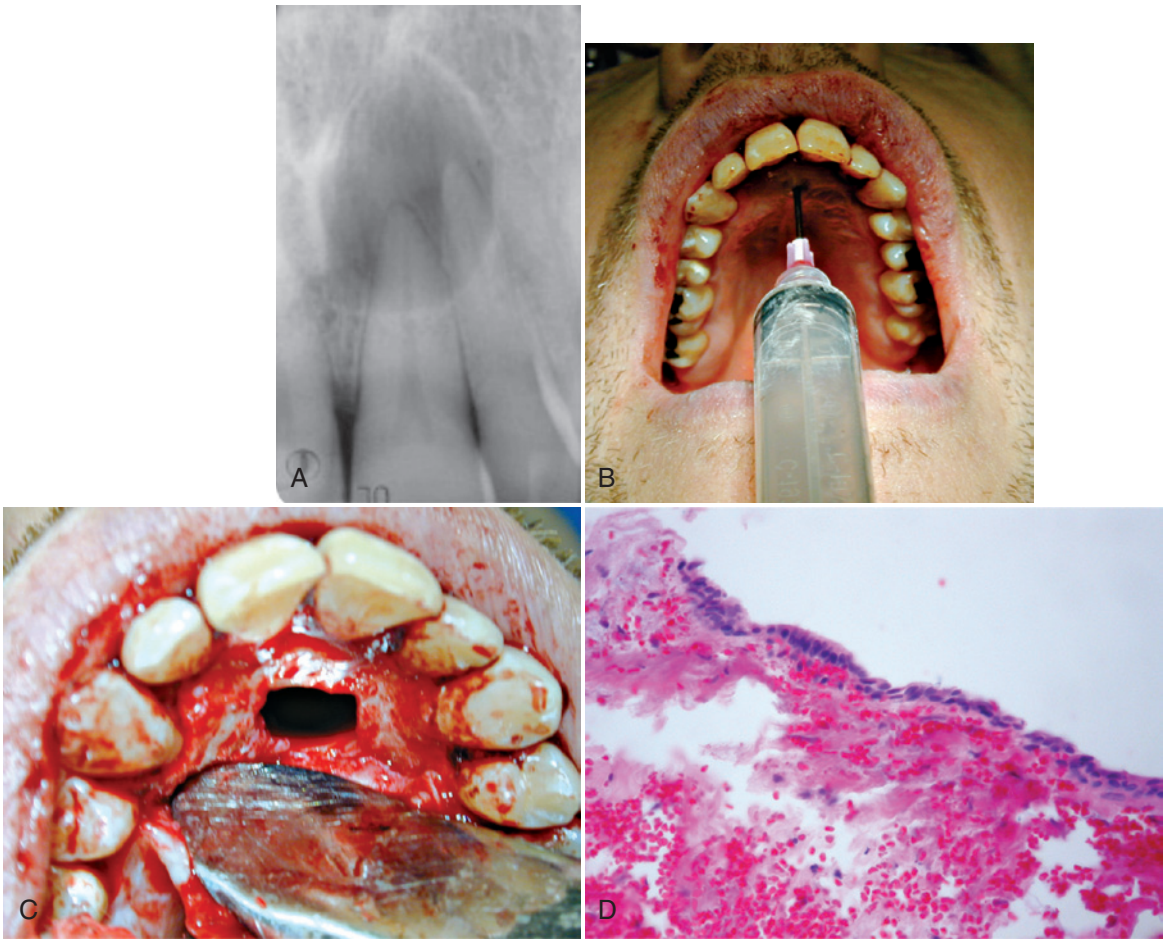

FIG. 9-3 A, Indication for surgery (biopsy): large radiolucent lesion in the area of the maxillary left central and lateral incisors was detected on routine radiographic examination. All anterior teeth responded within normal limits to pulp vitality testing. B, After administration of a local anesthetic but before surgery, the lesion was aspirated with a large-gauge needle to rule out a vascular lesion. C, Buccal and palatal flaps were reflected. The lesion was directly accessible from the palate. An excisional biopsy was performed, and the sample was submitted for evaluation. D, Light microscopic section of the biopsy specimen; the lesion was diagnosed as a nasopalatine duct cyst. (×400.) (Courtesy Dr. Vince Penesis.)

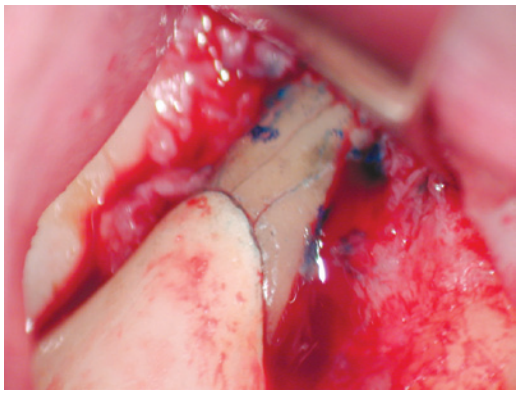

FIG. 9-4 Exploratory surgery was indicated to rule out or confirm a root fracture. Magnification and staining with methylene blue dye confirmed the presence of multiple root fractures. The tooth was subsequently extracted. (Courtesy Dr. Martin Rogers.)

procedures should be *regeneration,* which returns the tissues to their normal microarchitecture and function, rather than *repair,* a healing outcome in which tissues do not return to normal architecture and function. Repair typically results in the formation of scar tissue.

The wounding process varies, depending on the types of tissue and injury. However, all wounds progress through three broad, overlapping phases in the process of healing; these are the inflammatory phase, the proliferative phase, and the maturation phase.[240,570] Although these phases can be identified in healing tissue, none of them has a clear beginning or end. Furthermore, in a wound such as an endodontic surgical site, which involves more than one tissue type, these phases progress at different rates in each type of tissue.

SOFT-TISSUE WOUND HEALING

Inflammatory Phase

Broadly speaking, the inflammatory phase of healing is similar for all tissues.[543] This phase can be broken down further into clot formation, early inflammation, and late inflammation.

Clot Formation

Clot formation begins with three events: (1) blood vessel contraction is initiated by platelet degranulation of serotonin, which acts on the endothelial cell and increases the permeability of the vessel, allowing a protein-rich exudate to enter the wound site; (2) a plug composed of platelets forms, primarily through intravascular platelet aggregation; (3) both the extrinsic and intrinsic clotting mechanisms are activated. Several other events occur simultaneously, including activation of the kinin, complement, and fibrinolytic systems and the generation of plasmin.[34,247,521] These events stabilize hemostasis, begin the production of a number of mitogens and chemoattractants, and initiate the process of wound decontamination. The result is a coagulum consisting of widely spaced, haphazardly arranged fibrin strands with serum exudate, erythrocytes, tissue debris, and inflammatory cells. Compression of the surgical flap with sterile iced gauze immediately after surgery is designed to minimize the thickness of the fibrin clot and thereby accelerate optimal wound healing.

Early Inflammation: Polymorphonuclear Neutrophil (PMN) Organization

As a result of production of chemoattractants by the various components of the clot, polymorphonuclear neutrophils (PMNs) begin to enter the wound site within 6 hours of clot stabilization. The number of PMNs increases steadily, peaking at about 24 to 48 hours after the injury. Three key steps mark PMN migration into the wound site: (1) *pavementing,* in which the red blood cells undertake intravascular agglutination, allowing the PMNs to adhere to the endothelial cells; (2) *emigration,* in which the PMNs actively pass through the vascular wall; and (3) *migration,* in which the PMNs use ameboid motion, under the influence of the various chemotactic mediators, to move into the injured tissues.[212]

The principal role of the PMNs is wound decontamination by means of phagocytosis of bacteria. The high number of PMNs in the wound site is relatively short lived; the number drops rapidly after the third day.

Late Inflammation: Macrophage Organization

About the time the PMN population is declining (48 to 96 hours after injury), macrophages begin to enter the wound site. They reach a peak concentration by approximately the third or fourth day. These cells, which are derived from circulating monocytes, leave the bloodstream under the influence of the chemoattractants in the wound site. The monocytes subsequently evolve into macrophages. Macrophages have a much longer life span than PMNs; they remain in the wound until healing is complete. Similar to PMNs, macrophages play a major role in wound decontamination through phagocytosis and digestion of microorganisms and tissue debris.

Macrophages are considerably more bioactive than PMNs and can secrete a vast array of cytokines. A key action of many of these bioactive substances is initiation of the proliferative phase of wound healing, which is accomplished by prompting the formation of granulation tissue. Two other major functions of macrophages are ingestion and processing of antigens for presentation to T lymphocytes, which enter the wound after the macrophages. Unlike PMNs, macrophages play an essential role in the regulation of wound healing.[109,224] A reduction in the number of macrophages in the wound site delays healing, because the wound does not progress to the next phase. For example, the age-related reduction in healing potential appears to be partly the result of loss of estrogen regulation of macrophages in healing tissues.[33]

Proliferative Phase

The proliferative phase is characterized by the formation of granulation tissue in the wound. Two key cell types, fibroblasts and endothelial cells, have a primary role in the formation of granulation tissue. Granulation tissue is a fragile structure composed of an extracellular matrix of fibrin, fibronectin, glycosaminoglycans, proliferating endothelial cells, new capillaries, and fibroblasts mixed with inflammatory macrophages and lymphocytes. Epithelial cells also are active during this phase of soft-tissue healing and are responsible for initial wound closure. Guided tissue regeneration (GTR) procedures are based on control of the epithelial cell growth rate during this phase.

Fibroblasts: Fibroplasia

Undifferentiated mesenchymal stem cells in the perivascular tissue and fibroblasts in the adjacent connective tissue migrate into the wound site on the third day after injury and achieve their peak numbers by approximately the seventh day. This action is stimulated by a combination of cytokines (e.g., fibroblast growth factor [FGF], insulin-like growth factor–1 [IGF-1], and 2-15 platelet-derived growth factor [PDGF]), which are produced initially by platelets and subsequently by macrophages and lymphocytes. As the number of macrophages declines and the fibroblast population increases, the tissue in the wound transforms from a *granulomatous* tissue to a *granulation* tissue.

Fibroblasts are the crucial reconstructive cell in the progression of wound healing, because they produce most of the structural proteins forming the extracellular matrix (e.g., collagen). Collagen is first detected in the wound about the third day after injury. The fibroblasts produce type III collagen initially and then, as the wound matures, type I collagen. As this network of collagen fibers is laid down, endothelial and smooth muscle cells begin to migrate into the wound. Subsequently, as wound healing progresses, the collagen fibers become organized by cross-linking. Regularly aligned bundles of collagen begin to orient so as to resist stress in the healing wound.[108,286,413,516]

A focused type of fibroblast, known as a *myofibroblast,* plays a significant role in wound contraction, particularly in incisional-type wounds.[316,524] Myofibroblasts align themselves parallel with the wound surface and then contract, drawing the wound edges together. These cells are eliminated by apoptosis after wound closure.[137,138]

Endothelial Cells: Angiogenesis

Capillary buds originate from the vessels at the periphery of the wound and extend into the wound proper. This occurs concurrently with fibroblast proliferation and can begin as early as 48 to 72 hours after injury. Without angiogenesis, the wound would not have the blood supply needed for further active healing. The capillary sprouts eventually join to form a network of capillary loops *(capillary plexuses)* throughout the wound.

In addition to a low oxygen concentration in the wound proper,[198,289] several factors have been identified as potent stimulators of angiogenesis, including vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), acidic FGF (aFGF), transforming growth factor–alpha

(TGF-alpha) and transforming growth factor–beta (TGF-beta), epidermal growth factor (EGF), interleukin-1 (IL-1), and tumor necrosis factor–alpha (TNF-alpha),[145] as well as lactic acid.[248,258,503] All of these have been shown to stimulate new vessel development.

Epithelium

The first step in epithelial healing is the formation of an epithelial seal on the surface of the fibrin clot. This process begins at the edge of the wound, where the basal and suprabasal prickle cells rapidly undergo mitosis. The cells then migrate across the fibrin clot at a remarkable rate (0.5 to 1 mm per day). This monolayer of epithelial cells continues to migrate by contact guidance along the fibrin scaffold of the clot below. Migration stops as a result of contact inhibition of the epithelial cells from the opposing wound edge. Once the epithelium from both sides of the wound is in contact, an epithelial seal is achieved. In wounds healing by primary intention, formation of an epithelial seal typically takes 21 to 28 hours after reapproximation of the wound margins.[222]

Maturation Phase

Under ideal conditions, maturation of the wound begins 5 to 7 days after injury. A reduction in fibroblasts, vascular channels, and extracellular fluids marks the transition to this phase of healing. During the early stages of wound maturation, the wound matrix is chiefly composed of fibronectin and hyaluronic acid. As the tensile strength of the wound increases, significant upregulation of collagen fibrogenesis occurs. Collagen remodeling ensues, with the formation of larger collagen bundles and alteration of intermolecular cross-linking. The result is a conversion of granulation tissue to fibrous connective tissue and a decreased parallelism of collagen to the plane of the wound. Aggregated collagen fibril bundles increase the tensile strength of the wound. As healing progresses in the wound, the collagen gradually reorganizes; this requires degradation and reaggregation of the collagen. The degradation of collagen is controlled by a variety of collagenase enzymes. Remodeling results in a gradual reduction in the cellularity and vascularity of the reparative tissue; the degree to which this occurs determines the extent of scar tissue formation. Active remodeling of scar tissue can continue very slowly for life.[138,247]

Maturation of the epithelial layer quickly follows formation of the epithelial seal. The monolayer of cells forming the epithelial seal differentiates and undergoes mitosis and maturation to form a definitive layer of stratified squamous epithelium. In this way an epithelial barrier is formed that protects the underlying wound from further invasion by oral microbes. The epithelial barrier typically forms by 36 to 42 hours after suturing of the wound and is characterized by a significant increase in wound strength.[222]

HARD-TISSUE HEALING: EXCISIONAL DENTOALVEOLAR WOUND

The inflammatory and proliferative phases of healing for hard tissue are similar to those for soft tissue. A clot forms in the bony crypt, and an inflammatory process ensues that involves PMNs initially and macrophages subsequently. This is followed by the formation of granulation tissue with an angiogenic component. However, the maturation phase of hard-tissue healing differs markedly from that for soft tissues, primarily because of the tissues involved: cortical bone, cancellous bone, alveolar bone proper, endosteum, periodontal ligament, cementum, dentin, and inner mucoperiosteal tissue.

Osteoblasts: Osteogenesis

The healing of an excisional osseous wound that is approximately 1 cm in diameter is similar to that of a fractured long bone. It progresses from hematoma to inflammation, eradication of nonvital debris, proliferation of granulation tissue, callus formation, conversion of woven bone to lamellar bone and, finally, remodeling of the united bone ends. The coagulum that initially forms delays healing and must be removed to allow wound healing to progress.

A major difference between soft- and hard-tissue wound healing is found in the role of the osteoclast. Functionally, osteoclasts act as organizational units to debride necrotic bone from the wound margin, much as macrophages remove tissue debris from the clot. Granulation tissue begins to proliferate from the severed periodontal ligament by 2 to 4 days after root-end resection.[223] This tissue rapidly encapsulates the root end. Simultaneously, endosteal proliferation into the coagulum occurs from the deep surface of the bony wound edge. The coagulum in the bony crypt is quickly converted into a mass of granulation tissue. In addition to those already discussed, several cell types migrate into the coagulum, including osteoprogenitor cells, preosteoblasts, and osteoblasts. These cells begin the formation of woven bone within the mass of granulation tissue. New bone formation is apparent about 6 days after surgery.[223]

Bone formation can be categorized into two types, each having several phases. The phases differ, depending on which type of formation is involved. One type of bone formation is a matrix vesicle–based process, and the other type is based on osteoid secretion. In both processes, osteoblasts produce the bone matrix. They secrete a collagen-rich ground substance that is essential for mineralization. Osteoblasts also cause calcium and phosphorus to precipitate from the blood.

In the formation of woven bone, which occurs by the matrix vesicle–based process, osteoblasts produce matrix vesicles through exocytosis (the release of substances contained in a vesicle within a cell by a process in which the membrane surrounding the vesicle unites with the membrane forming the outer wall of the cell) of their plasma membranes. As hydroxyapatite crystals accrue in the vesicles, they become enlarged and eventually rupture. This process begins with the deposition and growth of hydroxyapatite crystals in the pore regions. The crystals then amalgamate to form structures known as *spherulites*. Union of the separate spherulites results in mineralization.

The formation of lamellar bone does not require the production of matrix vesicles; rather, it proceeds by the osteoblast secretion process. Osteoblasts secrete an organic matrix composed of longitudinally arranged collagen matrix fibrils (mainly type I collagen). Mineralization occurs by mineral deposition directly along the collagen fibrils.[239,240] This stage has been associated with a rise in pH, most likely because of the enzyme alkaline phosphatase, which is secreted by osteoblasts and other cells and plays an important role in mineralization. The exact role of alkaline phosphatase during mineralization is unclear. The balance of evidence favors a positive catalytic role.[19,20] Some hypothesize[71,70,552,553] that alkaline phosphatase promotes mineralization through a combination of mechanisms involving various cells, extracellular matrix proteins, and elements. The interaction of alkaline phosphatase and

phosphoproteins in both bone and dentin appears to be especially critical to the mineralization process.[71,72,557,558]

Inhibitor molecules, such as pyrophosphate and acidic noncollagenous bone proteins, regulate mineralization. Several growth factors also have been identified as key components in the production of osseous tissue. These include TGF-beta, bone morphogenetic protein (BMP), PDGF, FGF, and IGF.[97,191,476,513] One clinical study demonstrated that the addition of autologous platelet concentrate to the surgical site decreased postsurgical pain and may accelerate the healing process.[134]

Three to 4 weeks after surgery, an excisional osseous wound is 75% to 80% filled with trabeculae surrounded by intensely active osteoid and osteoblastic cells. A reforming periosteum can be seen on the outer surface of the wound; it is highly cellular and has more fibrous connective tissue oriented parallel to the plane of the former cortical plate. At 8 weeks after surgery, the trabeculae are larger and denser and the osteoblasts are less active; these cells occupy about 80% of the original wound. Also, fewer osteoid cells are associated with the maturing trabeculae. The overlying periosteum has reformed and is in contact with the newly developed bone. The osseous defect typically is filled with bone tissue by 16 weeks after surgery. However, the cortical plate has not yet totally reformed. Maturation and remodeling of the osseous tissue continues for several more months.[223]

Local healing is also influenced systemically by the endocrine system and its three general categories of hormones: polypeptide regulators (parathyroid hormone, calcitonin, insulin, and growth hormone), steroid hormones (vitamin D_3, glucocorticoids, and the sex hormones), and thyroid hormones.[219]

Cementoblasts: Cementogenesis

During regeneration of the periradicular tissues, cementum forms over the surface of surgically resected root ends.[114] The exact spatial and temporal sequences of events leading to this formation of new cementum remains undefined; however, cementogenesis is important, because cementum is relatively resistant to resorption (osteoclasts have little affinity for attaching to cementum).

Cementogenesis begins 10 to 12 days after root-end resection. Cementoblasts develop at the root periphery and proceed centrally toward the root canal.[24] Considerable evidence indicates that the cells that regulate cementogenesis are derived from ectomesenchymal cells in the tooth germ proper rather than from bone or other surrounding tissues. The migration and attachment of precementoblasts to the root surface dentin is monitored by mediators from within the dentin itself.[140,210] Cementum covers the resected root end in approximately 28 days. Newly formed PDL fibers show a functional realignment that involves reorientation of fibers perpendicular to the plane of the resected root end, extending from the newly formed cementum to the woven bone trabeculae. This occurs about 8 weeks after surgery.[212,319,320]

SYSTEMIC MEDICATIONS AND WOUND HEALING

Bisphosphonates

Bisphosphonates are commonly used for the treatment of osteopenia, osteoporosis, Paget's disease of bone, multiple myeloma, and metastatic bone, breast, and prostate cancer. The potential association between bisphosphonate use and osteonecrosis of the jaw was first reported in 2003.[332,351] The current preferred term for this condition is antiresorptive agent-induced osteonecrosis of the jaw (ARONJ)[229] or medication-related osteonecrosis of the jaw (MRONJ). The change in terminology reflects the finding that, in addition to bisphosphonates, other antiresorptive agents can increase the risk for osteonecrosis of the jaw (e.g., denosumab). ARONJ can occur spontaneously but is more commonly associated with dental procedures that involve bone trauma. Patient variables that increase the risk for ARONJ include age (over 65 years old), chronic use of corticosteroids, use of bisphosphonates for more than 2 years, smoking, diabetes, and obesity.[278,334,340,583] It is important to note that the risk for ARONJ with commonly prescribed oral bisphosphonates appears to be very low, whereas the overall benefits of this drug class for reducing morbidity and mortality related to hip, vertebral, and other bone fractures are significant.[229] The estimated incidence for ARONJ in patients taking oral bisphosphonates ranges from zero to 1 in 2260 cases, although dental extractions may quadruple the risk of developing ARONJ.[7,340] A reasonable upper estimate of the risk of developing ARONJ in a patient who does not have cancer is about 0.1%.[229] One clinical study reported a much higher 4% risk for developing ARONJ in patients taking a specific oral bisphosphonate (alendronate sodium),[479] although this was a retrospective study using an electronic patient record at a dental school and may not have adequately controlled for other relevant variables. The majority of ARONJ cases have been reported in patients taking IV bisphosphonates (e.g., zoledronic acid and pamidronate). According to one report, approximately 20% of patients taking IV bisphosphonates may develop ARONJ.[7,340] A systematic review found that there was a strong association between cancer and risk of ARONJ, with the prevalence ranging from 0.7% to 13.3%.[352] Interestingly, this same review found that the higher-quality studies reported the highest prevalence of ARONJ in cancer patients.

Conservative and timely management of apical periodontitis is essential to reduce the risk of ARONJ in patients taking bisphosphonates. Because periapical pathosis may exacerbate or increase the risk for ARONJ, the "no treatment" option is not a viable choice. The potential risk for ARONJ should be thoroughly discussed with all patients that are taking bisphosphonates, as well as treatment options. Nonsurgical retreatment should usually be considered the first choice, especially for patients with a history of IV bisphosphonate use or other risk factors. Even so, surgical treatment may be indicated to manage chronic or acute apical periodontitis. When the only viable treatment options for the management of persistent periradicular inflammation are surgical root canal therapy or extraction, the question of which option is more or less likely to lead to ARONJ in at-risk patients remains unanswered. In general, the procedure that could most predictably eliminate the periradicular inflammation with the least amount of surgical trauma would be preferred. Conservative surgical technique, primary tissue closure, and use of chlorhexidine mouth rinses preoperatively and during the healing stage are recommended.[229] There is some limited evidence to support the use of prophylactic antibiotics and chlorhexidine mouth rinse to decrease the risk of ARONJ for patients undergoing surgical dental treatment.[229] Because bisphosphonates have a substantial half-life when

incorporated into bone, discontinuation of bisphosphonates prior to dental treatment is unlikely to provide any measurable benefit and may place the patient at greater risk for complications that the drug is intended to prevent.

The possibility of predicting which patients may be at greater risk for ARONJ based on serum levels of C-terminal cross-linking telopeptide of type I collagen (CTX) has been suggested based on clinical observations.[333] This observation may provide a useful clue for future risk assessment but requires additional refinement and validation prior to widespread acceptance. In fact, one study found that although serum CTX levels were lower in patients taking bisphosphonates (as expected), there was no association between lower CTX levels and increased risk of ARONJ.[208] This same study found that patients taking a once-a-year dose of 5 mg zoledronic acid were at no greater risk for ARONJ than a control group (1 case in 5903 patients). Once ARONJ develops, treatment options are limited. A possible treatment strategy proposed in a series of case reports is the use of teriparatide, an anabolic drug that stimulates osteoblast formation and is used for the management of osteoporosis (see also Chapter 3).[505]

Glucocorticoids

Glucocorticoid therapy has been shown to induce rapid bone loss within the first 3 months of treatment. Even inhaled steroids have been implicated as a cause of bone loss. Bone formation is inhibited partly through a decrease in osteoblast life span and function, a reduction in the mineral apposition rate, and a prolonged mineralization lag time. Biochemical markers of bone formation (i.e., osteocalcin and bone-specific alkaline phosphatase) are suppressed. In addition to this primary suppressive effect, glucocorticoids cause accelerated bone resorption. The number and activity of osteoclasts increase during early glucocorticoid exposure. With continued use of glucocorticoids, the rapid rate of osteoclast-mediated bone resorption slows, but suppression of bone formation continues as the overriding skeletal activity.[272] Bone loss therefore is progressive, because bone resorption chronically exceeds bone formation.

The adverse effects of glucocorticoids on bone are mediated by sex steroid deficiency and expression of locally produced growth factors and related proteins, such as reduced production of IGF-1 and alterations in IGF-binding proteins in osteoblasts. The direct effect on calcium metabolism through alteration of vitamin D metabolism leads to a state of secondary hyperparathyroidism.[447,581,582]

Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

Bone homeostasis is regulated by many factors, including prostaglandins (PGs).[267] PGs are important to both normal and pathologic bone turnover. PGs modulate osteoblast proliferation and differentiated functions.[426,427] The levels of prostaglandins E (PGE) and F (PGF) are elevated in the early phase of fracture healing, and administration of PGE_2 has increased the rate of osseous repair in several animal studies.[272] NSAID inhibition of the enzyme cyclooxygenase (COX), which is involved in the synthesis of PGs, is the same mechanism by which NSAIDs control pain. By inhibiting the COX enzymes and the subsequent production of prostaglandins, NSAIDs accomplish the desired anti-inflammatory effects—but also prevent the increased production of PGs required for bone healing. In vitro studies using animal models have shown that NSAIDs inhibit osteoblast proliferation and stimulate protein synthesis.[235] These drugs also have been shown to delay fracture healing and to affect bone formation adversely in animals and humans.[15,192] In one study, the use of NSAIDs reduced the amount of bony ingrowth into an orthopedic implant.[220]

In other studies, NSAIDs reduced bone resorption associated with experimentally induced periapical lesions,[18,390] and systemic NSAIDs may play a positive role in maintaining bone height around endosseous titanium dental implants.[257] Additional research is needed to clarify the specific influence of NSAIDs on healing after periradicular surgery.

Cyclooxygenase-2 (COX-2) Inhibitors

Although both COX-1 and COX-2 have been identified in osteoblasts, the different roles of the two cyclooxygenases in bone formation remain unclear. A study using COX-1 and COX-2 knockout mice compared the roles of the two enzymes in fracture healing. COX-2 was shown to have an essential role in both endochondral and intramembranous bone formation during wound healing. COX-2 knockout mice showed a persistent delay in cartilage tissue ossification. No difference in fracture healing was observed between the COX-1 knockout mice and the wild-type control used in this study.[603] Additional studies have shown a persistent delay in healing with COX-2 NSAIDs[202,203]; however, this difference in effect was not apparent clinically in a study examining the healing of spinal fusion wounds.[182]

PREOPERATIVE EVALUATION OF MEDICALLY COMPLEX PATIENTS

The preoperative assessment must take into account both the type of procedure planned and the type of patient (i.e., physical health and psychological status). Healthy patients clearly can be expected to tolerate surgical procedures better than medically complex patients (see Chapters 3 and 26). Clinicians should anticipate and prepare for the inevitability of treating more medically complex patients as the population ages. It is beyond the scope of this brief section to cover all possible medical considerations; rather, the most common issues that may require modification of an endodontic surgical treatment plan are presented. Relatively few absolute contraindications to periradicular surgery exist for patients well enough to seek care in an ambulatory dental office. Nonetheless, if any question arises about a patient's ability to tolerate a surgical procedure, medical consultation is advised. A thorough medical history and assessment of vital signs are required parts of the presurgical evaluation.

The American Society of Anesthesiologists (ASA) has developed a widely used system for establishing surgical risk. Patients categorized as ASA 1 are healthy and usually require no modification of the surgical treatment plan. Patients classified as ASA 4 or ASA 5 are not treated in a dental office; these individuals have significant medical problems that take priority over dental considerations. Patients considered ASA 2 or ASA 3 are commonly seen on an outpatient basis and may require medical consultation and modification of the surgical treatment plan. Patients in the ASA 2 and ASA 3 categories have mild to moderate systemic disease and often are undergoing treatment with one or more prescription medications. The ASA

classification system should only be used as a general guide because, when used alone, the ASA system is not a reliable predictor of operative risk.[201] In addition, even experienced anesthesiologists exhibit differences of opinion when using the ASA system to classify patients.[227,395] The patient's psychological status and anticipated procedural stress should be considered in addition to the ASA classification of physical health. A stress reduction protocol may be helpful for patients who report moderate dental anxiety with concurrent mild to moderate systemic disease (see Chapter 28).

Surgical procedures typically require a larger amount of local anesthetic with a vasoconstrictor than nonsurgical root canal treatment. An essential part of the presurgical evaluation is an assessment of the patient's cardiovascular status and tolerance for local anesthetics that contain epinephrine. In particular, patients with advanced cardiovascular disease, geriatric patients, and patients taking certain medications may have a reduced tolerance for local anesthetics containing a vasoconstrictor (see Chapters 3 and 4). Treatment-induced stress associated with surgical procedures can cause a significant increase in heart rate and systolic blood pressure when compared to nonsurgical root canal therapy.[189] The use of local anesthetics with vasoconstrictors was addressed in the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7).[231] An updated version of this report (JNC 8) was issued in 2013. In patients with cardiovascular disease, 0.036 to 0.054 mg of epinephrine (approximately two to three cartridges of local anesthetic with 1:100,000 epinephrine) should be safe for most patients except those with severe cardiovascular disease or other specific risk factors.[38,231,594] Local anesthetics with vasoconstrictors should be avoided or used with extreme caution in patients with the following cardiovascular conditions: severe or poorly controlled hypertension, arrhythmias that are refractory to treatment, myocardial infarction within the past month, stroke within the past 6 months, coronary artery bypass graft within the past 3 months, and uncontrolled congestive heart failure.[311] Patients unable to tolerate vasoconstrictors may not be good candidates for periradicular surgery, because a local anesthetic with a vasoconstrictor is essential for obtaining adequate hemostasis and visibility during this type of procedure. Surgery can be performed using only local anesthetics without vasoconstrictors, but this is not recommended.

The potential for multiple drug interactions is increasing because of the aging population and the introduction of many new drugs. Many elderly patients have diminished liver and kidney function and therefore cannot metabolize and excrete drugs as efficiently as younger, healthy patients. The potential for drug interactions and decreased metabolism and excretion of drugs should be considered even for commonly used agents such as local anesthetics and analgesics.

A transient bacteremia is a virtual certainty with periradicular surgery; therefore, patients at high risk for bacterial endocarditis should receive appropriate antibiotic prophylaxis as recommended by the American Heart Association. New guidelines for prevention of infective endocarditis were published in 2007 and represent a significant change from previous American Heart Association guidelines.[588] For example, antibiotic prophylaxis is no longer recommended for patients with a history of mitral valve prolapse (with or without regurgitation), rheumatic heart disease, bicuspid valve disease, aortic stenosis, and certain congenital heart conditions. Antibiotic prophylaxis is now only recommended for patients with valvular disease associated with the highest risk of adverse outcomes from infective endocarditis. For patients in the highest risk category, antibiotic prophylaxis is recommended for dental procedures that involve manipulation of gingival tissues or the periapical region of teeth or perforation of the oral mucosa. For all other patients with valvular disease, the risks associated with routine antibiotic prophylaxis are greater than the potential benefits.[588] Although these guidelines represent the best available current evidence, practitioners should be aware that a recent study from England found a significant increase in cases of infective endocarditis after widespread adoption of the new guidelines in 2008. However, the authors point out that their data do not establish a causal relationship and recommend adherence to the current guidelines at this time (Dayer MJ, et al. Lancet 2014).

New clinical practice guidelines for the management of patients with prosthetic joints were co-developed by the American Association of Orthopaedic Surgeons and the American Dental Association in December 2012.[578] The working group conducted a systematic review and presented three recommendations. Recommendation 1 states that "the practitioner might consider discontinuing the practice of routinely prescribing prophylactic antibiotics for patients with hip and knee prosthetic joint implants undergoing dental procedures." The level of evidence to support this recommendation was classified as "limited," but that was the highest level of evidence assigned to any of the three recommendations. Two case-control studies provide evidence for this recommendation.[51,498] In recommendation 2, the group was unable to recommend for or against the use of topical antimicrobial agents (grade of recommendation = inconclusive). Recommendation 3 was a consensus recommendation to support the maintenance of appropriate oral hygiene (grade of recommendation = consensus). A recent American Dental Association Council on Scientific Affairs report added further clarification by concluding that: "In general, for patients with prosthetic joint implants, prophylactic antibiotics are not recommended prior to dental procedures to prevent prosthetic joint infection." (Sollecito TP, et al. JADA 2015).

Management of patients undergoing anticoagulant therapy depends on the type of anticoagulant, the reason for anticoagulant therapy, and the type of oral surgery planned. Warfarin (Coumadin), a commonly prescribed anticoagulant used to treat or prevent thromboembolisms, blocks the formation of prothrombin and other clotting factors. The international normalized ratio (INR) value is the accepted standard for measuring prothrombin time (PT). The desired therapeutic range for the INR usually is 2 to 3.5, depending on the underlying medical indication for anticoagulant therapy. Limited oral surgery procedures, such as simple forceps extraction of one to three teeth, may be performed safely on patients with INR values within the normal therapeutic range.[8,89,255,478] Periradicular surgery, however, may present a greater challenge for hemostasis, even for patients well maintained within the therapeutic range. The clear field visibility normally required for proper surgical management of the root end may not be possible in patients undergoing anticoagulant therapy. The patient's physician must be consulted for assistance in developing an appropriate treatment plan. Some patients may be able to tolerate discontinuation of warfarin therapy 2 days before a planned surgical procedure, which allows the INR to drift downward.

In a prospective cohort study, Russo and colleagues[461] reported that suspension of warfarin 2 days before a surgical procedure resulted in no bleeding problems and no thromboembolic events. They found that the average time spent at an INR below 2 (critical value) was 28 hours and that 90% of the patients returned to the desired therapeutic INR value within 7 days. However, this strategy may place certain patients at greater risk for a thromboembolic event; therefore, discontinuation of anticoagulant therapy would not be recommended.

In general, patients undergoing anticoagulant therapy should present minimal risk for significant bleeding during or after oral surgery, and local measures to control bleeding should be adequate.[88,569] In a review prepared for the American Dental Association Council on Scientific Affairs and Division of Science, Jeske and Suchko[260] recommended against routine discontinuation of anticoagulant therapy before dental procedures, including surgical procedures. Regardless of the management approach selected, consultation with the patient's physician and an INR test on the day of surgery are strongly recommended. A retrospective study of an urban dental school population found that INR values for 43% of patients taking warfarin were not within the recommended therapeutic range.[266] To underscore the challenge of maintaining anticoagulation levels within an appropriate therapeutic range, warfarin has been implicated in more emergency hospital admissions than any other medication.[83]

Hospitalization and conversion to heparin therapy may be considered in special cases, but the patient, physician, and surgeon must carefully weigh the potential risks against the expected outcome and benefits. A new category of heparin anticoagulant, low-molecular-weight heparins (LMWHs), allows patient self-administration and may be an alternative for patients who need to maintain a high level of anticoagulation but want to reduce the cost and time required for traditional heparin conversion therapy.

Novel oral anticoagulant drugs (NOACs) have recently emerged as an alternative to warfarin for most indications (e.g., previous stroke, atrial fibrillation, and deep vein thrombosis) *except* mechanical heart valves. Examples include: apixaban (Eliquis), dabigratran (Pradaxa), edoxaban (Lixiana), and rivaroxaban (Xarelto). NOACs have fewer interactions with foods and other drugs, and there is no need for routine blood work monitoring. However, although NOACs are known to prolong prothrombin time (PT) by inhibiting factor X to Xa conversion, there is currently no reliable test to accurately measure level of anticoagulation. Medical consultation may be indicated to help evaluate the relative benefits and risks of discontinuing NOAC drugs for one or two days prior to endodontic surgery.

Low-dose aspirin therapy is known to increase bleeding time by irreversibly inhibiting platelet aggregation. A common practice has been to advise patients to discontinue aspirin therapy for 7 to 10 days before oral surgery.[28] At low-dose therapeutic levels (less than 100 mg per day), aspirin may increase bleeding time and complicate surgical procedures. However, Ardekian and colleagues[28] concluded that low-dose aspirin therapy should *not* be discontinued before surgical procedures and that bleeding could be controlled by local measures. Higher-dose therapy may create a greater risk of bleeding during or after surgery.

Although a patient undergoing aspirin therapy may not be at high risk for significant bleeding during or after surgery, a concern in periradicular surgery is the visibility problems created by oozing blood. The clinician should consult the patient's physician about the medical reason for aspirin therapy and should weigh the risks and benefits of discontinuing aspirin before the proposed surgery. It should be possible to perform periradicular surgery without discontinuing aspirin therapy if necessary, but visibility during the procedure may be compromised, which may adversely affect the prognosis.

Herbs, dietary supplements, and vitamins can contribute to bleeding problems during surgery, and patients often fail to report use of these substances in the preoperative evaluation.[385] In a survey of surgical patients, approximately one third reported using a nonprescription medication that might inhibit coagulation or interact with anesthetics.[384] In particular, ginkgo biloba, ginger, garlic, ginseng, feverfew, and vitamin E inhibit platelet aggregation and can increase the risk of bleeding.[96] Ingredients in over-the-counter (OTC) weight loss products can potentiate the effect of epinephrine and increase cardiac stress, although the most obvious example of this phenomenon, ephedra, has been removed from the U.S. market by a Food and Drug Administration (FDA) order.

Patients with inherited or acquired bleeding disorders are also at risk for excessive bleeding during and after periradicular surgery. Impaired liver function associated with past or current alcohol or drug abuse may also predispose a patient to excessive bleeding during surgery. A thorough medical history and patient interview should help identify these patients and, in consultation with the patient's physician, allow for any necessary treatment modifications.

Many other medical conditions, such as recent myocardial infarction, stroke, cardiac arrhythmias, diabetes, radiation therapy for head and neck cancer, immunocompromising conditions, seizure disorders, adrenal suppression, liver or kidney disease, and pregnancy may require modification of the treatment plan. However, these conditions usually present more general problems that are not unique to a surgical procedure. The value of a thorough medical history and patient interview cannot be overemphasized.

ANATOMIC CONSIDERATIONS

Evaluating the access to the surgical site is one of the most important steps in case selection for periradicular surgery. Anatomic studies can provide some guidance, but individual variation is great, and there is no substitute for a complete clinical examination. A small oral opening, active facial muscles, shallow vestibule, and thick buccal alveolar bone all can significantly increase the difficulty of the procedure, even in cases that appear straightforward on radiographic examination.

Posterior Mandible

The primary anatomic structure of concern for periradicular surgery in the posterior mandible is the neurovascular bundle that courses through the mandibular canal and exits through the mental foramen. The relationship among the mandibular canal, the mental foramen, and the root apices of mandibular teeth has been well studied; however, anthropometric averages have limited value in the treatment of individual patients. An understanding of the typical anatomic relationships is important, but even more important is an evaluation of the individual patient to develop a case-specific risk assessment.

The depth of the vestibular fornix generally is a good predictor of the possible difficulty involved in obtaining surgical

FIG. 9-5 Lateral view of the skull showing the anterior nasal spine and the proximity of the maxillary anterior root apices to the floor of the nose *(red arrow),* as well as the typical location of the mental foramen *(black arrow).*

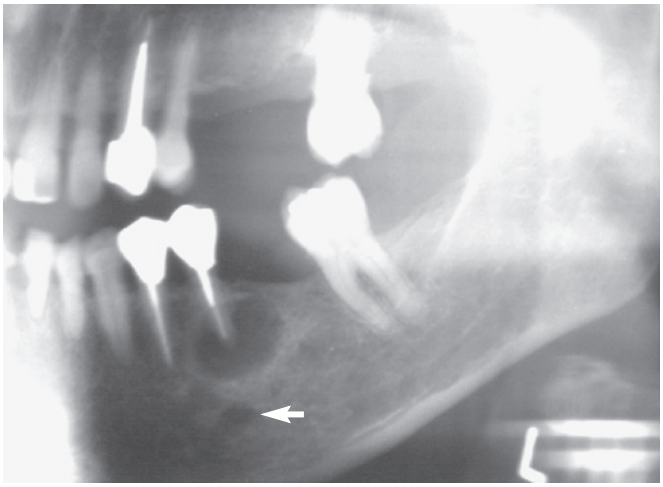

FIG. 9-6 Preoperative evaluation of a mandibular left second premolar included a panoramic radiograph to help locate the mental foramen *(white arrow),* which was not visible on a standard periapical radiograph.

access to the mandibular posterior teeth.[305] A shallow vestibule usually portends thicker alveolar bone and more difficult access to the root end.

The mental foramen, another key anatomic structure, usually is located between and apical to the mandibular first and second premolars[118,356]; however, this varies considerably, and the practitioner must examine each patient carefully to determine its location (Figs. 9-5 and 9-6). Vertical location of the mental foramen may vary even more than horizontal location. Moiseiwitsch[356] found that the average location was 16 mm inferior to the cementoenamel junction (CEJ) of the second premolar, although the range was 8 to 21 mm, which would place approximately 20% of the foramina at or coronal to the root apex. Fortunately, the mental foramen usually is easily visualized with standard periapical and panoramic radiographs. A vertically positioned periapical radiograph often may be more useful than a horizontally positioned one, especially in individuals with longer roots. Also, the mental foramen usually can be palpated.

When a vertical releasing incision is indicated, it typically is made at the mesial line angle of the mandibular canine. This location is always mesial to the mental foramen, because the foramen is located in the area ranging from the apex of the mandibular first premolar to slightly distal to the second premolar. The nerve bundle exits from the foramen in a distal direction. An alternative technique for gaining access to mandibular posterior teeth involves a distal releasing incision between the mandibular first and second molars.[355] This approach may be especially useful for access to the mandibular second premolar and first molar. Care must be taken to avoid the facial artery as it crosses the level of the inferior vestibular fornix near the mandibular first molar. Inadvertent contact with the facial artery is unlikely if the incision is not extended beyond the depth of the vestibule.

Regardless of the approach selected, it obviously is important to avoid making an incision in the immediate vicinity of the mental foramen. The mental nerve is encased in a relatively tough sheath, and permanent damage can be avoided if careful blunt dissection is used in the area. A misguided vertical releasing incision, however, may sever the nerve, causing permanent injury. Trauma to the nerve from blunt dissection in this area or from pressure from a misplaced retractor may cause temporary paresthesia but is much less likely to do permanent injury. (Documentation and management of nerve injuries are discussed later in the chapter.)

The borders of the mandibular canal often are more difficult to visualize with conventional radiographic techniques. A parallel periapical radiograph, either horizontally or vertically positioned, usually can provide a reasonably accurate image of the relationship between the superior border of the mandibular canal and the root apices. However, the mandibular canal sometimes cannot be readily visualized. Such cases should be approached with extreme caution, because an increased risk of paresthesia secondary to nerve injury may be an unacceptable risk for many patients. Cone-beam computed tomography (CBCT) imaging can be very useful for identifying the location of the mandibular canal and determining its relationship to the root apices (see Chapter 2).[68,285,556]

In the buccolingual dimension, the mandibular canal commonly follows a curving pathway from the buccal half of the mandible near the distal root of the second molar to the lingual half of the mandible near the first molar, then curving back to the buccal near the second premolar as it exits the mental foramen.[136] The average vertical distance from the superior border of the mandibular canal to the distal root apex of the mandibular second molar is approximately 3.5 mm; this increases gradually to approximately 6.2 mm for the mesial root of the mandibular first molar and to 4.7 mm for the second premolar.[136,312] This relationship typically provides a greater margin of safety for surgery on the mandibular first molar compared with the second premolar and especially the second molar. Surgery on the mandibular second molar may be further complicated by relatively thick overlying buccal bone, lingual inclination of the roots, and a more buccal location of the mandibular canal. This is not to say that periradicular surgery should not be performed on mandibular second molars; rather, it points out that the relative risks and benefits must be carefully considered. Often the most prudent choice for a mandibular second molar is an intentional replantation procedure or extraction and placement of an implant.

Posterior Maxilla

The primary anatomic structure of concern in the posterior maxilla is the maxillary sinus. CBCT allows for a more precise preoperative three-dimensional evaluation of the relationship between the roots of maxillary posterior teeth and the sinus.[69,79] Perforation of the sinus during surgery is fairly common, with a reported incidence of about 10% to 50% of cases.[157,177,456] Even without periradicular pathosis, the distance between the root apices of the maxillary posterior teeth and the maxillary sinus sometimes is less than 1 mm.[225] An inflammatory periradicular lesion often increases the likelihood of sinus exposure during surgery. Fortunately, perforation of the maxillary sinus rarely results in long-term postoperative problems.[225] In a report of 146 cases of sinus exposure during periradicular surgery, Watzek and colleagues[579] found no difference in healing compared with similar surgical procedures without sinus exposure. The sinus membrane usually regenerates, and a thin layer of new bone often forms over the root end, although osseous regeneration is less predictable.[50,157,579] The general rule of placing a vertical releasing incision at least one tooth mesial and distal to the surgical site is especially important when sinus perforation is possible, because the exposure site should be completely covered with the mucoperiosteal flap to provide primary closure.

If the maxillary sinus is entered during surgery, special care must be taken to prevent infected root fragments and debris from entering the sinus. The most commonly used root resection technique involves grinding the root apex with a high-speed drill for approximately 3 mm in an apical to coronal direction; therefore, an opening could allow infected debris into the sinus. A sinus opening can be temporarily occluded with a material such as Telfa gauze, although the gauze should be secured so as to prevent inadvertent displacement into the sinus. A suture can be placed through the packing material to prevent displacement and aid retrieval. Jerome and Hill[259] suggested securing the apical root segment by drilling a small hole in the root tip and threading a piece of suture material through the hole. The root is then resected at the appropriate level, and the root-end fragment is removed in one piece. If a root fragment or other foreign object is displaced into the sinus, it should be removed. An orascope or endoscope may be useful for visualizing the foreign object, but referral for evaluation and surgical removal may be indicated if the fragment cannot be located and removed.

The palatal roots of maxillary molars present a special challenge for surgical access. Palatal roots may be reached from either a buccal (transantral) or palatal approach. Wallace[572] described a transantral approach in which a buccal flap is reflected, the buccal roots are resected, the osteotomy access into the sinus is enlarged to approximately 1 by 1.5 cm, and the palatal root tip is resected, ultrasonically prepared, and filled. The sinus may be packed with moist gauze to catch debris, and it should be irrigated with sterile saline upon completion of the surgery. In some cases, careful reflection and retraction of the sinus membrane may be possible, allowing access to the palatal root end without direct invasion of the sinus.[16] Many who have tried this approach find it more challenging than it might initially seem. The enhanced illumination and magnification provided by a dental operating microscope, endoscope, or orascope are essential aids in this type of surgery.[40]

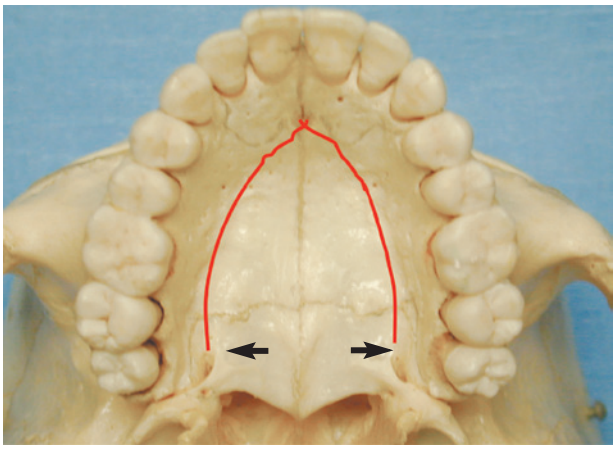

FIG. 9-7 Palatal view showing the position of the greater palatine foramen *(arrows)*. The approximate location of the anterior palatine artery is marked in red.

A palatal approach to the palatal root of maxillary molars may seem more direct than a transantral approach, but it can present certain difficulties. Visibility in the surgical field is reduced and manipulation of instruments is more difficult than with most routine buccal approaches. Patients with a deep, vertical palatal vault are better candidates for this approach than individuals with a wide, shallow palate. A sinus tract or a large lesion on the palatal root may allow easier access and better visualization of the palatal root, because only limited bone removal would be required. The position of the anterior palatine artery must be carefully considered when the incision is made and the flap reflected. This artery emerges from the greater palatine foramen distal to the maxillary second molar at the junction of the vertical section of the alveolar process and the flat portion of the palate and continues anteriorly (Fig. 9-7). A vertical releasing incision can be placed between the maxillary first premolar and canine, where the artery is relatively narrow and branches off into smaller arteries. If needed, a short distal vertical releasing incision may be made distal to the second molar, but it should not approach the junction of the alveolar process and roof of the palate. If the anterior palatine artery is severed, local clamping and pressure may not stop the hemorrhage, and ligation of the external carotid artery may be necessary. Because of the palatal vault's concave shape, repositioning the flap may be challenging. An acrylic surgical stent may be fabricated before surgery to assist repositioning of the flap and to help prevent pooling of blood under the flap.

Anterior Maxilla and Mandible

Periradicular surgery on anterior teeth generally involves fewer anatomic hazards and potential complications than in posterior teeth. Nonetheless, access to the root apex in some patients may be unexpectedly difficult because of long roots, a shallow vestibule, or lingual inclination of the roots. As shown in Figure 9-5, the root apices of maxillary central and lateral incisors can be quite close to the floor of the nose and the bony anterior nasal spine. Figure 9-8 demonstrates isolation and protection of the nasopalatine neurovascular bundle during maxillary anterior surgery. The average maxillary canine is about 26 mm long and usually presents no difficulty for surgical access; however, the combination of a shallow vestibule and

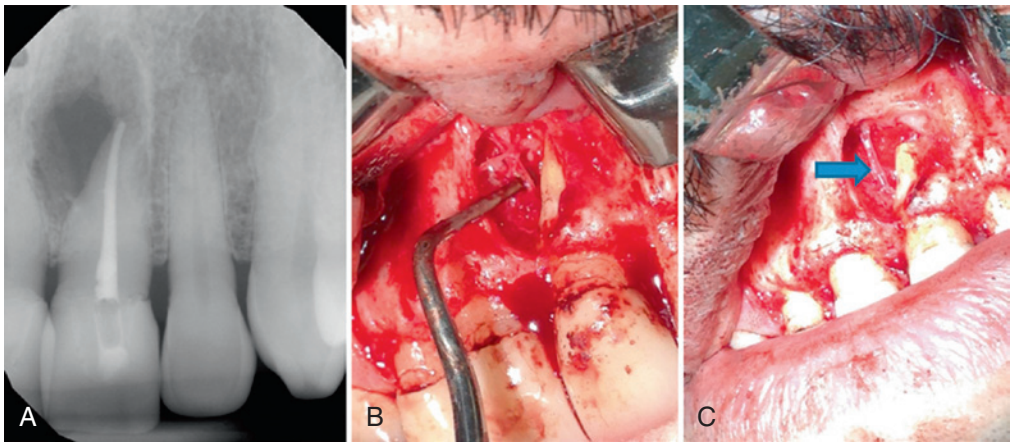

FIG. 9-8 **A**, Radiograph demonstrating periradicular radiolucency associated with previously treated tooth #9. **B-C**, After full thickness flap reflection, the nasopalatine neurovascular bundle was identified (**B**), protected, and isolated (*arrow* in **C**) during curettage of cystic lesion. (Courtesy Dr. Tim Rogers.)

a longer than average root length could complicate access to the root apex area. In such cases, the creation of an osteotomy apical to the root end may be impossible. An alternative approach for long-rooted teeth and root tips in the vicinity of critical anatomic structures is to enter the bone and resect the root at a level approximately 3 mm coronal to the apex. After the root tip is removed, the area apical to the root can be inspected and curetted as needed.

Periradicular surgery on mandibular incisors often is more challenging than expected. The combination of lingual root inclination, a shallow vestibule, and a prominent mental protuberance all can increase the degree of difficulty, as can proximity to adjacent roots and the need for perpendicular root-end resection and preparation to include a possible missed lingual canal.

CONE-BEAM COMPUTED TOMOGRAPHY

Radiographic examination is an essential component in all aspects of endodontic treatment from diagnosis and treatment planning to assessing outcome (see also Chapter 2). Information gained from conventional films and digital periapical radiographs is limited by the fact that the three-dimensional anatomy of the area is compressed into a two-dimensional image. As a result of superimposition, periapical radiographs reveal limited aspects of the three-dimensional anatomy. In addition, there may also be geometric distortion of the anatomic structures imaged. These problems may be overcome using small volume cone-beam computed tomography (CBCT) imaging techniques, which can produce three-dimensional images of individual teeth and the surrounding tissues. CBCT may be particularly useful in diagnosis and treatment planning for periradicular surgery[112,163,249,318] (Fig. 9-9). The term *cone-beam computed tomography* is often used interchangeably with *cone-beam volumetric tomography* (CBVT).

Differences Between CT and CBCT Imaging

The benefits of three-dimensional medical computer tomography (CT) imaging are already well established in certain dental specialties. Current CT scanners have a linear array of multiple detectors, allowing multiple slices to be taken simultaneously, resulting in faster scan times and often less radiation exposure to the patient.[507] The slices of data are then "stacked" and can be reformatted to obtain three-dimensional images. The high-radiation dose, cost, availability, poor resolution, and difficulty in interpretation have resulted in limited use of CT imaging in endodontics. These issues may be addressed by cone-beam innovations in CBCT technology.

In 2000, the Food and Drug Administration approved the first CBCT unit for dental use in the United States. Cone-beam technology uses a cone-shaped beam of radiation to acquire a volume in a single 360-degree rotation, similar to panoramic radiography. The volume of acquired images by a CBCT is composed of voxels. Essentially, a voxel is a 3D pixel. Because the data are captured in a volume as opposed to slices, all the voxels are isotropic, which enables objects within the volume to be accurately measured in different directions. Unlike the CBCT voxel, a medical CT voxel is not a perfect cube, and measurements made in multiple planes are not accurate. In addition to increased accuracy and higher resolution, CBCT offers significant scan-time reduction, radiation dose reduction, and reduced cost for the patient.[472,595,606] CBCT systems can be classified into two categories: limited (dental or regional) CBCT or full (ortho or facial) CBCT. The field of view (FOV) of limited CBCT ranges in diameter from 40 to 100 mm, whereas the FOV of full CBCT ranges from 100 to 200 mm.[112] Another difference between the limited CBCT and full CBCT is that a voxel is generally smaller for the limited version (0.1 to 0.2 mm versus 0.3 to 0.4 mm). Thus, limited CBCT systems offer higher resolution and are better suited for endodontic applications. Limited volume CBCT scanners capture small volumes of data that can include just two or three individual teeth. The most important and clinically useful feature of CBCT technology is the highly sophisticated software that allows the huge volume of data collected to be reconstructed. Tomographic slices, as thin as one voxel, may be displayed in a number of ways. One option, for example, is for the images to be displayed in the three orthogonal planes—axial, sagittal and coronal—simultaneously. The axial and proximal (sagittal in the anterior, coronal in the posterior) views are of particular value, because they are generally not seen with conventional periapical radiography. The ability to reduce or eliminate

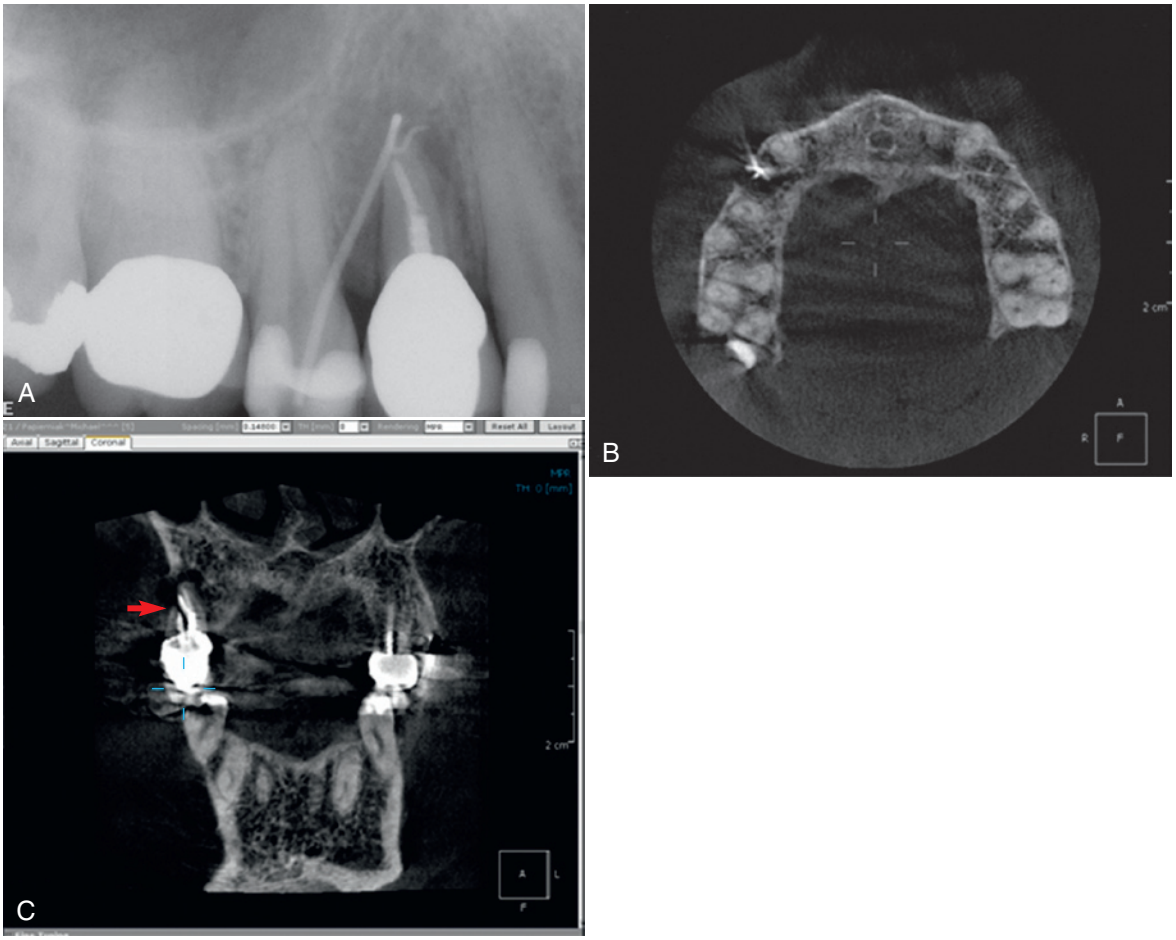

FIG. 9-9 A, Periapical radiograph with gutta percha tracing the sinus tract to tooth #5. Periodontal probings were WNL. This tooth was initially referred for exploratory surgery to rule out root fracture. CBCT confirmed root fracture and the treatment plan was changed to extraction with possible bone graft and guided tissue regeneration for ridge augmentation prior to implant placement. B, Axial CBCT view demonstrating extent of lesion. C, Coronal CBCT view demonstrating root fracture *(red arrow)* and perforation of buccal cortical plate in area of root fracture but intact bone in the cervical area.

superimposition of the surrounding structures makes CBCT superior to conventional periapical radiography.[314]

Scan times are typically 10 to 40 s, although the actual exposure time is significantly less (2 to 5 s) as scans involve a number (up to 360) of separate, small, individual exposures rather than one continuous exposure. With medical CT scanners, the scanning and exposure times for the skull can be significantly longer. Most CBCT scanners are much smaller than medical CT scanners, taking up about the same space as a dental panoramic machine. They are also significantly less expensive than medical CT scanners. With the help of viewer software, the clinician is able to scroll through the entire volume and simultaneously view axial, coronal, and sagittal 2D sections that range from 0.125 to 2 mm thick. Comparing the radiation dose of different CBCT scanners with medical CT scanners may be confusing because different units of radiation dose are often used. There are three basic measurement units in radiation dosimetry: the radiation absorbed dose (D), the equivalent dose (H), and the effective dose (E). The *radiation absorbed dose* is defined as the measure of the amount of energy absorbed from the radiation beam per unit mass of tissue and is measured in joules per kilogram. The *equivalent dose* is defined as a measure that indicates the radiobiologic effectiveness of different types of radiation and thus provides a common unit. The *effective dose* is calculated by multiplying the equivalent dose by different tissue weighting factors, which converts all doses to an equivalent whole-body dose and allows doses from different investigations of different parts of the body to be compared. The unit remains as the Sievert (Sv) and can be used to estimate the damage from radiation to an exposed population. The reported effective doses from CBCT scanners vary but can be almost as low as those from panoramic dental x-ray units and considerably less than that from medical CT scanners.[398] The higher effective doses from particular models of CBCT scanners is in part due to the larger size of the field of view used as well as the type of image receptor employed. The small volume scanners capture information from a small region of the jaw, approximately the same area as a periapical radiograph. The effective dose has been reported to be in the same range as 2 to 3 standard periapical radiograph exposures, whereas the effective dose for a full mouth series of periapical radiographs has been reported to be similar to the effective dose of a large volume CBCT scan.[124,194] If multiple teeth in different quadrants require endodontic evaluation or

treatment, a large volume CBCT scan with a large field of view may be more appropriate. If endodontic information is required from multiple teeth in one jaw, a large volume CBCT scan with the field of view only limited to the jaw of interest could be the investigation of choice. This has the advantage of reducing the effective dosage from the full large volume CBCT scan by up to 65%.[321]

Potential Applications of CBCT in the Management of Endodontic Posttreatment Disease

Clinical radiographic examinations are usually limited to two-dimensional views captured using radiographic film or digital sensors. Crucial information related to the true three-dimensional anatomy of teeth and adjacent structures is obscured. Even with paralleling techniques, distortion and superimposition of dental structures in periapical views is unavoidable. A major reported advantage of CBCT is the accuracy of measurements in all dimensions.[331] The ability to view thin sagittal, coronal, and axial slices effectively eliminates the problem of superimposition of anatomic structures. For example, roots of maxillary posterior teeth and surrounding tissues can be viewed without superimposition of the zygomatic buttress, alveolar bone, maxillary sinus, and other roots (Fig. 9-10). CBCT enables clinicians to detect changes in apical bone density at an earlier stage when compared to conventional periapical radiographs and therefore has the potential to detect previously undiagnosed periradicular pathoses.[163,314,371] CBCT may also prove to be a useful tool for noninvasive differentiation of periapical cysts and granulomas,[489] a previously unattainable goal using conventional radiographic imaging.

Three-dimensional imaging allows for clear identification of the anatomic relationship of the root apices to important neighboring anatomic structures such as the mandibular canal, mental foramen, and maxillary sinus (Fig. 9-11). Velvart and colleagues reported that the relationship of the inferior alveolar canal to the root apices could be determined in every case when utilizing medical CT, but in less than 40% of cases when using conventional radiography.[556] It is likely that similar results could be achieved with CBCT using considerably less radiation. Rigolone and coworkers concluded that CBCT may play an important role in periapical microsurgery of palatal roots of maxillary first molars.[442] The distance between the cortical plate and the palatal root apex could be measured using CBCT and the presence of the maxillary sinus between the roots could be assessed. Additional information such as the thickness of the cortical plate, cancellous bone pattern, fenestrations, and inclination of the roots can be obtained prior to surgical entry.[371] Root morphology (shape, size, curvature, and number of canals) can be visualized in three dimensions. Unidentified (and untreated) canals in root-filled teeth can be routinely visualized in axial slices. CBCT has been used to determine the location and extent of invasive external root resorptive defects.[398]

One of the most useful potential applications of CBCT may be the assessment of endodontic treatment outcomes, both nonsurgical and surgical. When compared to two-dimensional film and digital images, CBCT is dimensionally precise and can detect smaller changes in bone density.[331]

It is worth remembering that CBCT still uses ionizing radiation and is not without risk. It is essential that patient exposure is kept as low as reasonably practical and that justifiable selection criteria for CBCT use are developed. CBCT may be indicated when it is determined that the additional information obtained is likely to result in a more accurate diagnosis and enhanced patient safety.

PATIENT PREPARATION FOR SURGERY

Informed-Consent Issues Specific to Surgery

The general principles of informed consent discussed in Chapter 29 form the basis for informed consent for periradicular surgery. The patient must be thoroughly advised of the benefits, risks, and other treatment options and must be given an opportunity to ask questions. The main consent issues specific to surgical procedures are closely related to the anatomic considerations discussed in the previous section. That is, major neurovascular bundles may be traumatized, and a sinus exposure may occur. Paresthesia after mandibular posterior surgery is uncommon but should be discussed with the patient because this potential complication is a risk some patients may be unwilling to assume. Postoperative swelling, bruising, bleeding, and infections are possible complications that typically are self-limiting or readily manageable. Although the incidence of serious complications related to surgical procedures is very low, patients should be advised of any risks unique to their situation. Prompt attention to any surgical complications and thorough follow-up are essential from a medical-legal standpoint.

Premedication: NSAIDs, Antibiotics, Chlorhexidine, and Conscious Sedation

Administration of an NSAID, either before or up to 30 minutes after surgery, enhances postoperative analgesia.[494] NSAIDs generally have proved more effective in the management of postoperative oral surgery pain than placebo or acetaminophen and codeine combinations.[9,41,143] The combination of preoperative administration of an NSAID and use of a long-acting local anesthetic may be particularly helpful for reducing postoperative pain.[144] Many types of NSAIDs are available, but ibuprofen remains the usual standard for comparison. Ibuprofen (400 mg) provides analgesia approximately equal to that obtained with morphine (10 mg) and significantly greater than that from codeine (60 mg), tramadol (100 mg), or acetaminophen (1000 mg).[346] The analgesic effectiveness of ibuprofen tends to level off at about the 400-mg level *(ceiling effect),* although a slight increase in analgesic potential may be expected in doses up to 800 mg. (See Chapter 4 for further discussion of analgesics.)

The value of antibiotic prophylaxis before or after oral surgery is controversial, and the current best available evidence does not support the routine use of prophylactic antibiotics for periradicular surgery.[11] For most patients, the risks of indiscriminate antibiotic therapy are believed to be greater than the potential benefits.[526] The incidence of infection after oral surgery in healthy patients is very low. Peterson reported that only 1% of patients developed infections after third molar extractions.[355] A systematic review of the use of antibiotics to prevent complications after placement of dental implants found that evidence was lacking either to recommend or discourage the use of antibiotics for this purpose.[162] However, the

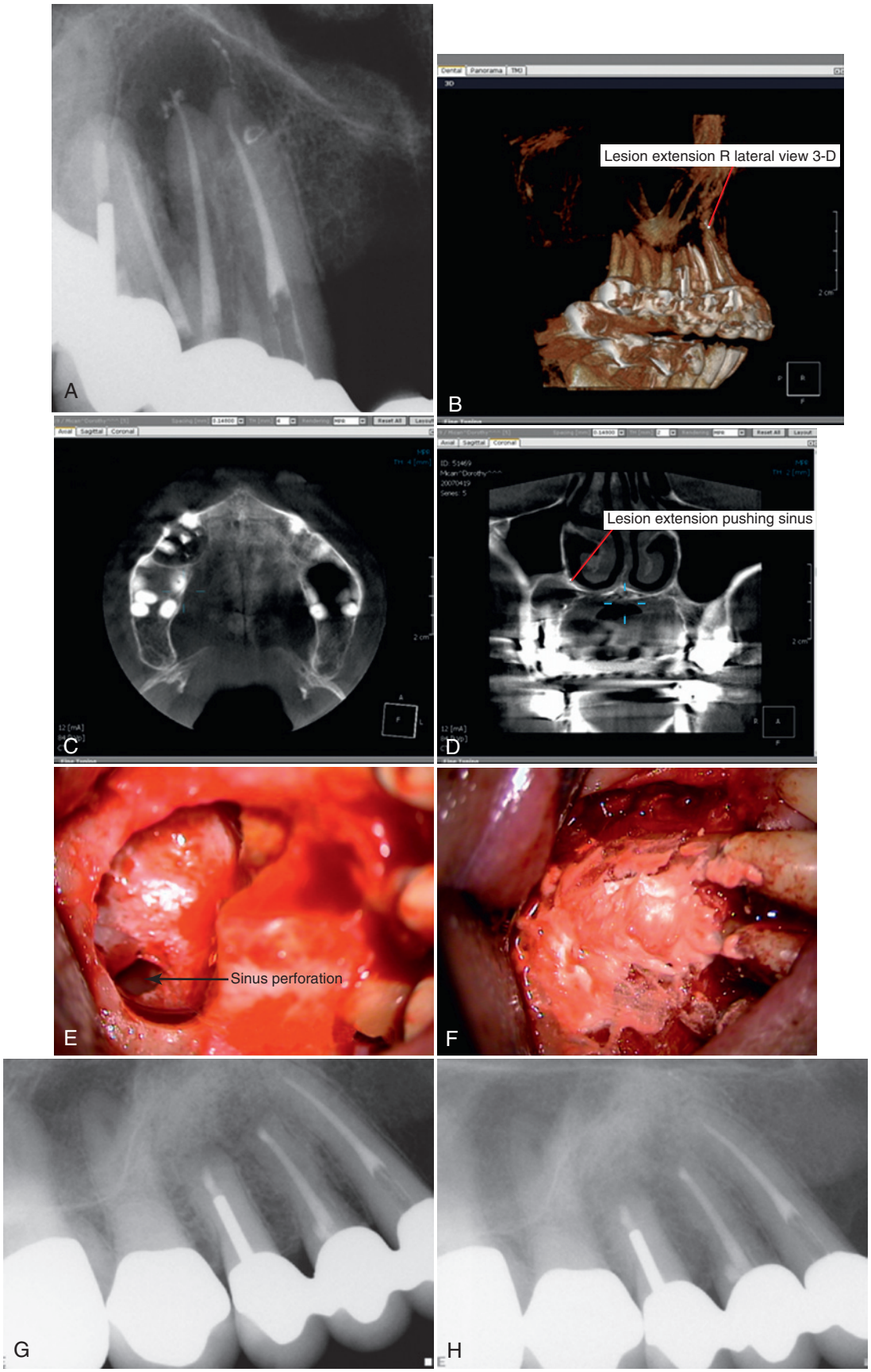

FIG. 9-10 **A**, Preoperative periapical radiograph showing large radiolucency associated with teeth #4, 5, and 6. **B**, CBCT reconstruction *(lateral view).* Rotation of the 3D volume on a computer monitor demonstrated that intact bone was present around the apex of tooth #6 and the lesion was associated only with #4 and 5 (this could not be detected with multiple angled periapical radiographs). **C**, Axial CBCT view demonstrating facial-palatal extent of the lesion. **D**, Coronal CBCT view showing displacement of the sinus membrane (a finding that suggests a sinus membrane perforation will be detected during surgery). **E**, Clinical image after surgical access removal of granulation tissue, and root-end resection (note sinus perforation [*arrow*]). **F**, Placement of bone graft and Capset. **G**, Immediate postoperative radiograph. **H**, Two-year follow-up radiograph demonstrating good healing.

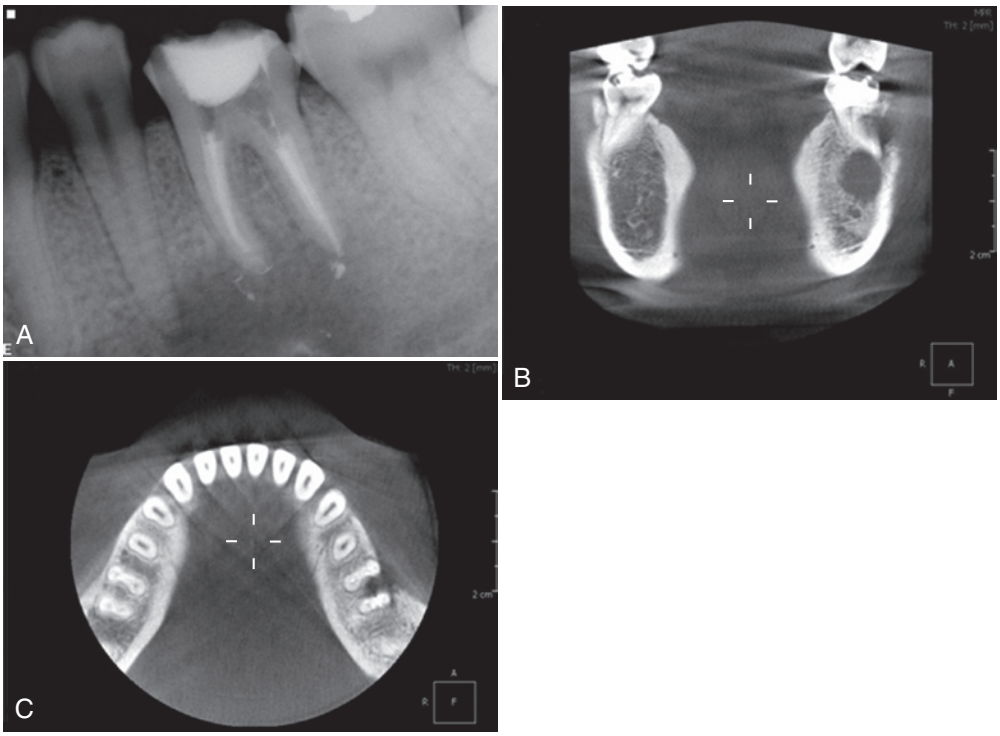

FIG. 9-11 A, Periapical radiograph demonstrating persistent periradicular pathosis following nonsurgical retreatment of tooth #19. The extension of the lesion in the vicinity of the mandibular canal and possible involvement of tooth #20 was uncertain based on interpretation of periapical and panoramic radiographs. Tooth #20 responded WNL to pulp vitality test. B, Coronal CBCT demonstrates that the periradicular lesion does not extend apical or lingual to the mandibular canal. C, Axial CBCT view demonstrating perforation of the buccal cortical plate and extension into, but not through, the furcation of #19 and presence of intact bone around #20.

use of prophylactic antibiotics for more invasive procedures, such as orthognathic surgery, has significantly reduced the risk of postoperative infection and complications.[146] Although routine use of prophylactic antibiotics for periradicular surgery is not currently recommended, clinical judgment is important in determining exceptions to the general rule. For example, immunocompromised patients may be good candidates for prophylactic antibiotic coverage. Certain categories of medically complex patients also may benefit from antibiotic coverage. Diabetic patients have shown impaired healing capacity after nonsurgical root canal treatment,[78,175] and a like pattern of delayed or impaired healing may emerge in studies of surgical outcomes. The global problems associated with overprescription of antibiotics are significant and should prompt due caution in the decision on whether to use these drugs prophylactically.

Chlorhexidine gluconate (0.12%) often is recommended as a mouth rinse to reduce the number of surface microorganisms in the surgical field, and its use may be continued during the postoperative healing stage.[7,281,545] Although no solid evidence supports this practice in periradicular surgery, the use of chlorhexidine follows from the general surgical principle of surface disinfection before incision and opening into a body cavity. In addition, chlorhexidine has proved to be a safe, effective adjunct in the treatment of periodontitis, and short-term use (i.e., several days) poses little or no risk. Chlorhexidine may be useful for reducing the risk of postoperative infection after oral surgery,[67,598] although the evidence in this area is conflicting. Postoperative use of chlorhexidine mouth rinse can reduce bacterial growth on sutures and wound margins,[389] but it may interfere with fibroblast reattachment to the root surface.[14] A useful empirical regimen is to have the patient rinse for 30 seconds twice a day beginning 1 or 2 days before surgery and continuing until the sutures are removed.

Conscious sedation, either by an orally administered sedative or by nitrous oxide/oxygen inhalation analgesia, may be useful for patients who are anxious about the surgical procedure or dental treatment in general. Benzodiazepines with a short half-life are particularly useful because they generally have a wide margin of safety, good absorption after oral administration, and limited residual sedative effects. When these drugs are used in sedative-hypnotic doses, the blood pressure, pulse, and respiration must be monitored, and states differ in their requirement for additional training or certification of staff members who do this monitoring. With oral conscious sedation, pulse oximetry should be used to monitor the pulse and blood oxygen saturation during the surgery.[84] As with all orally administered drugs, the dosage cannot be closely titrated; therefore, the effect of the agent varies somewhat. A typical protocol is a single dose at bedtime the evening before the procedure and a second dose 1 hour before the start of surgery. The patient should not drive to or from the office and should have a responsible adult for assistance as needed. In appropriate doses, benzodiazepines and similar drugs may allow for a more relaxed patient and thus a less stressful surgical experience for both patient and surgeon.

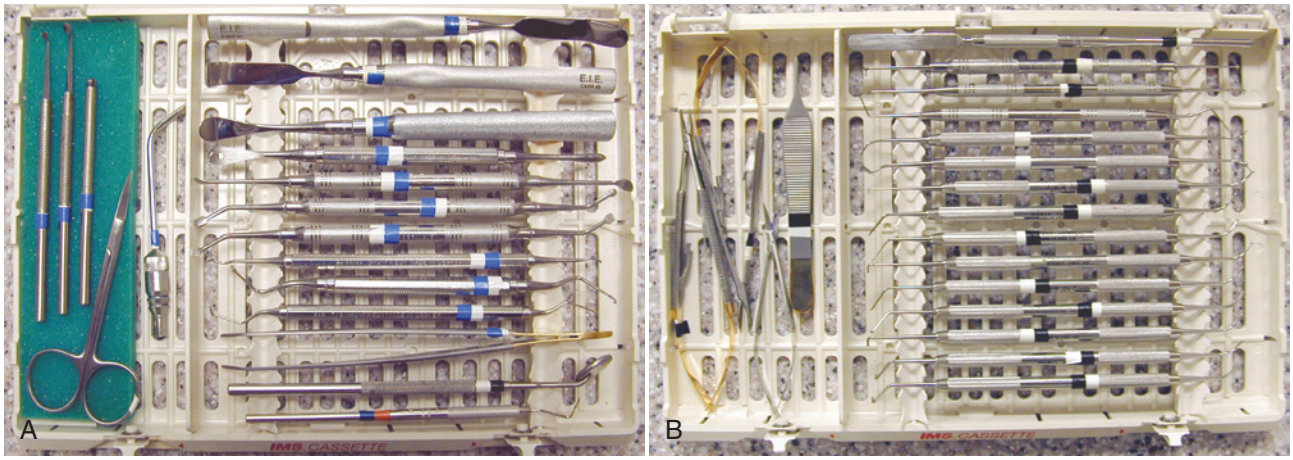

FIG. 9-12 A, Basic tray setup for initial surgical access. Surgical instruments shown are distributed by Hu-Friedy ([HF] Chicago), CK Dental Specialities ([CKDS] Orange, CA), EIE ([EIE]), and G. Hartzell & Son ([GHS]). *Left to right* (left section of tray): Small round micromirror (CKDS); medium oval micromirror (CKDS); handle for microscalpel (CKDS); scissors (S18 [HF]); surgical suction tip (GHS). *Top to bottom* (main section of tray): Carr #1 retractor (EIE); Carr #2 retractor (EIE); TRH-1 retractor (HF); periosteal elevator (HF); Ruddle R elevator (EIE); Ruddle L elevator (EIE); Jacquette curette (SJ 34/35 [HF]); spoon curette (CL 84 [HF]); scaler (7/8 [HF]); surgical forceps (TP 5061 [HF]); mouth mirror (HF); periodontal probe (HF). B, Instrument tray for root-end filling and suturing. *Left to right* (left section of tray): Two Castroviejo needle holders (Roydent Dental Products, Rochester Hills, MI); Castroviejo scissors (S31 [HF]); micro–tissue forceps (TP 5042 [HF]). *Top to bottom* (main section of tray): Cement spatula (HF); Feinstein super plugger (F1L); microexplorer (CX-1 [EIE]); endoexplorer (DG-16 [EIE]); right Super-EBA Placing & Plugging instrument (MRFR [HF]); left Super-EBA Placing & Plugging instrument (MRFL [HF]); small anterior microburnisher and plugger (HF); small left microburnisher and plugger (HF); small right microburnisher and plugger (HF); medium anterior microburnisher and plugger (HF); medium left microburnisher and plugger (HF); medium right microburnisher and plugger (HF); large anterior microburnisher and plugger (HF); large left microburnisher and plugger (HF); large right microburnisher and plugger (HF).

INSTRUMENTS AND OPERATORY SETUP

The development of microsurgical techniques and new materials has changed the typical surgical instrument tray dramatically. Instruments have been designed to take full advantage of the increased visibility obtained with dental operating microscopes, endoscopes, and orascopes. Better visualization of the surgical site would have limited value without microsurgical instruments, such as ultrasonic tips for root-end preparation and micromirrors for inspecting the root end. Figure 9-12 shows a typical basic surgery tray arrangement. This setup is not a definitive guide to surgical armamentarium but rather an adequate, efficient starting point for most periradicular surgical procedures. Although the number of instruments can easily be doubled or even tripled, the ease of locating a specific instrument is inversely proportional to the number of instruments on a tray. Specialized instruments can be kept readily available in separate sterilized bags or trays and opened as needed. A skilled surgeon can use a wide variety of instruments (Figs. 9-12 to 9-24) to achieve excellent results.

Periradicular surgery can be performed without the benefit of enhanced magnification and illumination; however, those who use microscopes, endoscopes, and orascopes report dramatically improved visualization and control of the surgical site.[39,92,280,350] Studies reporting the highest surgical success rates and those cited previously in this chapter all involved some form of enhanced magnification and illumination as part of the standard operating protocol. A systematic review and meta-analysis found that the outcome for endodontic surgery performed with high magnification and illumination (microscope or endoscope) was significantly higher than surgery performed with loupes only or no magnification.[482]

FIG. 9-13 Comparison of microsurgical scalpel *(top)* to #15C surgical blade. Microsurgical scalpels are particularly useful for the intrasulcular incision and for delicate dissection of the interproximal papillae.

LOCAL ANESTHESIA FOR SURGERY

Local anesthesia for surgical root canal procedures differs from that for nonsurgical root canal treatment primarily in the need

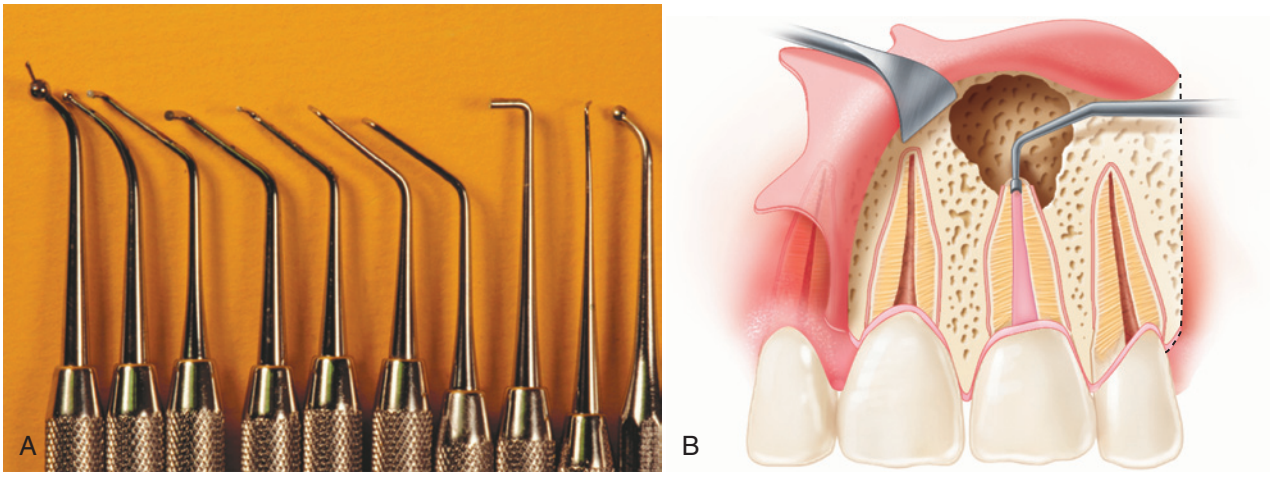

FIG. 9-14 A, Microcondensers in assorted shapes and sizes for root-end filling. B, The microcondenser should be selected to fit the root-end preparation.

FIG. 9-15 Comparison of standard #5 mouth mirror *(top)* to diamond-coated micromirrors (CK Dental Specialties).

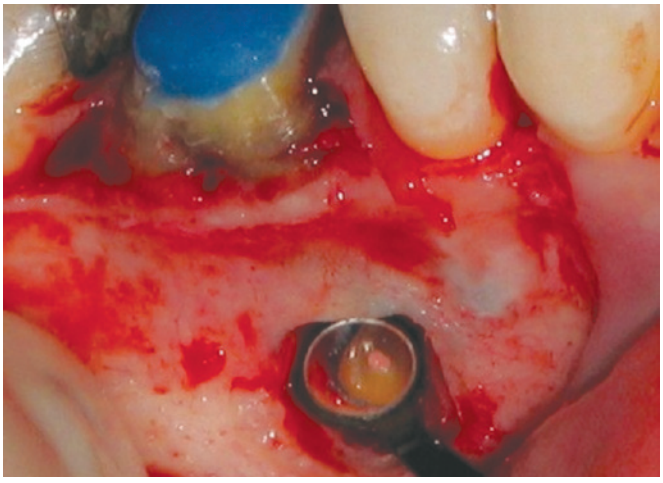

FIG. 9-16 Micromirror used to inspect resected mesial root of a mandibular first molar.

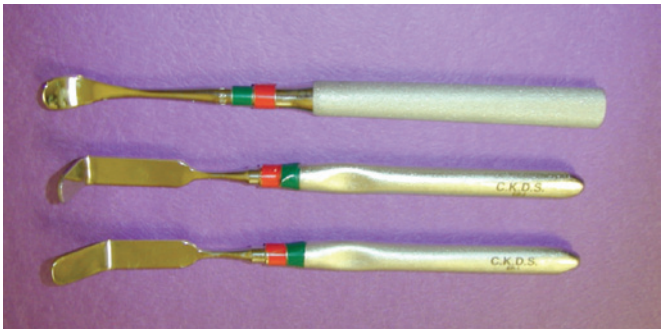

FIG. 9-17 Retractors used in periradicular surgery. *Top to bottom,* EHR-1, ER-2, and ER-1 (equivalent to Carr #2 and #1 retractors) (CK Dental Specialties).

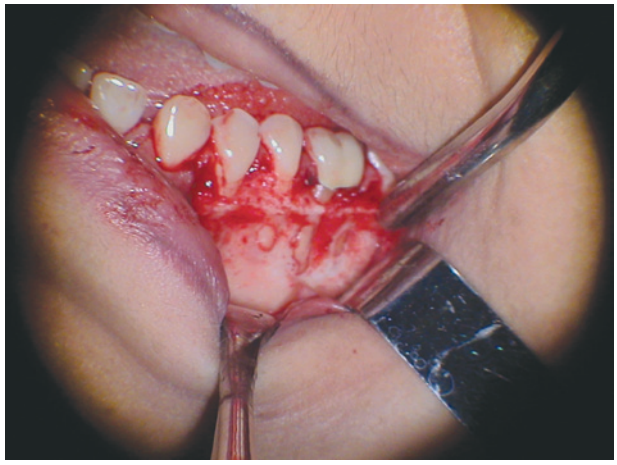

FIG. 9-18 Retractors positioned to expose the surgical site and protect adjacent soft tissues from injury. Care must be taken to rest the retractors only on bone, not on the reflected soft-tissue flap or on the neurovascular bundle as it exits the mental foramen.

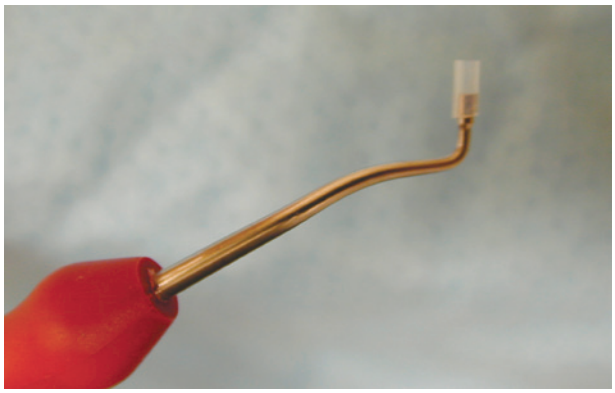

FIG. 9-19 Teflon sleeve and plugger specially designed for placement of MTA (DENTSPLY Tulsa Dental Specialties).

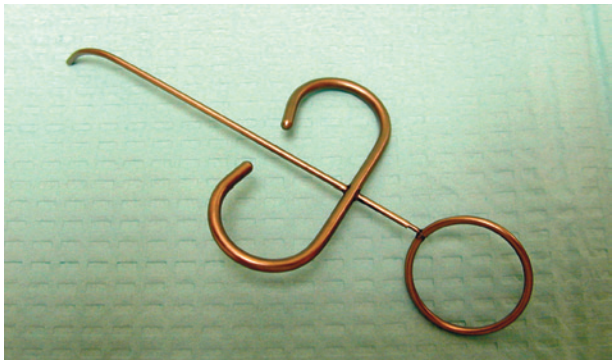

FIG. 9-20 Messing gun–type syringe (CK Dental Specialties) can be used for placement of various root-end filling materials.

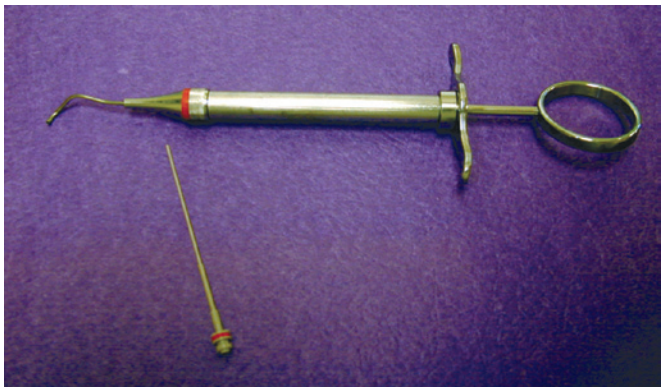

FIG. 9-21 Another delivery system designed specifically for MTA placement (Roydent). Kit includes a variety of tips for use in different areas of the mouth and a single-use Teflon plunger.

for localized hemostasis in addition to profound local anesthesia. In fact, use of a local anesthetic with a vasoconstrictor may be the single most important local measure to help control hemorrhage and provide a clear surgical field. Otherwise, the same regional block and local infiltration techniques used for nonsurgical treatment (see Chapter 4) are used for surgical root canal procedures. Infiltration of the surgical site with a local anesthetic containing 1:50,000 epinephrine is the technique of choice to obtain vasoconstriction and hemostasis.[280] The local anesthetic is first slowly deposited in the buccal root apex

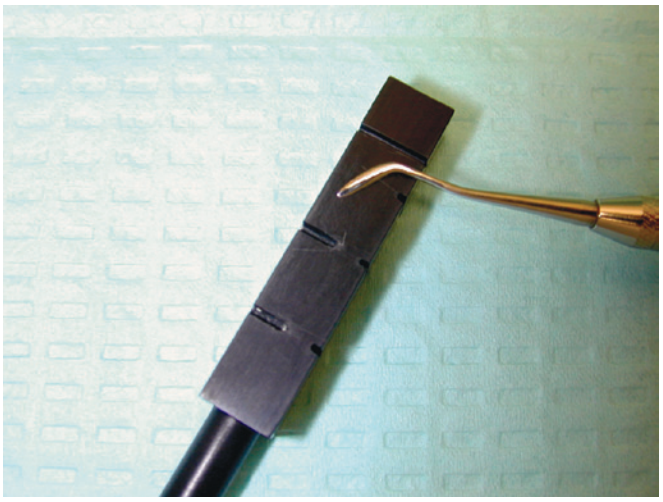

FIG. 9-22 Hard plastic block with notches of varying shapes and sizes (G. Hartzell & Son). MTA is mixed on a glass slab to the consistency of wet sand and then packed into a notch. The applicator instrument is used to transfer the preformed plug of MTA from the block to the root end.

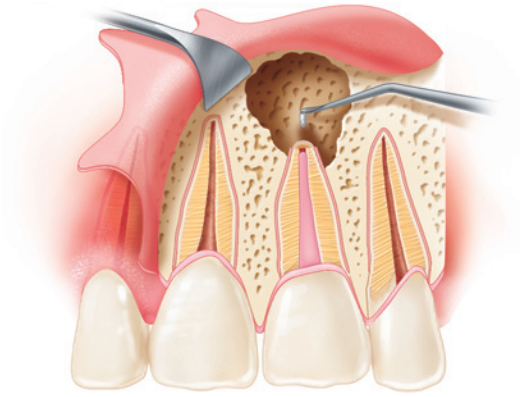

FIG. 9-23 Placement of a root-end filling.

FIG. 9-24 Surgeon, assistant, and patient positioned for initiation of surgery. The patient should be given tinted goggles or some other form of eye protection before the procedure begins.

area of the alveolar mucosa at the surgical site and extended two or three teeth on either side of the site. Usually palatal or lingual infiltration is also required, although this requires a much smaller amount of local anesthetic than the primary buccal infiltration. After the injections for anesthesia, the surgeon should wait at least 10 minutes before making the first incision.

Long-acting local anesthetics (e.g., 0.5% bupivacaine with 1:200,000 epinephrine) have been shown to reduce postoperative pain and analgesic use after surgical removal of impacted third molars.[125,204] However, use of a local anesthetic with 1:200,000 epinephrine may result in greater blood loss during surgery.[234,493] To maximize postoperative analgesia and minimize intraoperative bleeding, a local anesthetic can be used with higher epinephrine concentrations (1:100,000 or 1:50,000) for the primary surgical anesthesia and supplemented with one cartridge of long-acting local anesthetic immediately after surgery. Long-lasting local anesthetics are particularly beneficial in mandibular surgery but much less so for surgery in the maxillary arch.

Every effort must be made to ensure profound local anesthesia before surgery begins. Usually a minimum of 10 to 20 minutes is required from the time of injection to the start of surgery to ensure both profound local anesthesia and adequate vasoconstriction for hemostasis. The patient should be asked about the usual signs of soft-tissue anesthesia, and a sharp explorer can be used to test the surgical area for sensation. Even when the surgeon pays careful attention to local anesthetic technique, a patient sometimes has inadequate anesthesia or loss of anesthesia during the surgical procedure. Providing supplemental infiltration anesthesia is difficult after a full-thickness flap has been reflected. A supplemental block injection may be useful for mandibular teeth and maxillary posterior teeth. In the maxillary anterior area, a palatal approach to the anterior middle superior nerve may be helpful. The key to this approach is slow injection of approximately 1 mL of local anesthetic in the area of the first and second maxillary premolars midway between the gingival crest and the palatal midline. An intraosseous injection also may be used to regain lost anesthesia, but even when it is effective, the area of local anesthesia often is smaller than desired for a surgical procedure. As a last resort, the procedure can be terminated short of completion and the patient can be rescheduled for surgery under sedation or general anesthesia.

SURGICAL ACCESS

The goals of periradicular surgery are to access the affected area, remove the diseased tissue, evaluate the root circumference and root canal system, and place a biocompatible seal in the form of a root-end filling that can stimulate regeneration of the periodontium. The formation of new cementum on the surgically exposed root surface and on the root-end filling material is essential to regeneration of the periodontium.

Successful endodontic surgery requires the surgeon to use several conceptual elements in planning the procedure. A vision of the immediate postoperative surgical end point (i.e., replacement of the reflected tissues) is essential for designing each phase of the surgery. Visualization of a three-dimensional image of the surgical procedure allows the surgeon to anticipate and prepare for unusual circumstances. In surgical root canal treatment, once a procedure is started, it must be completed uninterrupted within a limited time; this is a paramount difference between nonsurgical and surgical root canal treatment. For this reason, it is absolutely essential that the surgeon plan the procedure thoroughly and include alternative plans of action that anticipate unusual findings during the process of discovery.

Several general principles are important for designing the access to a diseased region: (1) the surgeon must have a thorough knowledge of the anatomic structures in relation to each other, including tooth anatomy; (2) the surgeon must be able to visualize the three-dimensional nature of the structures in the soft and hard tissue (this reduces unnecessary tissue damage); (3) the trauma of the surgical procedure itself must be minimized, which includes the preservation of tooth and supporting structures; (4) the tissue and instruments must be manipulated within a limited space with the aim of removing diseased tissues and retaining healthy tissues.

Soft-Tissue Access

In designing the soft-tissue access window to the diseased tissue, the surgeon must take into consideration various anatomic features, such as frenum-muscle attachments, the width of attached gingiva, papillary height and width, bone eminence, and crown margins. The supraperiosteal blood vessels of the attached gingiva extend from the alveolar mucosa and run parallel to the long axis of the teeth, lying in the reticular layer superficial to the periosteum.[172] A vertical, rather than an angled, releasing incision severs fewer vessels,[325] reducing the possibility of hemorrhage. Also, the blood supply to the tissue coronal to the incision is not compromised,[477] which prevents localized ischemia and sloughing of these tissues. Ultimately, the result is less bleeding during the procedure and enhanced healing. For these reasons, an angled releasing incision is contraindicated in periradicular surgery.

Vertical Incision

The general principles for placement of a vertical relieving incision are as follows:

1. The incision should be made parallel to the supraperiosteal vessels in the attached gingiva and submucosa (Fig. 9-25).
2. No cuts should be made across frenum and muscle attachments.
3. Frenum and muscle attachments should not be located in the reflected tissue, if possible.
4. The incision should be placed directly over healthy bone.

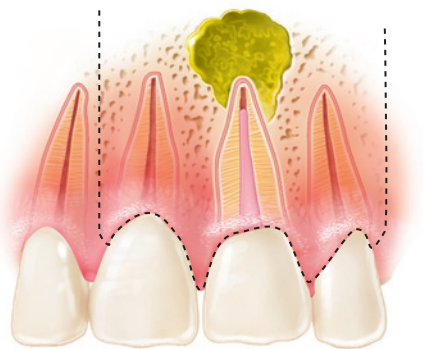

FIG. 9-25 Intrasulcular incision with two vertical releasing incisions (rectangular flap).

5. The incision should not be placed superior to a bony eminence.
6. The dental papilla should be included or excluded but not dissected.
7. The incision should extend from the depth of the vestibular sulcus to the midpoint between the dental papilla and the horizontal aspect of the buccal gingival sulcus.

Horizontal Incision

Three types of horizontal incisions can be used to gain access to a surgical site in hard tissue:

- *An intrasulcular incision that includes the dental papilla.* This incision extends from the gingival sulcus through the PDL fibers and terminates at the crestal bone of the alveolar bone proper. The incision then passes in a buccolingual direction adjacent to each tooth of the dental papilla and includes the midcol region of each dental papilla. Thus, the entire dental papilla is completely mobilized.
- *An intrasulcular incision that excludes the dental papilla (papillary-based incision, Fig. 9-26).* This technique consists of a shallow first incision at the base of the papilla and a second incision directed to the crestal bone.
- *An incision made in the attached gingiva (a submarginal or Ochsenbein-Luebke flap).*[322] With this technique, at least 2 mm of attached gingiva must be retained to prevent mucogingival degeneration.[296] Consequently, the incision must be placed at least 2 mm from the depth of the gingival sulcus. Extensive periodontal probing should be done to establish the depth of the gingival sulcus before the incision is made. The average width of the attached gingiva is 2.1 to 5.1 mm in the maxilla and 1.8 to 3.8 mm in the mandible.[10,199,341,563] It is widest over the central and lateral incisors, narrows over the canine and the first premolar, and then widens over the second premolar and the first molar. These variances were similar for both the maxilla and mandible.[317] Overall, this incision technique has a narrow margin of safety. It generally is recommended for use in the maxilla, especially where the aesthetics of existing crown margins are a concern.

Studies have compared the incision techniques that include and exclude the dental papilla in patients with healthy marginal periodontal conditions.[554,555,557] These researchers found that the papilla base incision resulted in rapid, recession-free healing. In contrast, complete mobilization of the papilla led to a marked loss of papilla height. The authors suggested that use of the papilla base incision in aesthetically sensitive regions could help prevent papilla recession and surgical cleft, or double papilla.

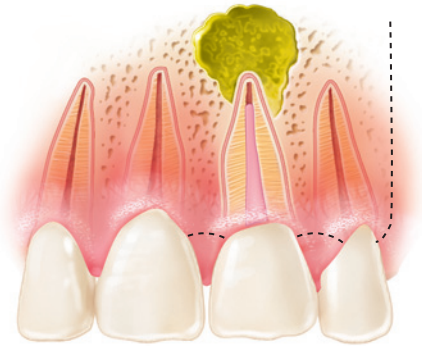

FIG. 9-26 Papillary-based incision with one vertical releasing incision.

Flap Design

Combinations of vertical and horizontal incisions are used to achieve various flap designs (Figs. 9-27 and 9-28). The full mucoperiosteal and limited mucoperiosteal are the two major categories of flap design used during periradicular surgery, the main differentiating feature being the position of the horizontal incision. In each case the entire body of soft tissue is reflected as one unit and includes the alveolar mucosa, the gingival tissues, and the periosteum. The number and position of the vertical relaxing incisions therefore governs the major variation in design:

1. Full mucoperiosteal (intrasulcular incision including the dental papilla or papillary based) (Figs 9-27 and 9-28):
   a. Triangular: one vertical relieving incision
   b. Rectangular: two vertical relieving incisions
   c. Trapezoidal: two angled vertical relieving incisions
   d. Horizontal: no vertical relieving incision
2. Limited mucoperiosteal (Figs 9-29 and 9-30):
   a. Curve submarginal (semilunar)
   b. Freeform rectilinear submarginal (Ochsenbein-Luebke)

Tissue Reflection

Elevation and reflection of the entire mucoperiosteal complex, maintaining the microvasculature in the body of the tissue flap, increases hemostatic control during surgery. Tissue reflection should begin from the vertical releasing incision at the junction

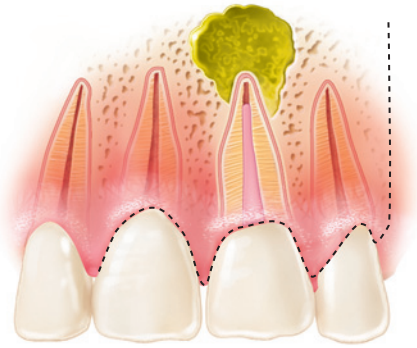

FIG. 9-27 Intrasulcular incision with one vertical releasing incision (triangular flap).

FIG. 9-28 Reflection of the triangular flap to expose the root-end area.

of the submucosa and the attached gingiva (Figs. 9-31 and 9-32). By initiating the reflection process at this point, damage to the delicate supracrestal root-attached fibers is avoided. Using a flap reflection technique that reduces reflective tissue forces in the intrasulcular incisional wound and avoids curettage of the root surface conserves the root-attached tissues and helps prevent apical down-growth of epithelium and loss of soft-tissue attachment.[222] Force should be applied such that the periosteum and superficial tissues are reflected as a complete unit. Using a gentle rocking motion, the surgeon initially should reflect the tissue in a horizontal direction.[213] The underlying bone of the cortical plate is irregular, and it is critical to avoid damaging the fragile tissues during elevation.

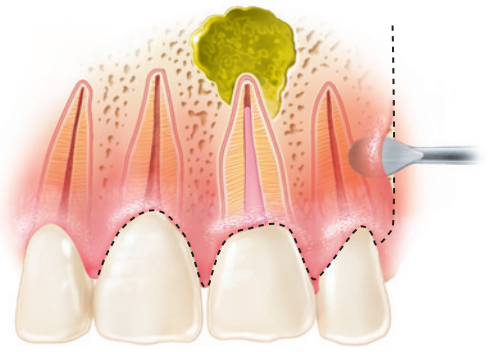

FIG. 9-31 Elevator placed in the vertical incision for the first step in undermining flap reflection.

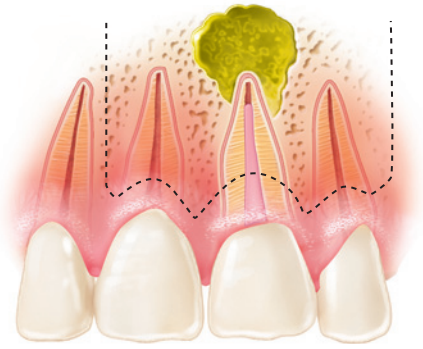

FIG. 9-29 Submarginal (Ochsenbein-Luebke) flap.

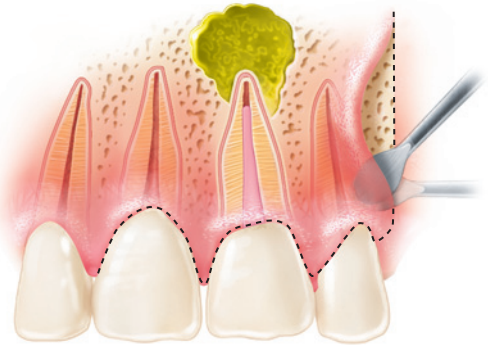

FIG. 9-32 Continuation of reflection of full-thickness flap.

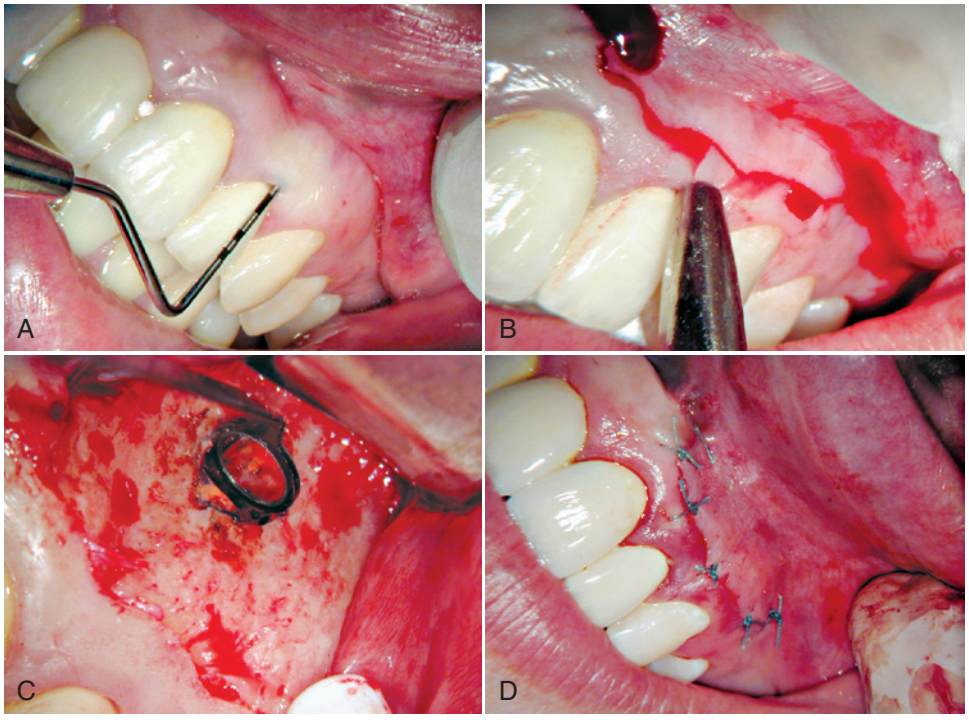

FIG. 9-30 Clinical case involving a submarginal incision and flap. **A,** Periodontal probing of the entire area was performed before this type of flap was selected and the incision started. A submarginal incision often is used in aesthetic anterior areas of the mouth where postoperative gingival recession might expose the crown margins. **B,** The incision should be at least 2 mm apical to the depth of the sulcus. **C,** With the flap reflected, osteotomy and root-end resection were performed. Methylene blue dye was placed to mark the outline of the root end and to help identify cracks or fractures before root-end cavity preparation and filling. **D,** The flap was repositioned and sutured with 5-0 Tevdek. (Courtesy Dr. Martin Rogers.)

The operator should take great care not to slip during the tissue reflection process by using an appropriate instrument stabilized with adequate finger support. Slipping can result not only in puncture of the immediate overlying tissue but also damage to the surrounding structures.

As space permits, the elevator should be directed coronally, undermining the attached gingiva. As the interdental papilla is approached, a narrower instrument may be required to undermine and gently elevate the tissue in this region, to avoid crushing the delicate free gingival tissues. This process should be continued gradually until the osseous tissues overlying the diseased tooth structure have been adequately exposed. Generally, elevation of the flap 0.75 cm apical to the estimated apex of the root should allow adequate space to perform the surgical procedure.

No single instrument is essential for the flap elevation procedure, because every instrument has both advantages and disadvantages. Surgeons should familiarize themselves with the various instruments available.

Tissue Retraction

After the tissue is reflected, it must be retracted to provide adequate access for bone removal and root-end procedures. The main goals of tissue retraction are to provide a clear view of the bony surgical site and to prevent further soft-tissue trauma. Accidental crushing of the soft tissues leads to more postoperative swelling and ecchymoses.[222,223]

The general principles of retraction are as follows: (1) retractors should rest on solid cortical bone; (2) firm but light pressure should be used; (3) tearing, puncturing, and crushing of the soft tissue should be avoided; (4) sterile physiologic saline should be used periodically to maintain hydration of the reflected tissue; and (5) the retractor should be large enough to protect the retracted soft tissue during surgical treatment (e.g., prevent it from becoming entangled in the bone bur). No one retractor suffices for all surgical procedures, therefore the surgeon should have a selection of retractors available for the various situations that arise during surgery. If difficulty is encountered in stabilizing the retractor, a small groove can be cut into the cortical plate to support it.

Hard-Tissue Access

Two biologic principles govern the removal of bone for hard-tissue access to diseased root ends: (1) healthy hard tissue must be preserved, and (2) heat generation during the process must be minimized.

Temperature increases above normal body temperature in osseous tissues are detrimental. Heating osseous tissue to 117° to 122°F (47° to 50°C) for 1 minute significantly reduces bone formation and is associated with irreversible cellular damage and fatty cell infiltration.[161,338] Two critical factors determine the degree of injury: how high the temperature increases and how long it remains elevated. As the temperature rises above 104°F (40°C), blood flow initially increases. It stagnates at 198°F (46°C) applied for 2 minutes. Heating osseous tissue to 133°F (56°C) deactivates alkaline phosphatase.[422,423] Studies using animal bone have shown that at temperatures above 109°F (42.5°C), for every 1°C elevation in temperature, the exposure time for the same biologic effect decreases by a factor of approximately 2.[158-161] Temperatures above 117°F (47°C) maintained for 1 minute produce effects similar to those at 118°F (48°C) applied for 30 seconds. This correlation means that the decisive exposure time declines quickly as the temperature increases. Temperatures above 127°F (53°C) applied for less than 1 second can adversely affect osteogenesis.[158-161]

Several factors determine the amount of heat generated during bone removal, including the shape and composition of the bur, the rotational speed, the use of coolant, and the pressure applied during cutting.

The round bur has the best shape for removing osseous tissue, and it should be used with a gentle brushstroke action.[519] This type of bur also readily allows access of coolant to the actual cutting surfaces. Studies comparing the heat generated with round and fissure burs found more favorable results with the round burs.[85,111,216,338,360] Cutting with round burs produced a wound site with less inflammation, which is more favorable for rapid wound healing. Although fissure-type burs cut efficiently on the sides, the tip of the bur is very inefficient because it allows no coolant access. The net result is increased inflammation and a reduced healing response.

Use of a diamond bur to remove osseous tissue is inefficient and retards ultimate wound healing. Because of its larger surface area, more of a diamond bur is in contact with the bone tissue. As a result, less coolant reaches the cutting surface, and the bur has a greater tendency to become clogged with residual bone fragments. The net effect is greater heat generation, increased inflammation, and reduced healing.[85,586]

Use of a coolant during bone cutting is essential. If an appropriate irrigant is not used, temperatures can exceed those known to impair bone healing[274]; histologically, healing can be delayed up to 3 weeks.[169] It also is critical that the coolant reach the cutting surface. Temperatures can rise above 212°F (100°C) when excess pressure is applied during cutting; this burrows the bur into the bone, where little or no irrigant can reach the cutting tip,[519] hence the recommendation for a gentle brushstroke technique.[213] Favorable results are obtained with these provided the surgeon follows the basic tenet of minimizing heat generation: using a round fluted bur with coolant and a brushstroke technique. A high-speed handpiece that exhausts air from the base rather than the cutting end is recommended to reduce the risk of air embolism (Fig. 9-33).

PERIRADICULAR CURETTAGE AND BIOPSY

Most periradicular lesions originate in the pulp and can be classified histopathologically as granulomas or

FIG. 9-33 Surgical handpiece with 45-degree angle head and rear air exhaust (Impact Air 45).

cysts.[53,58,292,295,297,359,367,382,488] Histologically, such lesions consist mainly of granulation tissue associated with angiogenesis, fibroblasts, connective tissue fibers, and inflammatory cells. Foreign material, cholesterol clefts,[365,370] and stimulated strands of epithelium also may be present. The stimulated epithelium can form into a stratified, squamous epithelium–lined cystic cavity.[366] These periradicular lesions (granulomas and cysts) are inflammatory lesions that develop in response to irritation caused by intraradicular and extraradicular microorganisms associated with the root canal system[363,368] or by foreign materials forced into the periradicular tissues.[600]

An important aspect of periradicular surgery is the removal of diseased tissue associated with the root apex. Because a large portion of this tissue is reactionary, the focus of surgical root canal treatment is removal of the irritant or diseased tissues. Histologically, an inflammatory periradicular lesion is similar to healing granulation tissue. If the irritant can be readily identified and successfully eliminated, it is not always necessary to completely curette all the inflamed periradicular tissues during surgery.[306] This is especially true when complete removal might result in injury to neural or vascular tissues. In addition to removing diseased tissue, periradicular curettage provides visibility and accessibility to facilitate treatment of the apical root canal system or removal of foreign materials in the periradicular tissues.

The need for a histopathologic assessment of all tissues removed from the body cannot be overstated. Although only a small percentage of periradicular lesions are associated with pathoses other than a periradicular cyst or granuloma, all lesions must be diagnosed definitively because of the potential gravity of the few rare diseases associated with periradicular lesions.[12,121,185,383,409]

The technical aspect of removing soft tissue from the bony crypt varies among surgeons and clinical settings. Various bone and periodontal curettes are available for this purpose, and no one instrument suffices for all cases. Regardless of the instrument selected, the basic principles are the same. A sharp instrument is always preferable to a blunt instrument. The soft-tissue lesion first should be peeled away from the osseous crypt, starting at the lateral borders. This can be accomplished efficiently by using the curette with the concave surface facing the internal wall of the osseous crypt. Once the soft-tissue lesion has been separated from the osseous crypt to the point where the crypt changes its convexity, the curette can be used in a scraping manner to remove the remainder of the lesion from the medial wall of the osseous defect.

LOCALIZED HEMOSTASIS

Localized hemostasis during periradicular surgery is essential to successful management of the resected root end. Appropriate hemostasis during surgery minimizes surgical time, surgical blood loss, and postoperative hemorrhage and swelling.[213] The hemostatic agents used during endodontic surgery are intended to control bleeding from small blood vessels or capillaries. Localized hemorrhage control not only enhances visibility and assessment of the root structure, but it also ensures the appropriate environment for placement of the current root-end filling materials and minimizes root-end filling contamination.

Many hemostatic agents have been advocated for use during surgery, and the action of these agents, their ability to control bleeding, and their effect on healing vary considerably. They generally aid coagulation by inducing rapid development of an occlusive clot, either by exerting a physical tamponade action or by enhancing the clotting mechanism and vasoconstriction (or both). No one local hemostatic agent is ideal; each has disadvantages. Therefore, further investigation is indicated to find the ideal local hemostatic agent.

Preoperative Considerations

A thorough review of the patient's body systems and medical history increases the likelihood of detecting an undiagnosed condition that might affect hemostasis during periradicular surgery. Review of the patient's medications, both prescribed and over-the-counter (OTC) drugs, is essential. Many OTC drugs can affect the clotting mechanism. The patient's vital signs (i.e., blood pressure, heart rate, and respiratory rate) should be assessed. Vital signs also can be used to monitor anxious patients. An increase in blood pressure and heart rate above a patient's known normal values indicates increased stress or poorly controlled hypertension. Easing the patient's anxiety before surgery reduces the possible hemostatic-potentiating effect of elevated cardiac output during surgery.[156] Anxiety and stress can be alleviated with planning, sedation, and profound local anesthesia.

Local Hemostatic Agents

Collagen-Based Materials

Various collagen-based hemostatic agents are available for use as local hemostatic agents. The principal differences are in the microstructure and density of the collagen. Collagen can act as a mild allergen, but the problem of allergenization and unwanted tissue reaction does not occur when highly purified animal collagen is used.[49] The mechanisms by which collagen products help achieve hemostasis involves stimulation of platelet adhesion, platelet aggregation and release reaction,[270,271] activation of factor XII (Hageman factor),[335,336] and mechanical tamponade by the structure that forms at the collagen-blood wound interface. Collagen shows minimal interference in the wound healing process, with a limited foreign body reaction.[215] It does not increase the incidence of infection and only slightly delays early bone repair.[246] Osseous regeneration in the presence of collagen typically proceeds uneventfully, without a foreign body reaction.[168]

Collagen-based materials can be difficult to apply to the bony crypt because they adhere to wet surfaces, particularly instruments and gloves.[466] Several collagen-based products are commercially available. They include CollaCote (Integra Life Sciences, Plainsboro, NJ) (Fig. 9-34, *A*), CollaStat (American Medical Products Corp, Eatontown, NJ), Hemocollagene (Septodont, United Kingdom) and Instat (Ethicon, Somerville, NJ). These materials act in essentially similar ways, and the surgical area undergoes a similar healing pattern.[485,504] Overall, studies of wound healing with collagen-based hemostatic agents have shown favorable results.

Surgicel

Surgicel (Ethicon) is a chemically sterilized material prepared through oxidation of regenerated alpha cellulose (oxycellulose). The basic element of Surgicel is polyanhydroglucuronic acid, which is spun into threads and then woven into gauze. Surgicel has a pH of 3. If the material is maintained in the wound for up to 120 days, a pH this low could retard healing.[408]

FIG. 9-34 A, Absorbable collagen (CollaCote) is a convenient, biocompatible packing material for localized hemostasis. B, Cotton pellets impregnated with racemic epinephrine (Racellet) also may be used for localized hemostasis.

It is primarily a physical hemostatic agent, which acts as a barrier to blood and then becomes a sticky mass that serves as an artificial coagulum. It does not enhance the clotting cascade through adhesion or aggregation of platelets. Surgicel is retained in the surgical wound,[373] and healing is retarded, with little evidence of resorption of the material at 120 days.[57] Use of Surgicel in extraction sockets resulted in greater postoperative pain compared with a control in a split mouth–designed study.[408]

Gelfoam

Gelfoam (Pharmacia, Peapack, NJ) is a gelatin-based sponge that is water insoluble and biologically resorbable. It stimulates the intrinsic clotting pathway by promoting platelet disintegration and the subsequent release of thromboplastin and thrombin.[164] The initial reaction to Gelfoam in the surgical site is a decrease in the rate of healing. Extraction sockets containing Gelfoam showed a greater inflammatory cell infiltrate, marked reduction in bone ingrowth, and a foreign body reaction at 8 days.[74] However, these effects were transitory and did not impair long-term bone healing.[392]

Bone Wax

Historically, bone wax has been advocated for controlling both hemostasis and debris in the bony crypt during periradicular surgery.[480] It is a nonabsorbable product composed of 88% beeswax and 12% isopropyl palmitate. Healing with bone wax is best described as poor. The bony crypt typically contains fibrous connective tissue and has no bony or hematopoietic tissue. Bone wax retards bone healing and predisposes the surgical site to infection[117,378] by producing a chronic inflammatory foreign body reaction[35] and impairing the clearance of bacteria.[263] The use of bone wax can no longer be recommended because it impairs healing and several good alternatives are available.[589]

Ferric Sulfate

Ferric sulfate (Cut-Trol, Ichthys Enterprises, Mobile, AL), a necrotizing agent with an extremely low pH, is one of the few products investigated for use in periradicular surgery. In studies using a rabbit model, Lemon and colleagues[301] and Jeansonne and colleagues[256] reported hemostatic control for 5 minutes and near normal healing with only a mild foreign body reaction, provided the surgical wound was adequately curetted and irrigated with saline. Failure to remove ferric sulfate from the surgical wound site resulted in severely impaired healing, a foreign body–type reaction, and, in some cases, abscess formation. The possibility of acute inflammation and necrosis of the surrounding soft tissue with careless use of this solution should not be underestimated.[298] A similar product, Monsel's solution (ferric subsulfate), has been used to control local hemostasis in dermatologic procedures. However, the popularity of this solution has declined because application to wound sites has resulted in tissue necrosis for up to 2 weeks,[130] differences in the degree of epidermal maturation, and tattoo formation.[536]

Calcium Sulfate

Calcium sulfate has been used as a substitute bone graft material to fill bone defects since the late 1800s. The presence of calcium sulfate in an osseous wound does not inhibit bone formation.[574] It is gradually removed from the site of implantation regardless of whether new bone has formed.[110] Use of calcium sulfate during periradicular surgery does not significantly affect healing, and deposition of cementum and osseous healing proceed normally.[27] As a hemostatic agent, calcium sulfate acts as a physical barrier. The material is placed in the bony crypt, allowed to set, and then partly carved away to allow access to the root end.[283] The remaining material lines the crypt walls, preventing bleeding. When the root-end filling has been placed and all extraneous root-end filling material removed, the residual calcium sulfate can be removed or left in situ.

Epinephrine Pellets

Epinephrine, a sympathomimetic-amine vasoconstrictor, is frequently used to control hemorrhage during oral surgery.[82,283] All granulation tissue should be removed from the root apex area prior to placement of the epinephrine pellet to assure direct contact with bone.[282] Vasoconstrictive amines exert their effects by binding to and interacting with adrenergic receptors in various body tissues. When epinephrine is bound to alpha$_1$- and alpha$_2$-adrenergic receptors, a powerful vasoconstricting effect results. Racemic epinephrine cotton pellets (Racellet #3; Pascal Co., Bellevue, WA) contain an average of 0.55 mg of racemic epinephrine hydrochloride per pellet, half of which is the pharmacologically active L-form. Racellets (see Fig. 9-31, *B*) provide good localized hemostasis in periradicular surgery.[558]

Two concerns arise with the use of Racellets in the surgical site: the cardiovascular impact of the additional epinephrine and the retention of cotton fibers in the wound, resulting in impaired wound healing.[213,265] A study by Vickers and coworkers[558] examined the cardiovascular effects of epinephrine pellets and concluded that no evidence existed of cardiovascular changes (blood pressure and pulse) compared with saline-saturated pellet controls. These authors hypothesized that the vasoconstrictive effect on the capillaries is localized and immediate and that little or no systemic uptake of epinephrine occurred.

Although the cardiovascular effect appears to be of little concern, retention of cotton fibers in the surgical site could result in inflammation and impaired wound healing. Therefore,

due diligence on the part of the surgeon is paramount when epinephrine pellets are used. Each pellet applied during surgery must be accounted for, and the bony crypt should be lightly curetted to remove any embedded cotton fibers before the wound is closed. The retention of cotton fibers in the crypt can be eliminated by substituting CollaCote saturated with 10 drops of 2.25% racepinephrine inhalation solution.[567] As previously noted, CollaCote is biocompatible and does not interfere with wound healing.

Cautery/Electrosurgery

Cautery stops the flow of blood through coagulation of blood and tissue protein, leaving an eschar that the body attempts to slough.[537] The effect of cautery in the bony crypt during periradicular surgery has not been studied to date. However, the effect of electrosurgery on alveolar bone has been studied in periodontal surgery. Tissue destruction was greater in areas exposed to electrosurgery and healing was delayed compared with surgical sites not exposed to electrosurgery. Twelve hours after surgery, a more extensive inflammatory reaction and greater destruction of periosteum were noted with electrosurgery.[36] At 24 hours, many empty lacunae were observed in the bone associated with electrosurgery, and this necrosis was even more extensive by 48 hours. At 96 hours the electrosurgical connective tissue wounds were still lined by coagulum, whereas the scalpel wound was beginning to repair.[381] The detrimental effect of applying heat to bone is proportional to both temperature and the duration of application.

MANAGEMENT OF THE ROOT END

Management of the resected root end during periradicular surgery is critical to the overall success of a case. The aim of surgery should be to create an environment conducive to regeneration of the periodontium—that is, healing and regeneration of the alveolar bone, periodontal ligament, and cementum overlying the root end and root-end filling material. The key to regeneration is the presence of appropriate inducible cell types, growth factors, and specific substances necessary for mineralization. Failure to create an environment conducive to this process results in tissue repair rather than regeneration and possibly less than ideal healing.

Determination of the Need for Root-End Resection and Filling

The basis for periradicular surgery is twofold. The first objective is to remove the etiologic factor; the second is to prevent recontamination of the periradicular tissues once the etiologic agent has been removed.

Etiologic factors typically can be categorized as intraradicular or extraradicular bacteria,[254,287,367,491,540,541,559,580] intraradicular or extraradicular chemical substances, or extraradicular physical factors.[370,496,497] The etiology cannot always be determined with complete certainty; frequently, a number of factors are involved.[98] However, most cases involve some form of bacterial participation (e.g., bacteria within apical ramifications). The only definitive means of eradicating such an irritant is physical removal through root-end resection. The rationale for root-end resection in such cases is to establish access to and remove the diseased tissues. This ensures that the optimum environment for wound healing is established.

As mentioned, the second objective of periradicular surgery is to prevent recontamination of the tissues after removal of the etiologic agent. It follows, then, that if the remainder of the canal system cannot be verified to be irritant free, a root-end filling should be placed to seal any remaining irritants within the canal system, thus preventing recontamination of the periradicular tissues.

Root-End Resection

Two main principles dictate the extent of the root-end resection. First, the cause (or causes) of an ongoing disease process must be removed; this includes removal of the diseased tissue and, when indicated, reduction of an apically fenestrated root. Second, adequate room must be provided for inspection and management of the root end.

The anatomy of each tooth root is complex (see Chapter 5). The surgeon must understand the anatomy of the apical third of the root to determine the extent of a root-end resection. Approximately 75% of teeth have canal aberrations (e.g., accessory or lateral canals) in the apical 3 mm of the tooth.[131,481] An apical resection of approximately 3 mm should include most accessory and lateral canals and thus eliminate most residual microorganisms and irritants (Fig. 9-35). When roots with more than one main canal are resected, isthmus tissue may be present, and the preparation should be modified to include the isthmus area (Fig. 9-36).

If the root apex is close to the buccal cortical plate, apical fenestration can occur, leading to persistent symptoms, especially tenderness to palpation over the root apex.[72] Reduction of an apically fenestrated root apex below the level of the surrounding cortical bone allows remodeling of the bone over the tooth structure. The buccal root of the maxillary first premolar often is closest to the buccal cortical plate.

The surgeon's convenience as a rationale for root-end resection depends on the individual case and the surgeon's abilities. The basic principle of the dental surgeon's convenience should be modified by the desire to minimize the trauma of the surgical procedure itself, including the preservation of tooth and supporting structures. Access to and visibility of the periradicular root structures historically has determined the extent of root-end resection. The surgeon must be able to inspect the

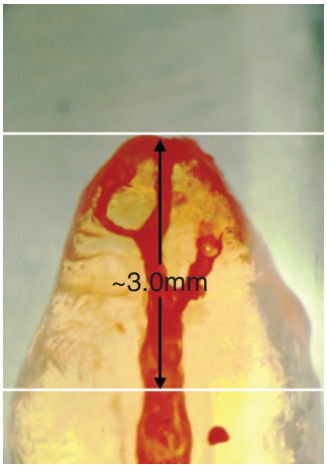

FIG. 9-35 Cleared section of a typical single-canal root that was injected with dye to demonstrate apical accessory canals. Most of the apical ramifications can be eliminated with a 3-mm resection.

resected root end, prepare a root-end cavity, and place a root-end filling. Enhanced visualization equipment (e.g., microscopes, endoscopes, and orascopes) have reduced the need to resect large amounts of the root to gain adequate visualization and access.[91,94,228,280,440,441,448,486] In some cases part of the root must be resected to gain access to the entire soft-tissue lesion, an additional palatally positioned root (e.g., maxillary premolars), or foreign material in the periradicular tissues.

A major consideration in determining the extent of root-end resection is the presence of anatomic structures such as the mental foramen or mandibular canal.[355,410-412] The surgeon should position the resection of the root to avoid possible damage to these structures.

Angle of Resection

Enhanced magnification and illumination techniques have eliminated the need to create a beveled root surface in most cases.[94,441] From a biologic perspective, the most appropriate angle of root-end resection is perpendicular to the long axis of the tooth (Figs. 9-37 and 9-38). The rationale for a perpendicular root-end resection is based on several anatomic parameters. First, a perpendicular resection approximately 3 mm from the anatomic apex is more likely to include all the apical ramifications in that region of the tooth.[339] Second, as the angle of resection increases, the number of dentinal tubules that communicate with the periradicular region and the root canal system increases significantly; therefore, the probability that irritants from within the canal system will gain access to the healing tissues also rises as the resection angle increases.[181,523] Third, extending the root-end cavity preparation beyond the coronal extent of the root surface is simpler if the root-end resection is perpendicular to the long axis of the tooth.[195] Finally, with a perpendicular root-end resection, the stress forces exerted in the apical region are more evenly distributed; this may reduce the propagation of apical fractures and provide a better environment for apical healing.[470]

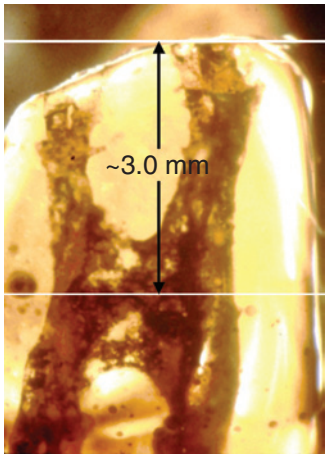

FIG. 9-36 Cleared section of a mesiobuccal (MB) root in a maxillary first molar. Root resection at the recommended 3-mm level exposes isthmus tissue connecting the MB-1 and MB-2 canals.

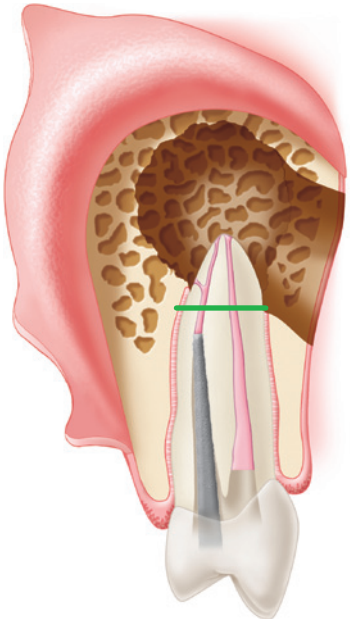

FIG. 9-38 Perpendicular or near perpendicular root-end resection *(green line)* can be achieved with the use of microsurgical instruments and enhanced magnification and illumination.

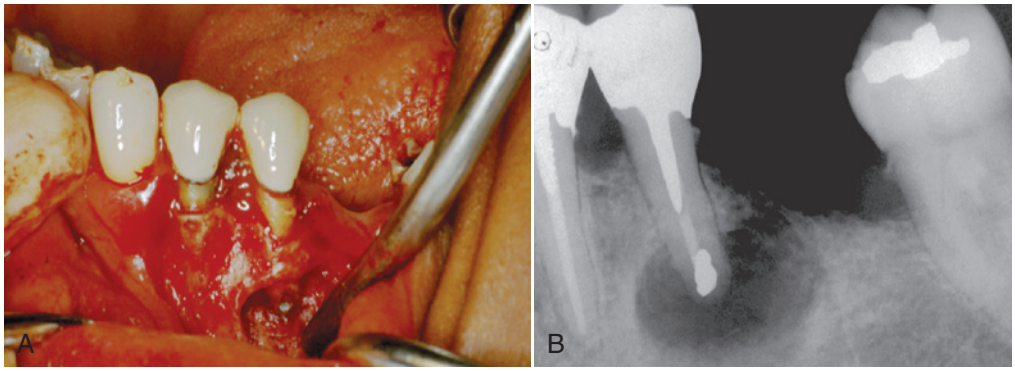

FIG. 9-37 Periradicular surgery circa 1990. **A**, The root end was prepared with a 45-degree bevel and rotary bur microhandpiece. Amalgam was a commonly used root-end filling material at this time. **B**, Immediate postoperative radiograph of a mandibular second premolar with amalgam root-end filling. Although many teeth treated this way healed successfully, newer materials and techniques described in this chapter are currently recommended.

Root-End Surface Preparation

As with all phases of endodontic surgery, the goal is to produce a resected root end with optimum conditions for the growth of cementum and subsequent regeneration of the periodontal ligament across the resected root end. Two important aspects of this process are the surface topography of the resected root end and chemical treatment of the resected root end. Healthy cementum on the root end is required for successful regeneration of periodontal tissues.[22] A number of substances found in cementum stimulate the migration, growth, and attachment of periodontal fibroblasts. Cementum extracts also activate fibroblast, protein, and collagen synthesis, which is necessary to reestablish a functional periodontal ligament.[52,471,562]

Resected Root-End Surface Topography

The conventional axiom for surface preparation of the resected root end has been to produce a smooth, flat root surface without sharp edges or spurs of root structure that might serve as irritants during the healing process. However, little information exists on whether smooth root ends heal differently or more quickly than rough root ends after root-end resection. A study investigating the effect of the surface topography of resected root ends on human PDL fibroblast attachment found no significant difference in fibroblast attachment to the root ends prepared with various instruments.[584] However, with a smooth resected root-end surface, the surgeon is better able to detect surface cracks and anatomic variations.[357] Given that human PDL fibroblast attachment to a smooth surface was not impaired, it would seem appropriate to produce as smooth a surface as possible to facilitate inspection of the resected root end. A smooth root end therefore should be considered advantageous.

Different types of burs tend to produce different patterns on the resected root surface.[212] Several studies have compared the root-end surface after resection.[357,377,585] Generally, crosscut fissure burs in both high-speed and low-speed handpieces produced the roughest and most irregular surfaces. Morgan and Marshall[357] compared the surface topography of root-end resection with a #57 straight fissure bur (Midwest Dental Products, Des Plains, IL), a Lindeman bone bur (Brasseler USA, Savannah, GA), and the Multi-Purpose bur (Dentsply Maillefer, Ballaigues, Switzerland), then finished with a either a multifluted carbide finishing bur (Brasseler USA) or an ultrafine diamond finishing bur (Brasseler). The Multi-Purpose bur produced the smoothest, most uniplanar resected root-end surface with the least amount of shattering. Regardless of the type of bur used, smearing and shredding of the gutta-percha across the root face occurred only when the handpiece was moved across the root face in reverse direction in relation to the bur's direction of rotation.[585] Burs that produce a smooth surface also tend to cut with less vibration and chatter, resulting in greater patient comfort.

Root-End Conditioning

Root surface conditioning removes the smear layer and provides a surface conducive to mechanical adhesion and cellular mechanisms for growth and attachment. It exposes the collagenous matrix of dentin and retains biologically active substances, such as growth factors, in the dentin proper. Experimental studies have shown that demineralized dentin can induce the development of bonelike mineralized tissue.[42,43,241,550,597] Some contend that root surface conditioning produces a biocompatible surface conducive to periodontal cell colonization without compromising the vitality of the adjacent periodontium.

Three solutions have been advocated for root surface modification: citric acid, tetracycline, and ethylenediamine tetraacetic acid (EDTA). All three solutions have enhanced fibroblast attachment to the root surface in vitro. However, citric acid is the only solution tested in an endodontic surgical application.

Citric acid traditionally has been the solution of choice. Periodontists have used an aqueous solution of citric acid (pH 1) for 2 to 3 minutes to etch diseased root surfaces to facilitate formation, new attachment, and cementogenesis.[434-438] Craig and Harrison[114] examined the effect of citric acid demineralization of resected root ends on periradicular healing. They found that 1- or 2-minute applications of 50% citric acid (pH 1) resulted in demineralized root ends and earlier complete healing than in the nondemineralized root ends. However, the periodontal literature has questioned the benefit of etching dentin surfaces with low pH agents. At a low pH, adjacent vital periodontal tissues may be compromised. Also, extended application (3 minutes) has been shown to discourage alveolar bone growth.[60,62]

EDTA, a solution with a neutral pH that endodontists have used as a canal irrigant, has been shown to be equally effective at exposing collagen fibers on dentin surfaces.[64] Unlike the lower pH solution, EDTA does not adversely affect the surrounding tissues.[63]

A series of studies that examined the effect of EDTA and citric and phosphoric acids in a periodontal application showed that application of 15% to 24% EDTA for approximately 2 minutes produces the optimum root surface.[59,61,64] These researchers concluded that EDTA, at neutral pH, was able to selectively remove mineral from a dentin surface, exposing a collagenous matrix. Citric and phosphoric acids, which have a low pH, appeared not only to remove the mineral component but also to denature the collagenous matrix.

Tetracycline has been shown to remove the dentin smear layer, leaving clean, open tubules, with application times as short as 30 seconds.[327] A histologic evaluation of new attachment in periodontally diseased human roots treated with tetracycline hydrochloride showed a trend for greater connective tissue attachment after tetracycline treatment of roots.[13] Studies comparing the effect of a 3-minute application of either EDTA (pH 7.3) or tetracycline HCl (pH 1.8) showed no significant difference in the treated tooth surfaces.[37] However, EDTA has been shown to be more favorable to human PDL cell attachment.[601]

Although the root surface conditioning effects of citric acid, EDTA, and tetracycline are well documented in the periodontal literature, this treatment has not translated into significant gains in periodontal attachment in periodontally diseased teeth.[330] Currently only citric acid has been assessed as a root-end conditioning agent. In an animal model, citric acid was shown to enhance periradicular healing. Nonetheless, its effect and the effect of other root-end conditioning agents on the outcome of human periradicular surgery have not been established. Based on periodontal research, it would appear that if a root surface conditioning agent were to be used during periradicular surgery, EDTA might be the most appropriate solution. However, the manufacturer (personnel communication, Dr. Torabinejad) has advised against the use of EDTA when mineral trioxide aggregate (MTA) is used as a root-end filling material, because it may interfere with the hard tissue–producing effect of MTA.

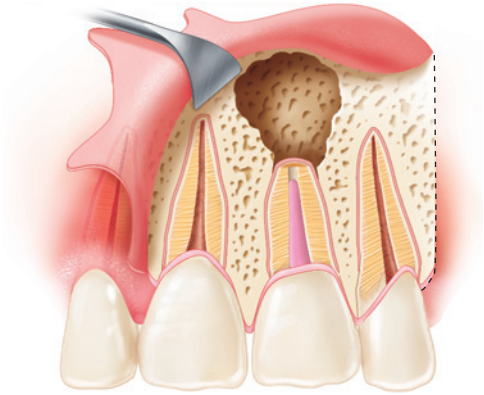

FIG. 9-39 Diagram of a perpendicular root-end preparation and 3-mm deep cavity preparation along the long axis of the root.

FIG. 9-40 Error in root-end cavity preparation: ultrasonic preparation did not follow the long axis of the mesial root and therefore did not allow for proper sealing of this root. Healing is unlikely.

ROOT-END CAVITY PREPARATION

The root-end cavity preparation is a crucial step in the establishment of an apical seal. The goal is to make a cavity in the resected root end that is dimensionally sufficient for placement of a root-end filling material and at the same time avoid unnecessary damage to the root-end structures. The ideal preparation is a class I cavity prepared along the long axis of the tooth to a depth of at least 3 mm (Figs. 9-39 and 9-40). The surgical procedure is most likely to be successful if the remaining canal system has been thoroughly cleaned and shaped to eliminate microorganisms and irritants.[178,449] Traditionally, a microhandpiece with a rotating bur has been used for this purpose. However, with the advent of ultrasonic tips designed specifically for this purpose (Fig. 9-41), root-end preparations now are most often performed with the ultrasonic technique.[91] Clinical evidence is emerging to support the benefit of an ultrasonic root-end preparation when compared to traditional bur preparation, especially in molar surgery.[132]

Ultrasonic root-end preparation techniques have several advantages over the microhandpiece method. Less osseous tissue must be removed to gain proper access to the resected root end. Also, the surgeon is better able to produce a more conservative preparation that follows the long axis of the tooth and remains centered in the canal. The risk of root-end perforation is reduced, partly because of enhanced manipulation of the instrument. In addition, ultrasonic root-end techniques produce a more consistent, deeper cavity preparation that requires less beveling of the root.[95,304,347,592] Ultrasonic apical preparation generates significantly less smear layer compared with burs alone[205]; root-end preparation with a bur produces a heavy smear layer at all levels of preparation.[214]

The major concern with ultrasonic root-end preparation is the potential for creating root fractures as a result of the ultrasonic vibration (Fig. 9-42).

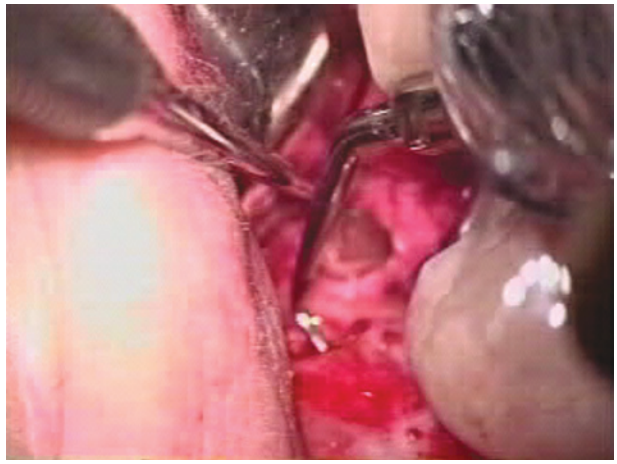

FIG. 9-41 Ultrasonic tip in use. Preparation along the long axis of the root is possible using tips designed for each area of the mouth. In this case, the tip is properly positioned for root-end cavity preparation of a maxillary first premolar but is dangerously close to the lip because of improper retraction. Heat generated by an ultrasonic tip can cause a thermal burn, which may result in scar tissue formation.

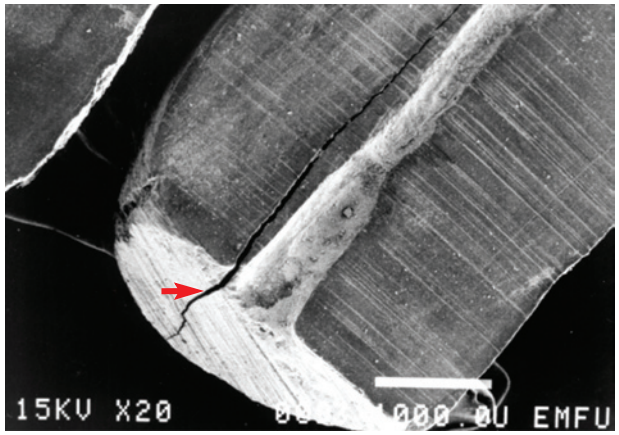

FIG. 9-42 Scanning electron microscope (SEM) image of a root end prepared in vitro with an ultrasonic device at high-power setting. A distinct fracture line can be seen *(red arrow)*. Root-end preparation with ultrasonic devices should be done at low power and with water coolant.

Ultrasonic Root-End Preparation and Apical Fractures

Several studies have investigated the fracture-inducing potential of ultrasonic root-end preparation techniques. This is possibly the most controversial question that arises in relation to

the use of ultrasonic root-end preparation tips. Three types of root-end fractures have been described: intracanal fractures (originating from the root canal system and extending into the dentin), extracanal fractures (originating on the root surface and extending into the dentin), and communicating fractures (extending from the root surface to the root canal system).[425] This classification system is not universally used in studies of root-end cavity preparation; therefore, it is difficult to establish the significance, if any, of one type of fracture over another. When a strain gauge was used to measure root deformation, ultrasonic root-end preparation was shown to produce significantly greater strain, on average, than that generated by a microhandpiece. However, this did not translate into an increase in cracks observed on the resected surface of roots after root-end cavity preparation.[48,303,375,425] Several other in vitro studies that used different models to assess root-end fractures conversely concluded that ultrasonic root-end preparation does induce apical fractures.[2,184,299,354,469] Thus, the degree to which apical fractures are induced during ultrasonic root-end cavity preparation is difficult to determine from in vitro studies.

On the other hand, in an in vivo study[358] and cadaver-based studies[86,207] designed to replicate the clinical scenario, root fractures were not attributed to ultrasonic use. In these studies ultrasonic root-end preparation did not induce a significant number of root-end fractures. Several explanations may account for the differences observed in these studies. The surrounding tissues in cadaver and clinical subjects may disperse the ultrasonic energy away from the root tip. Thermal energy produced during ultrasonic preparation may have been controlled more adequately in some studies than in others. The power setting used on the ultrasonic unit may have been in the low range; a low power setting has been shown to produce fewer fractures in an in vitro setting[176,299] and therefore is recommended for clinical use.

Significance of Ultrasonic Tip Design

Different types of ultrasonic tips are available for root-end preparation (Figs. 9-43 and 9-44), including tips of varying lengths and diameters constructed of stainless steel. These tips are left uncoated or are coated with diamond or zirconium nitride. Tips with a curvature of 70 degrees or greater are more susceptible to fracture under continuous loading, and fracture typically occurs at the bend.[573] Coating of ultrasonic tips undoubtedly improves the cutting efficiency compared with uncoated stainless steel tips; this translates into significantly less time required to prepare a root-end cavity.[200,405] In the coated tips, the diamond coating appears to be the most aggressive and requires the least amount of time to produce a root-end cavity preparation.[253] Furthermore, the type of tip (i.e., stainless steel, diamond coated, or zirconium nitride coated) appears to have little effect on the number or types of fractures that can be induced in the root end during root-end preparation.[76,200,253,375,425] The cavity walls of root-end preparations formed by stainless steel tips typically have cleaner canal walls than those formed by coated root-end preparation tips. Stainless steel tips appear to produce less superficial debris and smear layer. Coated root-end preparation instruments typically produce a heavily abraded, debris-covered cavity wall surface.[76,610] However, overall the quality of the coated-tip preparation has been suggested to be superior.[405]

Temperature Changes Induced by Ultrasonic Instruments

The importance of heat generation and temperature changes has already been discussed. All ultrasonic surgical tips should have an irrigation port. Use of an ultrasonic instrument in the periradicular tissues without adequate irrigation results in an extreme temperature increase in the tissues, although this specific effect has not been demonstrated during root-end preparation. Scaling without irrigation can increase the temperature in dentin as much as 95° F (35° C) above the baseline temperature[290]; such an increase can injure pulpal and periodontal tissues.[380]

Bonded Root-End Fillings

Root-end cavity preparation for bonded root-end filling materials requires a change in the standard root-end cavity preparation technique. A shallow, scalloped preparation of the entire root surface should be made using a round or oval bur; the preparation should be at least 1 mm at the deepest concavity.[26,453] An ultrasonic preparation can be made into the root

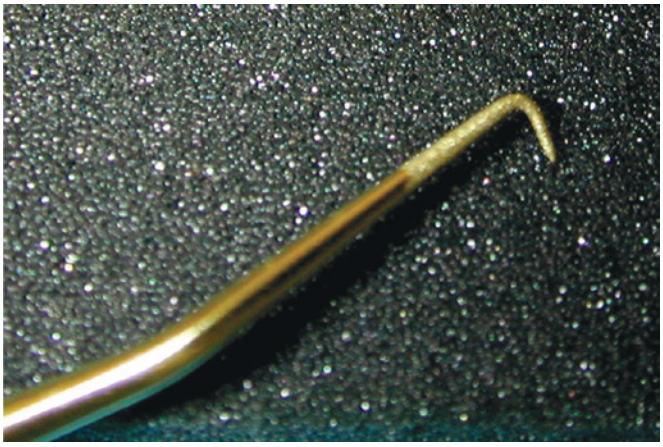

FIG. 9-43 Ultrasonic tips (Obtura Spartan) are available in a wide variety of configurations for use in different areas of the mouth. Most new tips have a special coating (zirconium nitride or diamond) that improves cutting efficiency.

FIG. 9-44 Ultrasonic tip with diamond coating and irrigation port (DENTSPLY Tulsa Dental Specialties).

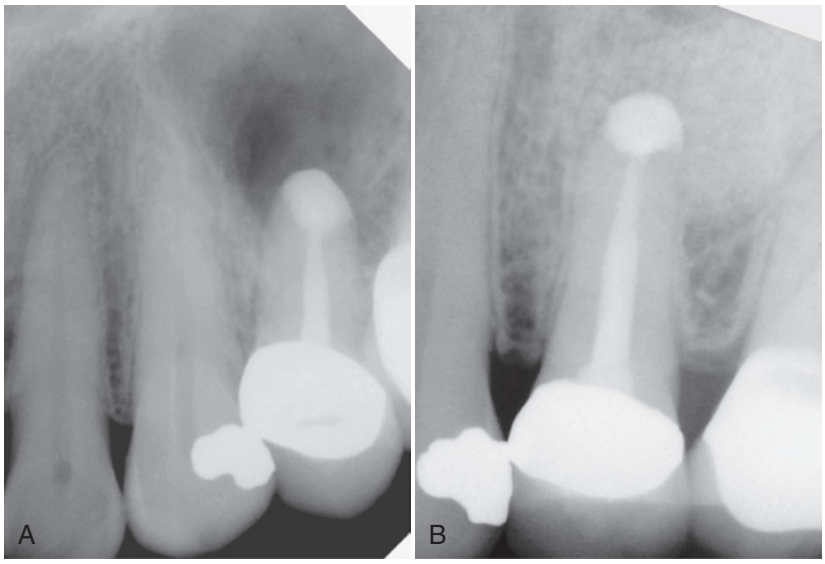

FIG. 9-45 A, Immediate postoperative radiograph of a bonded root-end filling in a maxillary first premolar. B, Follow-up radiograph at 20 months showing good periradicular healing.

canal system, but this may not be necessary. The root-end filling material is placed in a dome fashion and bonded to the entirety of the resected root end (Fig. 9-45).

ROOT-END FILLING MATERIALS

The ideal root-end filling material seals the contents of the root canal system within the canal, preventing egress of any bacteria, bacterial by-products, or toxic material into the surrounding periradicular tissues. The material should be nonresorbable, biocompatible, and dimensionally stable over time. It should be able to induce regeneration of the PDL complex, specifically cementogenesis over the root-end filling itself. Finally, the handling properties and working time should be such that the endodontic surgeon can place a root-end filling with sufficient ease.

Many materials have been used as root-end fillings, including gutta-percha, polycarboxylate cements, silver cones, amalgam, Cavit (3M ESPE, St. Paul, MN) zinc phosphate cement, gold foil, and titanium screws. However, this section focuses on root-end filling materials discussed in the literature within the past 10 years that are in common clinical use. These materials are zinc oxide–eugenol cements (IRM and Super-EBA), glass ionomer cement, Diaket, composite resins (Retroplast), resin–glass ionomer hybrids (Geristore), and mineral trioxide aggregate (ProRoot-MTA), and bioceramics.

Zinc Oxide–Eugenol (ZOE) Cements

Zinc oxide powder and eugenol liquid can be mixed to form a paste that is compacted into a cavity preparation. Use of this material dates back to the 1870s. Eugenol is released from ZOE mixtures, although this declines exponentially with time and is directly proportional to the liquid-powder ratio.[243] When ZOE comes in contact with water, it undergoes surface hydrolysis, producing zinc hydroxide and eugenol. This reaction continues until all the ZOE in contact with the free water is converted to zinc hydroxide.[243-245] Eugenol can have a number of effects on mammalian cells, depending on the concentration and length of exposure. These effects include cell respiration depression, macrophage and fibroblast cytotoxicity, depressed vasoconstrictor response, inhibition of prostaglandin, and suppressing or enhancing effects on the immune response.[139,349,561] Other materials have been added to the basic ZOE mixture in an effort to increase the strength and radiopacity and reduce the solubility of the final material. Commercially available ZOE materials include intermediate restorative material (IRM; Dentsply Caulk, Milford, DE) and Super-EBA (Bosworth Company, Skokie, IL).

Intermediate Restorative Material (IRM)

IRM consists of a powder containing greater than 75% zinc oxide and approximately 20% polymethacrylate mixed in equal parts with a liquid that contains greater than 99% eugenol and less than 1% acetic acid. IRM seals better than amalgam and is not affected by the liquid-powder ratio or root-end conditioning agents.[116,407] IRM appears to be tolerated in the periradicular tissue, but it has no dental hard-tissue regenerative capacity. The response is similar to that seen with other ZOE-based materials.[221,329,417-420] In vitro, IRM prevents adherence of enamel matrix proteins.[463]

Super-EBA

Super-EBA consists of a powder containing 60% zinc oxide, 34% aluminum oxide, and 6% natural resins. It is mixed in equal parts with a liquid that contains 37.5% eugenol and 62.5% o-methoxybenzoic acid. Super-EBA is available in two forms, fast set and regular set. Other than the setting time, the properties of the two forms appear to be the same.[593] Super-EBA has radiopacity[484] and sealing effects similar to those of IRM and is less leaky than amalgam.[232,388] The leakage pattern of Super-EBA does not appear to be affected by root-end conditioning or finishing techniques.[174,469] When Super-EBA and IRM were finished with a carbide finishing bur in a high-speed handpiece, marginal adaptation was better than with ball burnishing, which was equal to burnishing with a moistened cotton pellet.[170] The environment of the periradicular wound may affect the long-term stability of Super-EBA, which has been shown to disintegrate over time in an acid pH environment.[30]

Biologically, Super-EBA is well tolerated in the periradicular tissues when used as a root-end filling. However, it has no capacity to regenerate cementum. Bone healing has been demonstrated at 12 weeks, with some fibrous tissue persisting. Super-EBA root-end fillings show a basophilic-stained line adjacent to the filling material, which may indicate hard-tissue formation.[404,418,419,542] Collagen fibers appear to grow into the cracks of the material,[396] but the significance of this is unknown. Super-EBA has limited antibacterial effect.[101] The cytotoxicity of Super-EBA is similar to that of amalgam and IRM.[102,605] The incidence of persistent disease after endodontic surgery in which Super-EBA was used as a root-end filling material ranges from 4% to 20%. In comparative studies in which amalgam was also used as a root-end filling material, use of Super-EBA always resulted in less persistent disease. The follow-up period for these studies ranged from 0.5 to 4.6 years.[147,326,397,448,518,565]

Glass-Ionomer Cements

Glass-ionomer cement (GIC) consists of aqueous polymeric acids, such as polyacrylic acid, plus basic glass powders, such as calcium aluminosilicate. GIC sets by a neutralization reaction of aluminosilicate, which is chelated with carboxylate groups to cross-link the polyacids; a substantial amount of the glass remains unreacted and acts as reinforcing filler. Glass-ionomer cements can be either light or chemically cured. Silver has been incorporated into GIC to improve the physical properties, including compressive and tensile strength and creep resistance. Both forms of GIC have been suggested as an alternative root-end filling material.[44,415,416]

The seal and marginal adaptation of light-cured GIC are superior to those with chemical-cured GIC. The seal achieved with GIC generally is better than that with amalgam and similar to that with IRM.[105,106,252,444,520,591] Long-term surface changes can occur in silver-GIC that may affect the stability of silver-GIC in the periradicular tissues.[55] Glass-ionomer cements are susceptible to attack by moisture during the initial setting period, resulting in increased solubility and decreased bond strength.[188,508,596] Contamination with moisture and blood adversely affected the outcome when GIC was used as a root-end filling material; this occurred significantly more often in unsuccessful cases.[602] The cytotoxicity of chemical- and light-cured GIC does not differ significantly from that of Super-EBA or amalgam.[102,394] The tissue response to GIC is considerably more favorable than to amalgam and similar to that with ZOE-based materials.[100,104,133,416] In a comparative clinical study using amalgam or GIC for root-end filling, healing was evaluated clinically and radiographically after 1 and 5 years.[261,602] No difference was seen in healing capacity between the two materials. The overall success rate in both groups was 90% at 1 year and 85% at 5 years. This study showed that the 5-year follow-up result can be predicted in more than 95% of the cases at the 1-year follow-up. These authors concluded that GIC is a valid alternative to amalgam for use as an apical sealant after root-end resection and that it provides equally good long-term clinical results.[261,602]

Diaket

Diaket (ESPE GmbH, Seefeld, Germany) a polyvinyl resin initially intended for use as a root canal sealer, has been advocated for use as a root-end filling material.[587] It is a powder consisting of approximately 98% zinc oxide and 2% bismuth phosphate mixed with a liquid consisting of 2.2-dihydroxy-5.5 dichlorodiphenylmethane, propionylacetophenone, triethanolamine, caproic acid copolymers of vinyl acetate, and vinyl chloride vinyl isobutylether. Leakage studies comparing Diaket to other commonly used root-end filling materials have shown it to have a superior sealing ability.[190,264,313,571] Its sealing ability has not been directly compared to that of MTA. When Diaket was used as a root canal sealer, biocompatibility studies showed that it was cytotoxic in cell culture[276] and generated long-term chronic inflammation in osseous[502] and subcutaneous tissues.[393] However, when mixed at the thicker consistency advocated for use as a root-end filling material, Diaket has shown good biocompatibility with osseous tissues.[379,587] Histologically, a unique tissue barrier has been observed to form across the Diaket at the resected root end, the nature of which is unknown. This tissue resembles an osteoid or cementoid type of matrix with a close approximation of periodontal tissue fibers, suggesting a regenerative response to the root-end filling material.[587] In animal studies Diaket has shown a better healing response than resected gutta-percha in uninfected teeth[590] and a healing response similar to that with MTA. However, no cementum formation was evident.[433] This material is no longer available in the United States.

Composite Resins and Resin-Ionomer Hybrids

Composite resin materials have some desirable properties and may be considered for use as root-end filling materials. Generally, when assessed for sealability, composite resins perform well in invitro studies. Composite resins also tend to leak less than amalgam, Super-EBA, IRM, and GICs.[126,344,345,520] However, blood contamination during the bonding process reduces bond strength and increases leakage.[353,560] Marginal adaptation varies, depending on the conditions and the bonding agents.[17] Certain components of composite resins and dentin-bonding agents can have a cytotoxic effect on cells; this effect varies, depending on the agent and its concentration.[80,217,428,429,514] Studies have shown that once the composite resin sets, cells can grow on its surface.[328,362,402,604] The healing response of the periradicular tissues to composite resins in general appears to be very diverse, ranging from poor to good[25,542]; this may depend on the type of material used. Two composite resin–based materials, Retroplast (Retroplast Trading, Rørvig, Denmark) and Geristore (Den-Mat, Santa Maria, CA), have been advocated for use as root-end filling materials.

Retroplast

Retroplast is a dentin-bonding composite resin system developed in 1984 specifically for use as a root-end filling material. The formulation was changed in 1990, when the silver was replaced with ytterbium trifluoride and ferric oxide. Retroplast is a two-paste system that forms a dual-cure composite resin when mixed. Paste A is composed of Bis-GMA/TEGDMA 1:1, benzoyl peroxide N,N-di-(2-hyydroxyethyl)-p-toluidine, and butylated hydroxytoluene (BHT). This is mixed in equal parts with paste B, which is composed of resin ytterbium trifluoride aerosil ferric oxide. A Gluma-based dentin bonding agent is used to adhere the material to the root-end surface. The working time is 1½ to 2 minutes, and the radiopacity (due to the ytterbium trifluoride content) is equivalent to 6 mm of aluminum.

Only limited information is available on the physical and chemical properties of Retroplast, although a number of human

clinical studies have been published.[22,25,361,451-455,457-460] In all cases the material appeared to be well tolerated and promoted a good healing response. There is evidence that Retroplast promotes hard-tissue formation at the root apex, and some have suggested that this is a form of cementum. In a limited number of case reports, Retroplast root-end fillings have demonstrated regeneration of the periodontium with a cementum layer over the root-end restoration.[22,453,454] The healing response in these cases showed deposition of minimal cementum and the insertion of new Sharpey's fibers. The PDL fibers also entered the newly formed adjacent alveolar bone, indicating that tissue regeneration, including cementogenesis, may occur on composite material, consequently forming a biologic closure of the root canal.[25] In an investigation of 388 cases comparing root-end fillings of Retroplast or amalgam, radiographic healing after 1 year was as follows: with Retroplast, 74% showed complete healing, 4% showed fibrous healing, 15% were uncertain, and 7% were failures; with amalgam, 59% showed complete healing, 3% showed fibrous healing, 30% were uncertain, and 8% were failures.[453] Complete healing occurred significantly more often after root-end filling with Retroplast. The number of immediate postoperative complications did not differ significantly between the composite and the amalgam groups. A more recent clinical study of 351 cases reported a complete healing rate of 80% to 89%.[459] A 10-year follow-up of 34 of these cases showed complete healing in 33 of the cases.[458]

Resin-Ionomer Suspension (Geristore) and Compomer (Dyract)

The resin-ionomer suspension and compomer group of materials attempts to combine the various properties of composite resins and glass ionomers. Geristore and Dyract (Dentsply, Tulsa, OK) have been investigated for use as root-end filling materials, although the available published literature on both is limited. These two materials require light activation and resin-dentin bonding agents to attach to the tooth.

Geristore has been recommended both as a root-end filling material[93] and for use in restoring subgingival surface defects such as root surface caries, external root resorption lesions, iatrogenic root perforations, and subgingival oblique fractured roots. Clinical evaluation of Geristore as a restorative material for root caries and cervical erosions showed it to be an acceptable material.[183,372,475,548] When it is used for surgical repair of root perforations and as an adjunct to guided tissue regeneration, the results have been favorable in isolated case reports.[3,4,47,440,487] Geristore's dual-curing paste/paste formulation is a hydrophilic Bis-GMA with long-term fluoride release. Light activation for 40 seconds cures the material to approximately 4 mm. However, the top layer is harder until the material achieves uniform hardness at 1 day after activation.[512] In vitro leakage assessment of Geristore and Dyract indicates that the materials leak less than root-end fillings made of IRM, amalgam, or Super-EBA.[65,209] Geristore has a leakage pattern similar to that of MTA.[474] An acid pH significantly reduces dye leakage of Geristore.[446] These materials are less sensitive to moisture than conventional glass-ionomer cement; however, dry environments produce stronger bonds.[99] The effect of blood contamination during the bonding phase in a clinical scenario is unknown. Geristore appears to have the potential to allow regeneration of the periradicular tissue. In one study, PDL and gingival fibroblasts attached to Geristore, and the attachment improved with time and cell proliferation.[87] Studies investigating epithelial and connective tissue adherence to Geristore found clinical and histologic evidence of cellular attachment when the material was placed in subgingival cavities.[150,151,475] However, the healing response in the periradicular region is best described as unpredictable. In a study in dogs, 10 of the 18 root end–filled teeth developed abscesses. The author attributed this to the technical difficulty of placing Geristore root-end filling. However, a small number of specimens developed cementum on the root-end fillings. The cemental covering was never greater than 25% of the root-end filling surface, which was considerably less than the amount of cementum developed on both white and gray MTA.[315]

Mineral Trioxide Aggregate (MTA)

MTA (ProRoot MTA; DENTSPLY Tulsa Dental Specialties), a material developed specifically as a root-end filling,[529] has undergone numerous in vitro and in vivo investigations comparing its various properties to Super-EBA, IRM, and amalgam. In vitro sealing ability and biocompatibility studies comparing root-end filling materials have shown MTA to be superior to other commonly used materials.[277,300,527,530,531,533-535] When various in vitro leakage models were used, MTA prevented leakage as well as composite resin and GIC.[6,129,171,591] However, the setting and subsequent leakage of MTA are not affected by the presence of blood.[527] Torabinejad and colleagues[535] developed the original product (gray MTA). The main constituents of this material are calcium silicate ($CaSiO_4$), bismuth oxide (Bi_2O_3), calcium carbonate ($CaCO_3$), calcium sulfate ($CaSO_4$), and calcium aluminate ($CaAl_2O_4$). Hydration of the powder produces a colloidal gel that solidifies into a hard structure consisting of discrete crystals in an amorphous matrix. The crystals are composed of calcium oxide, and the amorphous region is composed of 33% calcium, 49% phosphate, 2% carbon, 3% chloride, and 6% silica.[529] In a study comparing the setting time, compressive strength, radiopacity, and solubility of MTA to those of amalgam, Super-EBA, and IRM, MTA was found to be less radiopaque than amalgam but more radiopaque than Super-EBA and IRM.[529] MTA had the longest setting time (2 hours, 45 minutes) and the lowest compressive strength at 24 hours after mixing (40 MPa), although compressive strength increased to 67 MPa at 21 days after mixing. The solubility of MTA after setting was similar to that of amalgam and Super-EBA. Initially MTA has a pH of 10.2, which rises to 12.5 3 hours after mixing.[529] The pH has been reported to be approximately 9.5 at 168 hours after mixing.[152] MTA is less cytotoxic than amalgam, Super-EBA, or IRM root-end fillings.[531] Endodontic surgery studies in dogs and monkeys have reported less periradicular inflammation and cementum deposition immediately adjacent to the root-end filling material.[27,173,236,528,532] Holland and colleagues[236,237] theorized that the tricalcium oxide in MTA reacts with tissue fluids to form calcium hydroxide, resulting in hard-tissue formation.

The importance of the presence of cementum-like tissue adjacent to MTA cannot be understated. Cementum deposition is essential to regeneration of the periodontal apparatus.[310] Augmentation of new cementum across the root end and root-end restoration is essential for ideal healing of the periodontium. A layer would also enhance the integrity of the apical barrier, making it more resistant to penetration by microorganisms; in effect, establishing a biologic barrier.[22] This is seen most frequently in sections where MTA was used as the filling material. MTA appears to be able to induce cementoblastic

cells to produce hard tissue. Cementogenesis in the presence of MTA has been evaluated by assessment of the expression of osteocalcin (OCN), cell growth, and the morphology of cementoblast-like cells.[522] Scanning electron microscope (SEM) analysis indicated that cementoblasts could attach to and grow on MTA. In addition, strong expression of the OCN gene was seen after application of MTA. MTA can also increase the production of both proinflammatory and anti-inflammatory cytokines from osteoblasts. The clinical significance of this reaction is not known. The effect of MTA on periradicular tissues probably is partly due to these reactions.

In a human outcomes assessment study comparing ProRoot MTA to IRM, the rate of persistent disease with MTA was 16% at 12 months and 8% at 24 months.[103] The rate of persistent disease with IRM was 24% at 12 months and 13% at 24 months. These authors concluded that the use of MTA as a root-end filling material resulted in a high success rate that was not significantly better than that obtained with IRM. A prospective clinical trial using MTA as a root-end filling material along with current microsurgical techniques reported 89% clinical success, with follow-up time ranging from 4 to 72 months.[468]

A variation of the original formula of gray MTA has been introduced. This material, which is a white cream color, is often called white MTA. The chemical composition of white MTA is very similar to that of the original. White and gray ProRoot-MTA materials differ by less than 6% in any one component. Both are fine powders with a mean particle size of approximately 10 μm (the range in particle size is approximately 0.1 to 100 μm). The radiopacity of both materials is equivalent to approximately 3.04 mm of aluminum.[45] When white MTA was implanted in the subcutaneous connective tissue of rats, the results were similar to those reported for gray MTA.[238] One study compared the tissue reaction evoked by the two materials when used as root-end fillings in canines.[315] The only statistically significant difference observed was for the presence of macrophages or multinucleated giant cells adjacent to material. Gray MTA had more samples with mild to moderate infiltration of macrophages or multinucleated giant cells, and white MTA had more samples with no macrophages and/or multinucleated giant cells adjacent to the material. All other parameters assessed were essentially the same.

Bioceramics

Bioceramics are a relatively new and potentially promising addition to the group of materials available for root-end filling. In vitro testing of EndoSequence Root Repair Material (ERRM; Brasseler, Savannah, GA) demonstrates biocompatibility and antimicrobial activity that is similar to MTA.[107,122,317,323] ERRM is composed of calcium silicates, monobasic calcium phosphate, and zirconium oxide.[107] The material is hydrophilic, radiopaque, and has high pH. ERRM is available as a putty and a syringable paste. Because this is a relatively new material, long-term clinical studies are not yet available.

Overview of Root-End Filling Materials

Many different materials have been advocated for use as root-end filling materials, and each has specific advantages and disadvantages. However, from the biologic perspective of regeneration of the periradicular tissues, MTA, followed by Retroplast, appears to have a clear advantage over the other available materials. Bioceramic materials may join this group, but require more clinical testing. Retroplast and other composite resin–based filling materials require meticulous hemostasis and a dry surgical field for optimum results. The most commonly cited disadvantage of MTA is its handling properties. Even when properly prepared, MTA is more difficult to place in the root-end cavity than most other materials. Several devices have been modified or developed specifically for use with MTA (see Figs. 9-19 to 9-23 and 9-46). Typical clinical cases showing the surgical procedures described in the previous sections are presented in Figures 9-47 to 9-49.

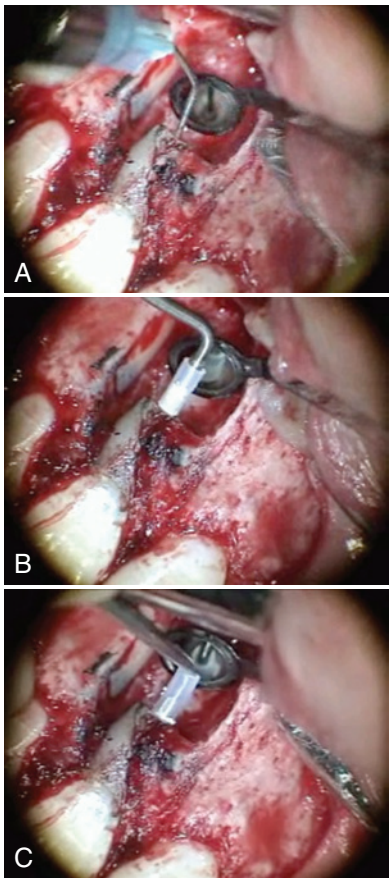

FIG. 9-46 A, Stropko syringe used to dry a root-end preparation before placement of the root-end filling material. B, Clinical use of an MTA delivery system (Dentsply Tulsa Dental). The device is loaded with MTA and placed over the root-end preparation. C, Pressing the plugger into the sleeve delivers the filling material to the root-end cavity preparation. The filling material then is compacted with microcondensers, and additional filling material is placed as needed.

CLOSURE OF THE SURGICAL SITE AND SELECTION OF SUTURE MATERIAL

Closure of the Surgical Site

The surgical site should be closed only after careful visual and radiographic inspection of the area. Before suturing, a radiograph should be taken with the flap held loosely in place to detect any foreign objects in the crypt or adhering to the flap. This image is also important for confirming the depth and density of the root-end filling. The osteotomy site then is gently curetted and irrigated with sterile saline or water to remove any remnants of hemostatic agents and packing

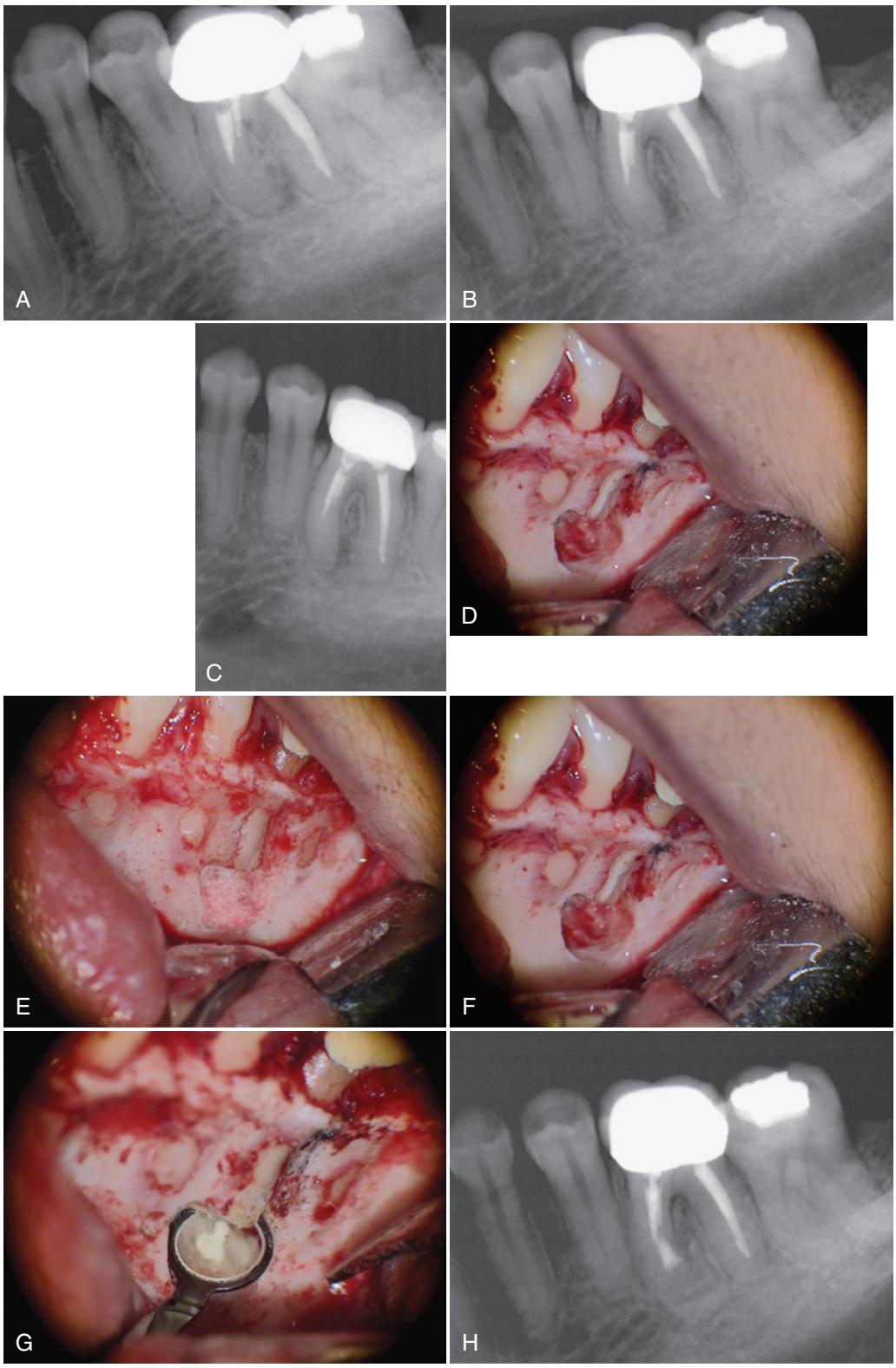

FIG. 9-47 A, Preoperative radiograph (mesial angle) of a left mandibular first molar. Periradicular disease and symptoms had persisted after nonsurgical retreatment by an endodontic resident. The mesial canals were completely obstructed at the midroot level. The preoperative evaluation included three periapical radiographs, two different horizontal angulations, and one vertically positioned radiograph. B, Straight view preoperative radiograph. C, Vertical periapical view preoperative radiograph. D, Osteotomy and root resection perpendicular to the long axis of the root were performed; a partial bony dehiscence over the mesial root can be seen. E, Racellets were packed into the bony crypt to establish hemostasis. F, The Racellets were removed (one usually is left in the deepest part of the crypt during root-end preparation and filling), and the root end was beveled perpendicular to the long axis of the root. Methylene blue dye can be useful for identifying the root outline and locating any cracks. Ultrasonic root-end preparation was completed to a depth of 3 mm, connecting the MB and ML canals. G, MTA root-end filling was placed and inspected. The bony crypt was then gently curetted to initiate bleeding and to remove any remnants of hemostatic materials. The flap was repositioned, and a radiograph was taken. H, An immediate postoperative radiograph confirmed the depth and density of the root-end filling and the absence of any foreign objects. Note that calcium sulfate and bone grafting material was placed in the osseous defect and over the root because of the large buccal dehiscence, although this is not routinely required in these cases. (Courtesy Dr. Vince Penesis.)

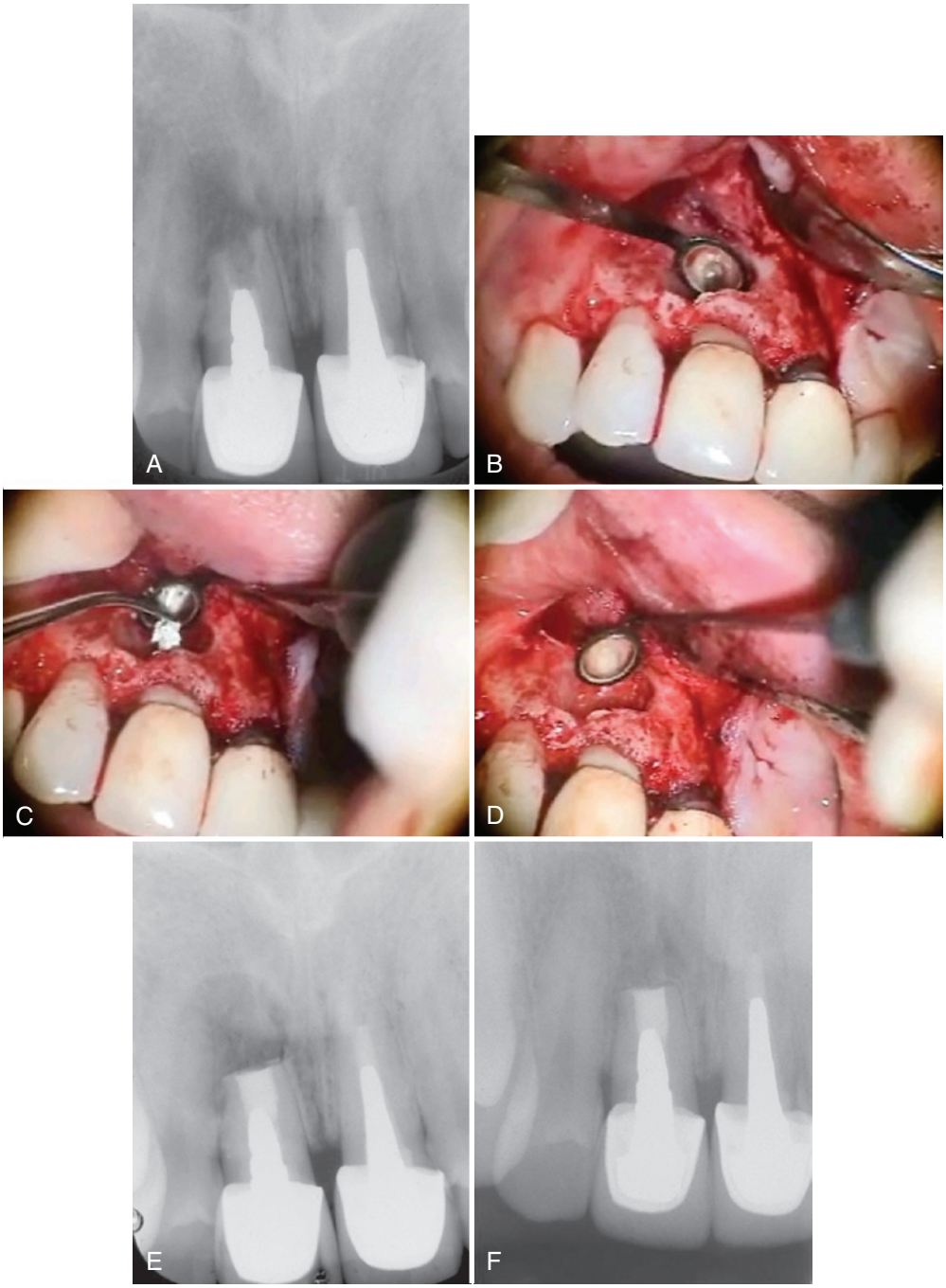

FIG. 9-48 A, Preoperative radiograph of a maxillary left central incisor showing evidence of previous surgery but no apparent root-end filling. Because of the short root length and inadequate band of keratinized gingiva, an intrasulcular incision and full-thickness triangular mucoperiosteal flap design were selected. B, The root was minimally resected, and the root-end cavity was prepared ultrasonically. C, MTA was placed and condensed into the root-end cavity preparation. D, The root-end filling was inspected before the flap was repositioned and sutured. E, Immediate postoperative radiograph. F, The 6-month follow-up radiograph showed good initial periradicular healing. (Courtesy Dr. Shawn Velez.)

materials. Some bleeding is encouraged at this point, because the blood clot forms the initial scaffold for subsequent healing and repair. If indicated, grafting materials or barriers may be placed at this time. Slight undermining of the unreflected soft tissue adjacent to the flap facilitates the placement of sutures. The flap is then repositioned and gently compressed with a piece of chilled, sterile moist cotton gauze to express excess blood and tissue fluids.

For the common flap designs discussed in this chapter, the corners are first identified and sutured in place with a single interrupted suture. Interrupted sutures are initially passed through the free portion of the flap approximately 2 to 3 mm

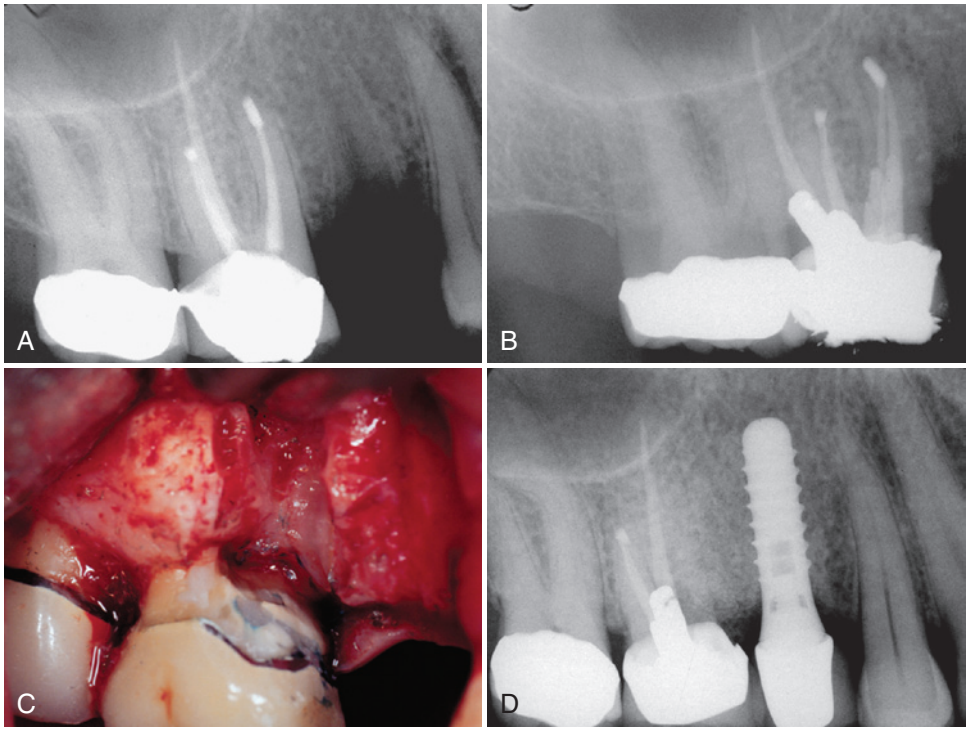

FIG. 9-49 **A,** A maxillary right first molar that was sensitive to percussion and tender to palpation over the MB root. Periodontal probing revealed a deep, narrow bony defect on the facial aspect of the MB root. The presence of a vertical root fracture was confirmed visually using magnification and methylene blue dye. The maxillary right second premolar had been recently extracted because of a vertical root fracture. **B,** A bonded amalgam core buildup was placed in the DB and P canals, and Geristore was placed in the MB canal system. **C,** The MB root was resected, and methylene blue dye was used to help define the extent of the fracture. **D,** At the 3-year follow-up visit, a new crown had been fabricated for the molar, and the premolar had been replaced with an implant.

from the edge and then connected to the attached tissue. The suture is secured with a simple surgeon's knot, which is positioned away from the incision line. The center of the flap then is located and sutured with either an interrupted or a sling suture. A continuous locking suture technique may be used to close a submarginal (Ochsenbein-Luebke) flap.[288] The primary advantage of a continuous suture technique is the ease of suture removal compared with multiple interrupted sutures. The disadvantages are possible difficulty with precise control of tension in each area, and the fact that the entire suture may loosen if one suture pulls through the flap. A sling suture is commonly used for the central tooth in the surgical site to close a full-thickness intrasulcular (rectangular or triangular) flap. The tension on this type of suture can be varied slightly to allow some control of the apicocoronal positioning of the flap. Interrupted sutures then are placed as needed.

When suturing is complete, chilled, sterile moist cotton gauze is again placed over the flap and pressure is applied for 5 minutes. Pressure to the area provides stability for the initial fibrin stage of clot formation and reduces the possibility of excessive postoperative bleeding and hematoma formation under the flap. The iced gauze also supports hemostasis. Final inspection of the area should confirm that all soft-tissue margins have been closely approximated and bleeding has been controlled. An additional injection of long-acting local anesthetic may be administered at this time, although care must be taken not to inject it directly under the newly repositioned flap. The patient is given a cold compress and instructed to hold it on the face in the surgical area, on for 20 minutes and then off for 20 minutes, for the rest of the day. The patient also is given verbal and written postoperative instructions, including after-hours contact information (Fig. 9-50). The patient should sit in an upright position for approximately 15 minutes and the surgical site should be inspected one more time before the patient is discharged.

Selection of the Suture Material

The properties of an ideal suture material for periradicular surgery include pliability for ease of handling and knot tying, a smooth surface that discourages bacterial growth and wicking of oral fluids, and a reasonable cost. Suture material in size 5-0 is most commonly used, although some clinicians prefer slightly larger (4-0) or smaller (6-0) suture. Sutures smaller than 6-0 tend to cut through the relatively fragile oral tissues when tied with the tension required to approximate the wound margins. Silk suture material has been commonly used in dental surgery for decades and is both inexpensive and easy to handle. However, silk tends to support bacterial growth and allows for a wicking effect around the sutures. For these reasons, other materials are preferable to silk.[92]

Resorbable suture materials (plain gut and chromic gut) are not routinely used for periradicular surgery, although this material may be indicated if the patient will be unavailable for the regular suture removal appointment (48 to 96 hours after

Care of Your Mouth After Endodontic Surgery

1.) Apply an ice pack to your face next to the surgery area (on for 20 minutes and off for 20 minutes) for the next 5 to 6 hours to help decrease postoperative swelling. Swelling is usually greatest the day after surgery and may be at its worst 2 or 3 days after surgery.

2.) Take all medications as directed. Approximately 45 minutes should be allowed for you to feel the effect of pain medication.

3.) Clean your mouth as usual (brushing, flossing, etc.) in all areas except the surgical site. Modify cleaning procedures of the teeth in the area of the surgical site to keep from disturbing the area. Do not rinse vigorously during the first 24 hours following surgery. Continue using the prescribed mouth rinse twice a day until after you return to have the sutures removed.

4.) Minor oozing of blood from the surgical site may occur in the first 24 hours after surgery. This will produce a pink tinge in the saliva and is not a cause for concern. However, if bleeding is excessive, please contact our office. You may apply pressure to the area with a tea bag or moist cotton gauze.

5.) Sutures have been placed and will need to be removed at your next appointment. Please do not lift or pull on your lip to examine the surgical site during the first 2 to 3 days because this may disturb the healing process.

6.) A soft diet is recommended for the first 2 or 3 days. Try to avoid foods that are hot, spicy, or hard to chew. It is very important that you drink plenty of fluids (nonalcoholic). This will help your mouth heal.

7.) Avoid cigarettes and all other tobacco products.

8.) A slight increase in body temperature may occur during the first 24 hours following surgery. This is normal. Infection after endodontic surgery is not typical but can occur. If an infection develops, it usually occurs 2 to 3 days after the surgery. Signs of an infection include sudden increase in pain or swelling, feverish feeling, sore glands in the neck area, and a general flulike feeling. If you think an infection has developed, please contact the office immediately.

If you have any questions, please contact the office during normal office hours at 312-XXX-5555. If you are experiencing problems after office hours, you may contact Dr. _____ at: 312-XXX-1212.

FIG. 9-50 Example of postoperative instructions. Written instructions provide an essential reference for the patient, because verbal instructions often are difficult to remember after surgery. The instructions may be modified as needed; it is important to provide instructions that the patient can understand. For example, the readability of these instructions is at approximately the eighth grade level using the Flesch-Kincaid Grade Level scale.

surgery) or if the suture will be used in areas of the mouth where access is difficult. The primary problem with resorbable suture materials is the variable rate of resorption—that is, sutures may weaken and dissolve too soon or, more commonly, remain in the incision area for longer than desired. Gut suture materials are packed in isopropyl alcohol. The handling properties of gut sutures can be improved by immersion in sterile water for 3 to 5 minutes before use.[430]

Suture materials with a smooth Teflon or polybutilate coating (e.g., Tevdec and Ethibond, respectively) are particularly well suited for use in periradicular surgery. Synthetic monofilament suture materials (e.g., Supramid and Monocryl) are also commonly used. These materials are easy to handle and do not promote bacterial growth or wicking of oral fluids to the same extent as silk. Gortex (expanded PTEE-Teflon) sutures have many desirable properties but are more expensive than the previously mentioned materials.

Tissue adhesives such as cyanoacrylate and fibrin glues may hold promise for wound closure after periradicular surgery.[113,196,403,599] Although the currently available research is insufficient to recommend these adhesives as a routine replacement for more traditional suture materials, future applications in periradicular surgery are possible.

GUIDED TISSUE REGENERATION AND ENDODONTIC SURGERY

The amount and location of bone adjacent to the root structures affect the prognosis of periradicular surgery. Kim and Kratchman[282] propose a six-category classification system to assist in predicting surgical prognosis and determining the need for bone grafting and barrier techniques. Class A (no lesion), class B (small periapical lesion), and class C (large periapical lesion without periodontal communication) all represent situations that are favorable for healing without supplemental grafting or barriers. Class D (similar to class C with independent periodontal pocketing), class E (endodontic-periodontal communication to the apex), and class F (apical lesion with complete loss of buccal bone) represent situations with a more guarded prognosis and usually require concurrent use of bone grafting and barrier techniques. Figs. 9-51 to 9-63 are examples of cases that required guided tissue regeneration (GTR).

An apicomarginal defect[141] or a localized bony defect distinguished by a total deficiency of alveolar bone over the entire root length has a significant adverse effect on the outcome, reducing the rate of complete healing by approximately 20%

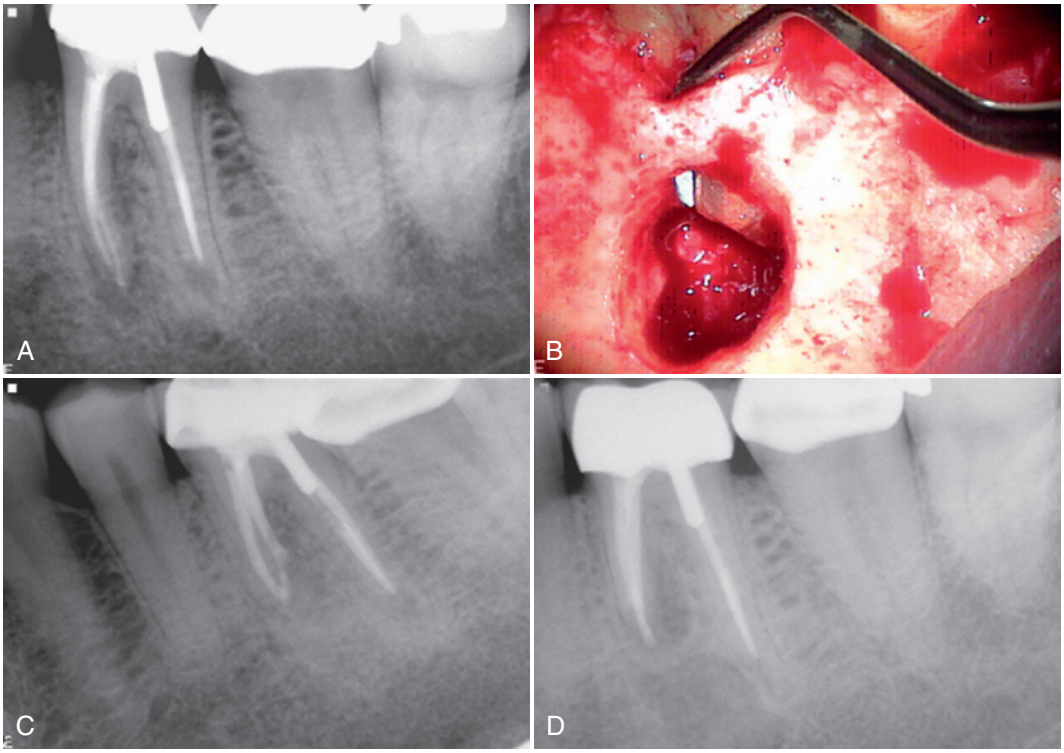

FIG. 9-51 **A,** Preoperative periapical radiograph of tooth #19. **B,** Clinical image demonstrating periodontal defect along the facial aspect of the mesial root. **C,** Immediate postsurgical radiograph. The mesial root end was prepared with ultrasonics and filled with MTA. A mixture of DFDBA and Capse was placed for guided tissue regeneration. **D,** One-year follow-up radiograph demonstrating good healing. Periodontal probings were WNL.

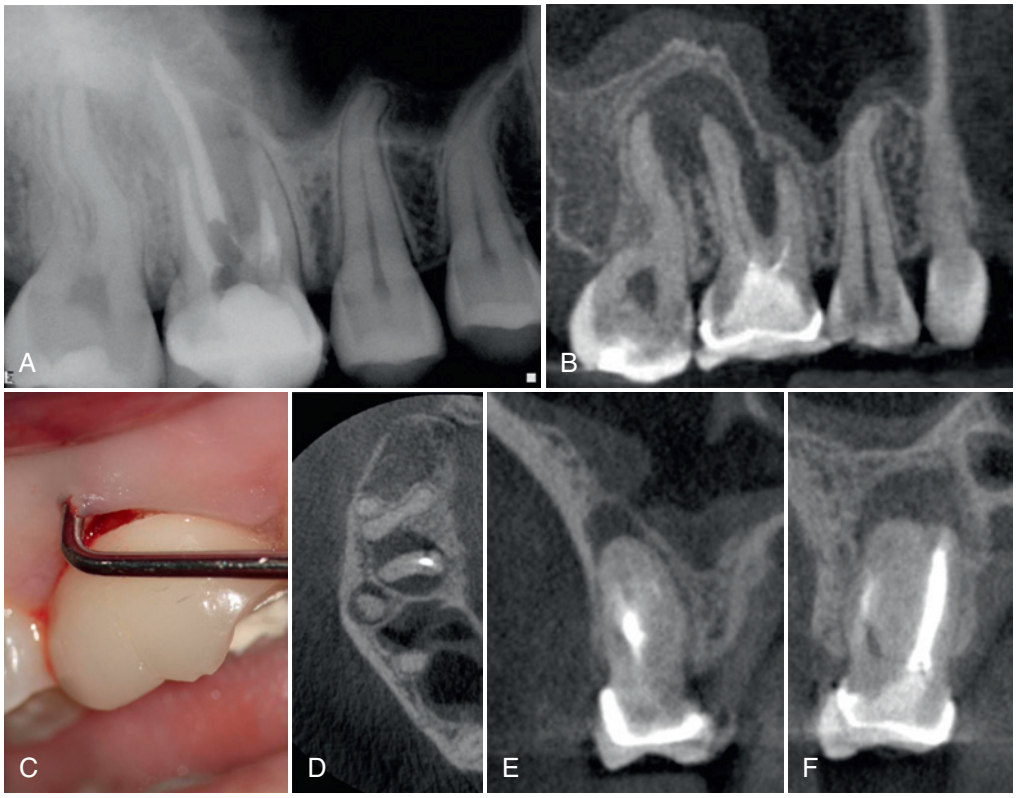

FIG. 9-52 (Case 1): **A,** Periapical radiograph of tooth #3. Retreatment RCT was attempted, but MB1 and MB2 were blocked. **B,** Sagittal view demonstrating the extent of the periapical lesion. **C,** Clinical view of periodontal probing to the apex. **D,** Axial view of the apical one third demonstrating the close proximity of the periapical defect to the MB root of tooth #2 (#2 responded WNL to pulp vitality testing). **E, F,** Coronal views of the MB and DB/P roots, respectively.

FIG. 9-53 (Case 1, continued): **A,** A 3D reconstruction of the tooth #3 area demonstrating the furcation periodontal defect. **B,** After flap reflection, clinical view demonstrating the periodontal defect. **C,** A 3D reconstruction with the buccal plate cropped to visualize the extent of the periradicular defect. **D, E,** DB and P root resection, root-end preparation, root-end filling with MTA. **F,** Periradicular defect grafted with Puros allograft (Zimmer Dental, Carlsbad, CA) and CopiOs pericardium membrane (Zimmer Dental).

or more when compared to teeth with an isolated endodontic-only lesion.[233,279,499] The presence of a periradicular lesion 15 mm or greater in diameter also has been linked to a poorer prognosis.[233] Advanced periodontitis with deep pocket formation has been associated with chronic periradicular inflammation after endodontic surgery and subsequent failure of the root-end surgery.[450] The cause of failure has been identified as in-growth of nonosteogenic tissues into the periradicular surgical site and down-growth of epithelial tissue along the root surface. Successful treatment may depend more on controlling epithelial proliferation than root-end management. Guided tissue regeneration techniques have been advocated for use in such cases.[206,547]

The basic principle of guided tissue and bone regeneration is that different types of cells repopulate a wound at different rates during healing. The soft-tissue cells are considerably more motile than the hard-tissue cells, therefore they tend to migrate into the wound more quickly during healing. A barrier interposed between the gingival tissue and the exposed root surfaces and supporting alveolar bone prevents colonization of the exposed root surface by gingival cells. This encourages selective repopulation of the root surface by PDL cells. The use of an absorbable barrier theoretically would allow PDL cells and other cells with osteogenic potential to repopulate the defect, resulting in new connective tissue attachment and bone formation. Dahlin and coworkers[119,120] demonstrated that in monkeys, a significant increase in osseous healing occurs when membranes are used in through-and-through bone defects in periradicular surgery of the lateral maxillary incisors. The use of resorbable guided tissue regeneration (GTR) membranes in endodontic surgery with buccal apicomarginal type defects also has been shown to enhance regeneration of the periodontium and surrounding bone in dogs.[149] This type of matrix barrier promoted greater amounts of connective tissue and alveolar bone and minimized the formation of junctional epithelium.

Several case reports have discussed the use of guided tissue regeneration techniques in conjunction with endodontic surgery.[5,32,81,123,154,273,337,414,421,432,515,544,549,607] These studies largely have reported favorable outcomes in cases involving large periradicular lesions, through-and-through bone defects, and repair of a surgical perforation or loss of the buccal cortical plate adjacent to the root.

Pecora and colleagues[400] compared the healing of 20 large periradicular defects (greater than 10 mm diameter) with and without the use of resorbable membrane. They reported that

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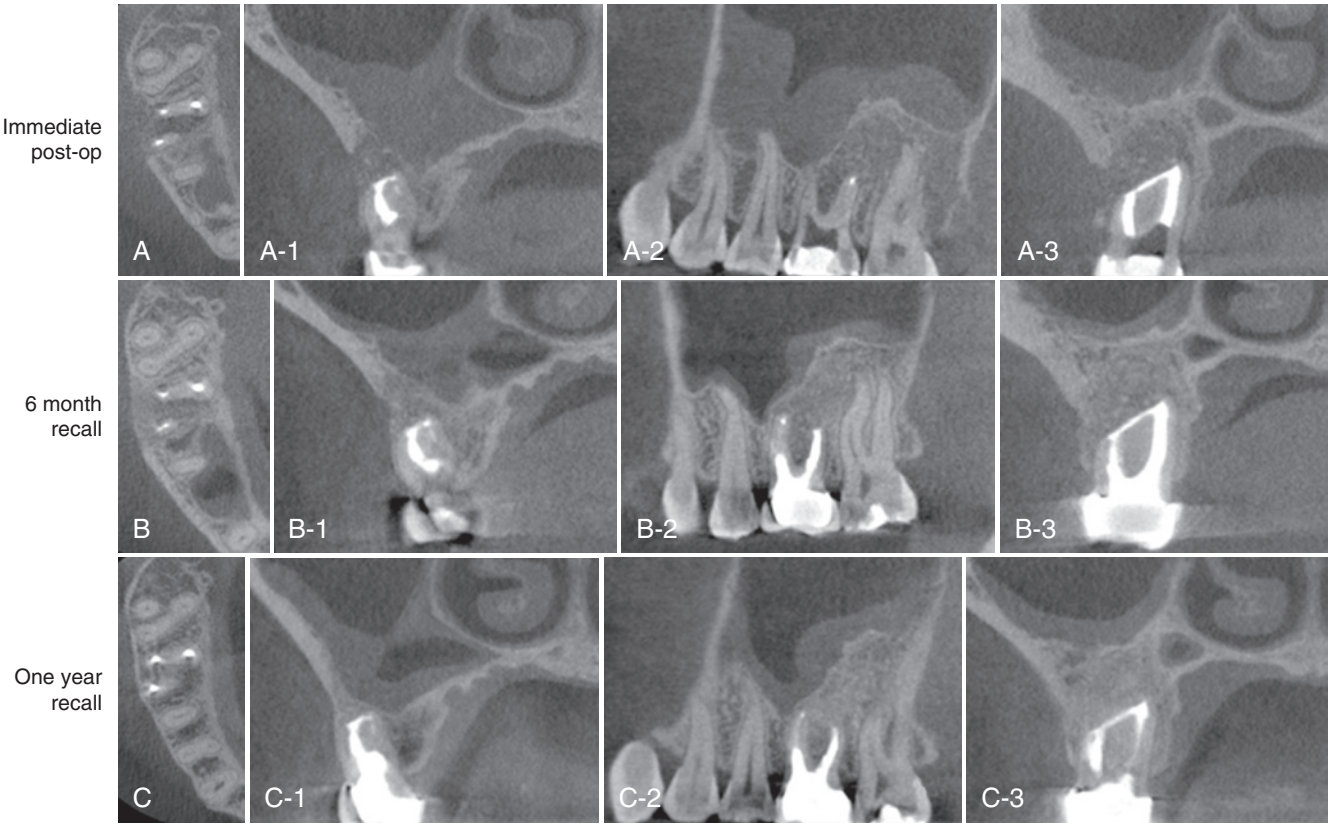

FIG. 9-54 (Case 1, continued): **A,** Axial view of immediate postoperative CBCT scan, A-1 coronal view of MB root, A-2 sagittal view and A-3 coronal view of DB and palatal roots. **B,** A 6-month recall, axial view, B-1 coronal view of MB root, B-2 sagittal view, and B-3 coronal view of DB and palatal roots. **C,** A 1-year recall, axial view, C-1 coronal view of MB root, C-2 sagittal view, and C-3 coronal view of DB and palatal roots.

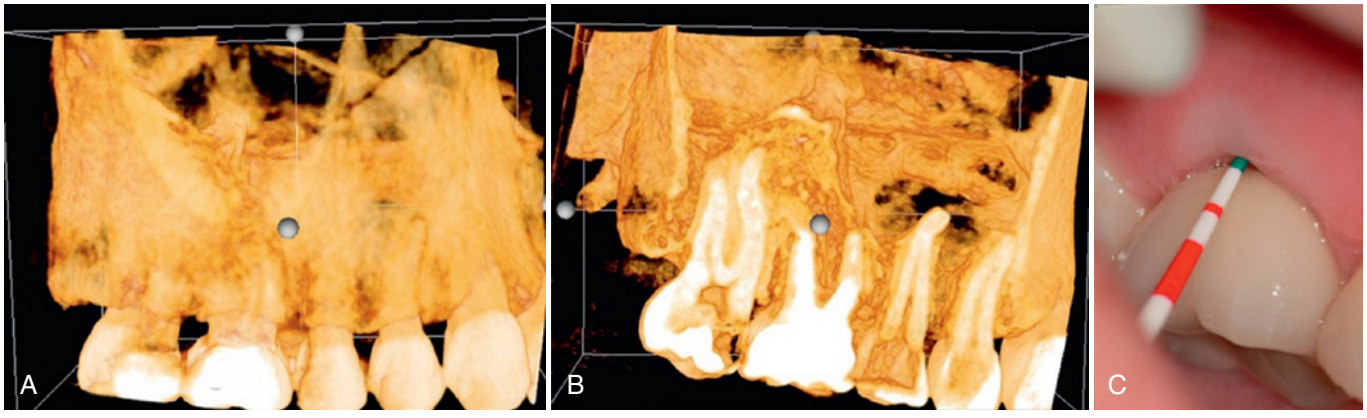

FIG. 9-55 (Case 1, 1-year recall): **A, B,** A 3D reconstruction of 1-year recall. **C,** Clinical picture showing resolution of the periodontal defect.

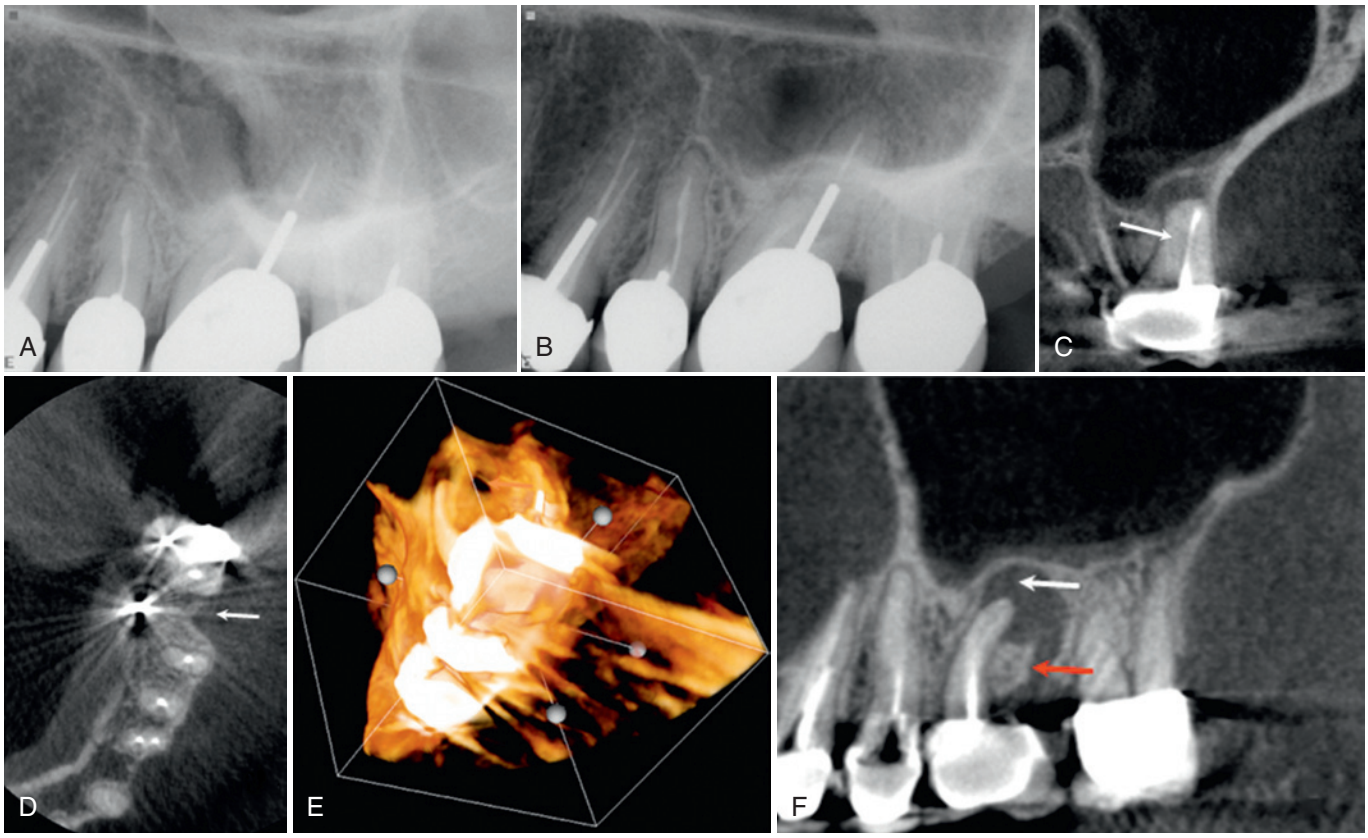

FIG. 9-56 (Case 2, tooth #14): **A, B,** Periapical radiographs of tooth #14, mesial and distal angles, respectively. **C,** Coronal view of the MB root demonstrating a missed MB2 canal *(arrow).* **D,** Axial view showing a previous DB root amputation site *(arrow)* that was not obvious in the periapical radiograph. **E,** A 3D reconstruction demonstrating a crestal defect. **F,** Sagittal view demonstrating the crestal defect *(red arrow)* communicating with the periapical lesion *(white arrow),* elevation of the floor of the maxillary sinus but no evidence of sinus perforation.

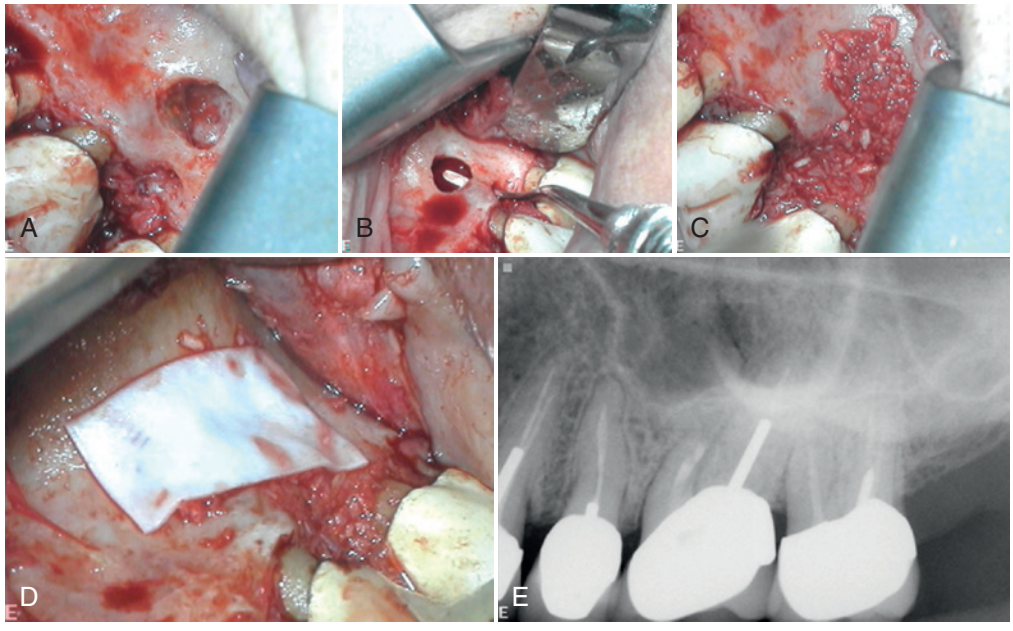

FIG. 9-57 (Case 2, continued): **A,** Clinical view after flap reflection demonstrating both crestal and periapical lesions. **B,** Communication between both defects. **C,** Both defects grafted with EnCore Combination Allograft (Osteogenics Biomedical, Lubbock, TX). **D,** CopiOs pericardium membrane (Zimmer Dental, Carlsbad, CA). **E,** Immediate postoperative radiograph.

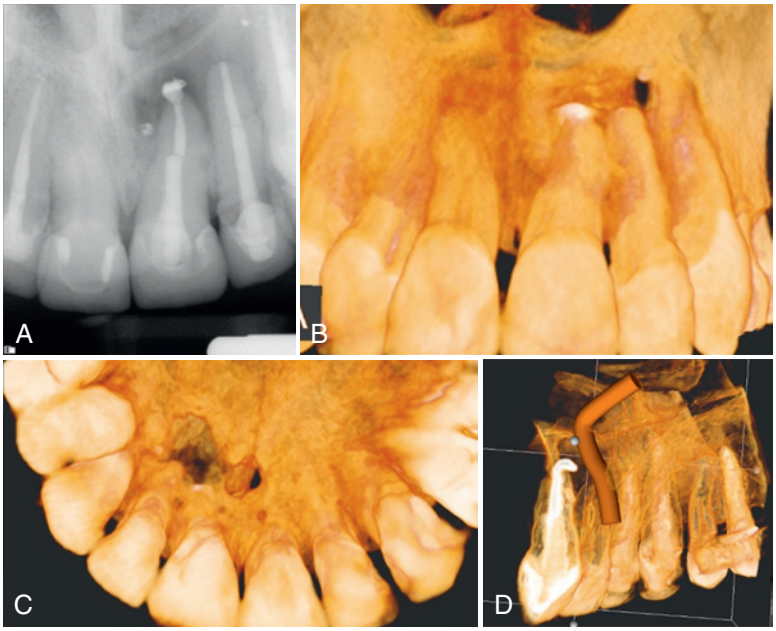

FIG. 9-58 (Case 3): **A,** Periapical radiograph of maxillary anterior region demonstrating a periapical lesion associated with tooth #9. **B,** CBCT reconstruction demonstrating the intact buccal cortical plate. **C,** Palatal view of the 3D reconstruction demonstrating perforation of the palatal plate. **D,** A 3D reconstruction showing the nasopalatine bundle.

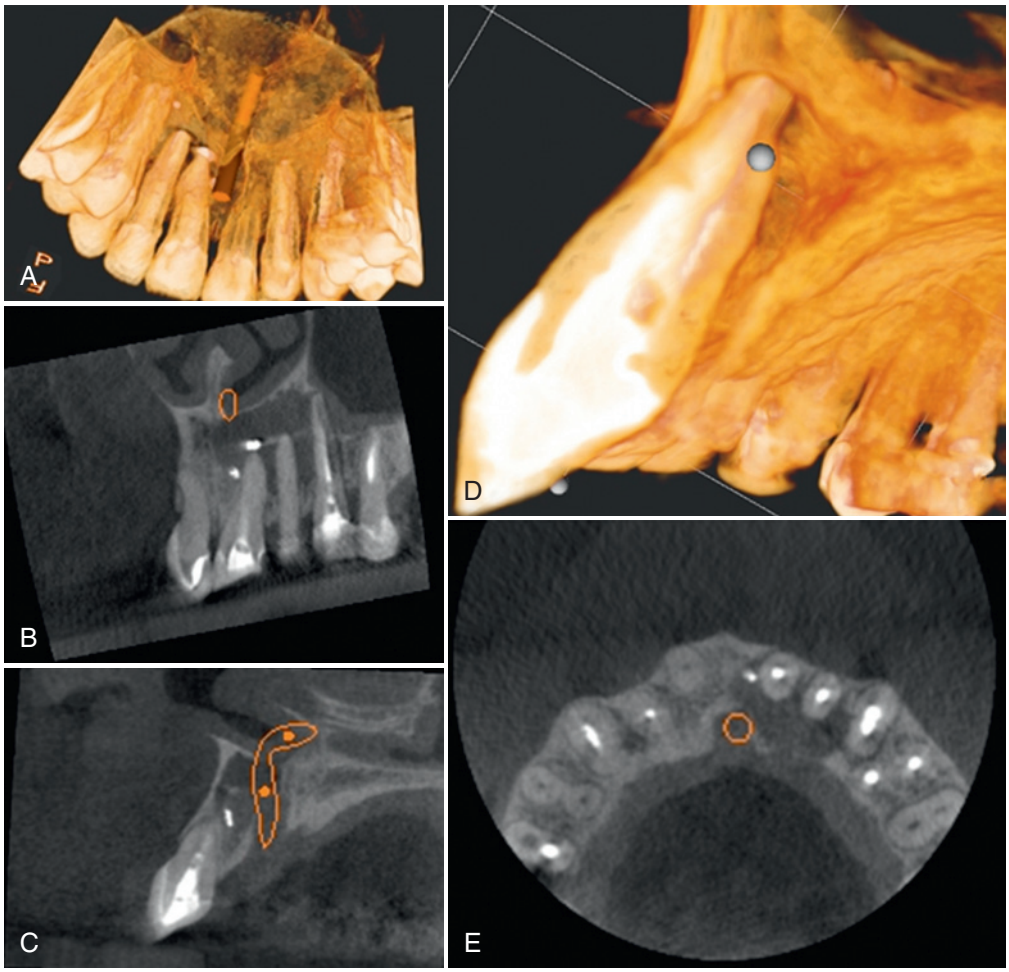

FIG. 9-59 (Case 3, continued): **A,** Palatal view CBCT reconstruction showing the exit of the nasopalatine neurovascular bundle from the incisive canal. **B,** Sagittal view demonstrating the periapical radiolucency involving teeth #9, 10, and 11. **C,** Coronal view the exit of the nasopalatine bundle from incisive canal. **D,** A 3D reconstruction demonstrating the periapical lesion extension to tooth #11. **E,** Axial view demonstrating the extension of the lesion, palatal plate perforation, and relation of the nasopalatine bundle to the periapical lesion.

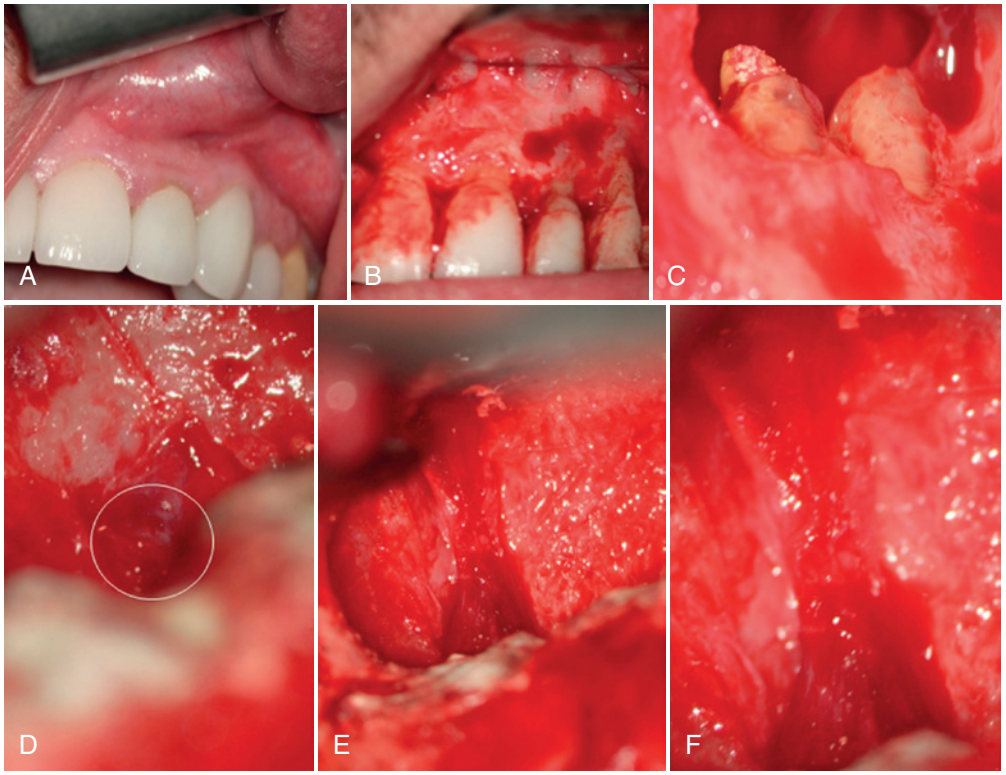

FIG. 9-60 (Case 3, continued): **A, B,** Clinical pictures before and after flap reflection showing the intact buccal plate. **C,** Periapical defect after degranulation showing the apices of teeth #9 and 10 prior to resection. **D,** Palatal bone perforation with palatal mucosa evident *(circle).* **E, F,** Clinical pictures of the nasopalatine bundle intact after degranulation.

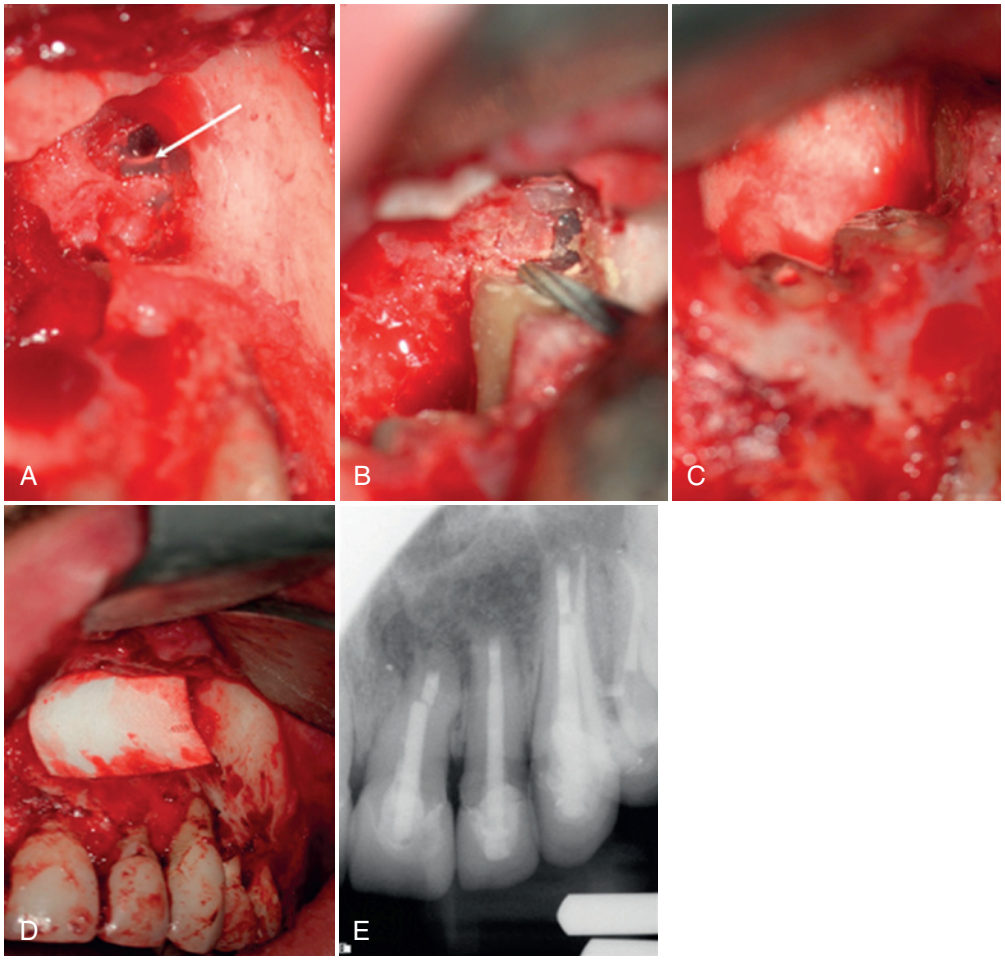

FIG. 9-61 (Case 3, continued): **A, B,** Lateral wall of the maxillary sinus distal to tooth #11. **C,** CopiOs membrane placed palatally to cover the palatal mucosa. **D,** CopiOs membrane covering the Puros allografting material. **E,** Through-and-through defect immediate postoperative radiograph.

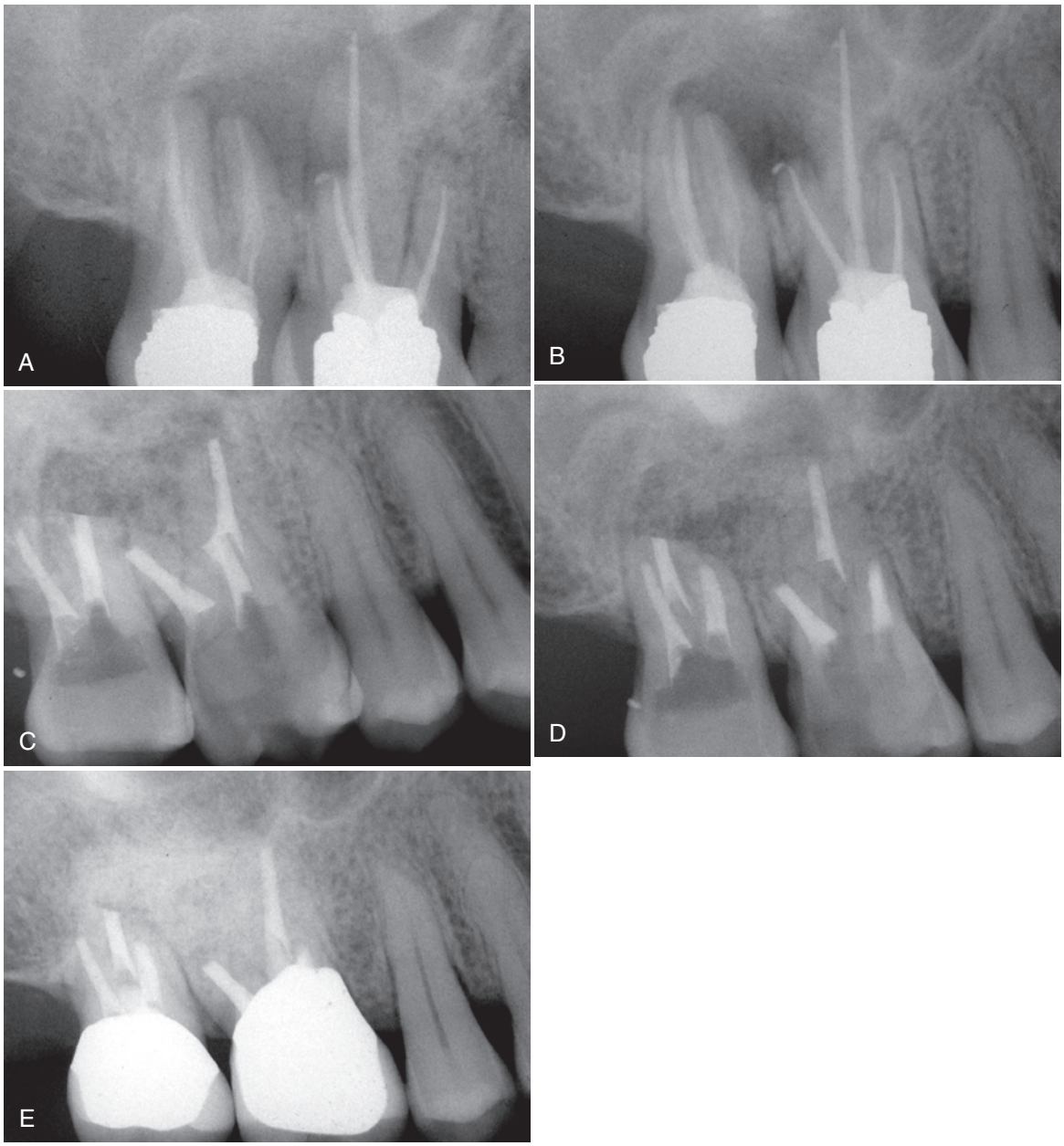

FIG. 9-62 **A,** Preoperative angled radiograph of maxillary right first and second molars. Both teeth had been treated previously, and the patient has reported a history of pain in the area for the past 5 years. The treatment plan included nonsurgical retreatment followed by periradicular surgery with bone grafting and guided tissue regeneration (GTR). **B,** Preoperative straight-on radiograph of the maxillary right first and second molars. **C,** Immediate postoperative radiograph showing root-end resections and fillings. The root ends were prepared with ultrasonics, conditioned with 17% ethylenediaminetetraacetic acid (EDTA), filled with Diaket, and smoothed with a superfine diamond finishing bur. The crypt was packed with BioOss xenograft material, and a Guidor resorbable membrane was placed. **D,** Immediate postoperative radiograph (straight on view). **E,** A 4-year follow-up radiograph. The patient was asymptomatic, and all objective findings were within normal limits. The teeth were restored with porcelain fused to metal crowns.

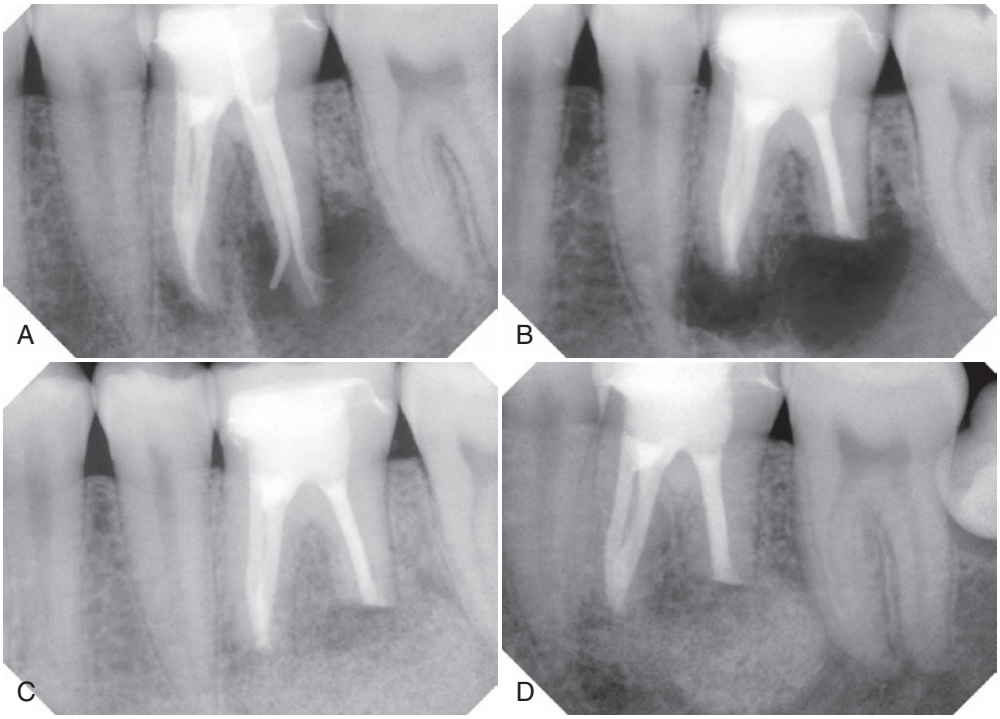

FIG. 9-63 A, Preoperative radiograph of a mandibular left first molar. Gutta-percha was inserted into the buccal sulcus and traced to the apex of the distal root. Nonsurgical root canal treatment had been performed 12 months earlier. B, Root-end resection and MTA root-end fillings (M and D roots). C, Immediate postoperative radiograph. BioOss xenograft material was placed. D, The 19-month follow-up radiograph showed good periradicular healing.

at 12 months after surgery, the sites in which membranes had been used had healed more quickly and that the quality and quantity of the regenerated bone was superior. One study evaluated periradicular and periodontal healing in cases involving apicomarginal defects when guided tissue regeneration (Bio-Oss and Bio-Gide membrane; Osteohealth Co., Shirley, NY) was performed in conjunction with periradicular surgery. At 12 months after surgery, 86% were considered healed clinically and radiographically. It was concluded that GTR should be considered as an adjunct to periradicular surgery in cases of apicomarginal defects.[142] However, use of a resorbable membrane when a standard apical osteotomy is performed and the buccal bone over the remainder of the root is intact has no beneficial effect on healing.[186]

Several different types of membranes are available. They can be grouped into two broad categories, nonresorbable and resorbable (Table 9-1). Resorbable membranes are generally better suited for endodontic uses because a second surgical procedure is not required to remove the membrane.

Membranes frequently require support so that the membrane does not collapse into the defect itself. Support for the membrane may be provided by using either a titanium-tented membrane or a graft material. Graft materials have two main functions: to act as a mechanical substructure that supports the membrane and the overlying soft tissues and to serve as a biologic component that enhances bone formation. Bone graft materials (Table 9-2) can be categorized as osteoconductive or osteoinductive. An osteoconductive material provides a framework into which bone can grow. The pore size of the material is similar to that of normal bone, and the material eventually is absorbed and remodeled. An osteoinductive material stimulates the production of new bone cells such that healing occurs more quickly. The bone morphogenic protein (BMP) family has been investigated extensively for use in this role. A combination of osetoconductive and osteoinductive materials also can be used for bone grafts.

The use of GTR techniques raises several additional issues that should be discussed with the patient before surgery. These include the cost of the additional material, the origin of the material (synthetic, animal, or human), the need to manage the wound for a longer period, and potential postoperative complications related specifically to these techniques and materials. Discussion of the composition of the materials to be used is very important, because some patients may have concerns based on religious or ethical grounds. The surgeon must discuss all the ramifications of using these materials with the patient before beginning the procedure, because it is not always possible to predict before surgery when grafting materials may be needed.

If GTR techniques are to be used during periradicular surgery, a resorbable membrane should be chosen and a protocol should be followed (Figs. 9-62 and 9-63):

1. The membrane is extended to cover 2 to 3 mm of bone peripheral to the margins of the crypt; it should be supported with a bone substitute graft material so that it does not collapse into the crypt or onto underlying tooth structures.
2. Tissue closure techniques should ensure total tissue coverage of the membrane. The traditional postoperative compression is eliminated, because this would collapse the membrane onto the underlying structures.
3. Smoking is contraindicated with GTR techniques because it consistently has been shown to affect the outcome adversely.[73,324,445,525,538,539]

TABLE 9-1

Examples of Membrane Materials

Composition	Trade Name/Manufacturer
Nonresorbable	
Polytetrafluoroethylene	Gortex (WL Gore & Associates Inc, Flagstaff, AZ) TefGen FD (Lifecore Biomedical, Chaska, MN) Bicon Barrier Membrane (Bicon, Boston, MA) Cytoflex (Unicare Biomedical, Laguna Hills, CA)
Resorbable	
Laminar bone	Lambone (Pacific Coast Tissues Bank, Los Angeles, CA)
Polylactic acid	Guidor* This product was used extensively in early research with very favorable results (Guidor USA) Atrisorb (CollaGenex Pharmaceuticals, Newtown, PA)
Polyglactic acid	Vicyl Mesh (Ethicon, Somerville, NJ)
Polylactic acid, polyglycolic acid, and trimethylene carbonate	Resolut (WL Gore & Associates Inc, Flagstaff, AZ)
Collagen	Biomend (Zimmer Dental, Carlsbad, CA) Bio-Guide (Osteohealth, Shirley, NY) Bicon Resorbable Collagen Membrane (Bicon, Boston, MA)

*No longer available.

TABLE 9-2

Examples of Bone Graft Materials

Graft Type	Description	Product/Manufacturer or Source
Autogenous graft	Obtained from patient's own body	Ramus, chin, iliac crest
Allograft	Demineralized freeze-dried human bone (DFDBA)	Osteofil (Regeneration Technologies, Alachua, FL) Grafton (Osteotech, Eatontown, NJ) Dynagraft (GenSci, Toronto, Ontario, Canada) Opteform (Exactech, Gainesville, FL) Puros (Zimmer Dental, Carlsbad CA) MTF DeMin Bone (DENTSPLY Friadent CeraMed, Lakewood, CO)
Xenograft	Inorganic bovine/porcine bone particles	BioOss (Osteohealth, Shirley, NY) OsteoGraf (DENTSPLY Friadent CeraMed, Lakewood, CO)
Ceramic/synthetic grafts	Calcium sulfate, calcium phosphate/ hydroxyapatite, bioactive glass	CapSet (Lifecore Biomedical, Chaska, MN) OsteoSet (Wright Medical Technology, Arlington, TN) HTR (Bioplant HTR, Kerr Corporation, West Collins, CA) Biogran (3i, Palm Beach Gardens, FL) Norian SRS (Synthes, West Chester, PA) NovaBone-C/M (NovaBone Products, LLC, Sales and Manufacturing, Alachua, FL) PerioGlas (NovaBone Products, LLC, Sales and Manufacturing, Alachua, FL)
Bioactive proteins	Bone morphogenic proteins (BMP)	Experimental
Combination graft	Allograft, xenograft, or ceramic/ synthetic grafts plus bioactive protein	PepGen P15 (DENTSPLY Friadent CeraMed, Lakewood, CO)

Ridge Preservation

With the growing use of dental implants for the replacement of missing teeth, clinicians should be aware of ridge preservation strategies, even if they do not place implants.[308] As an example, ridge preservation should be considered when a tooth is determined to have a vertical root fracture during an exploratory surgical procedure and is extracted. In this situation, there is often a complete absence of the buccal bony plate, and simple extraction of the tooth would predispose the patient to a loss of ridge height and width, thereby complicating future implant placement. GTR with graft and barrier placement (as previously described) at the time of extraction may be indicated to create a more favorable site for future implant placement.[31,251,342,608] An atraumatic extraction technique is desirable, as one of the goals is to preserve the maximum amount of existing bone. Periotomes are particularly useful for this type of bone-preserving extraction technique.

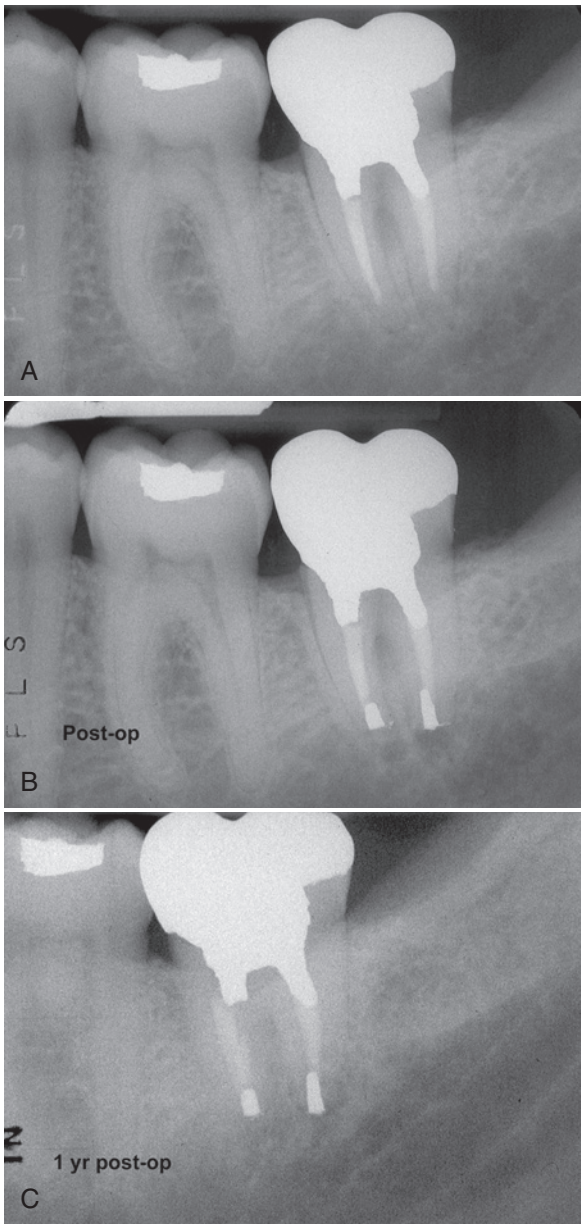

FIG. 9-64 Intentional replantation. **A**, Preoperative radiograph of a mandibular left second molar. The tooth was persistently sensitive to percussion and biting after nonsurgical retreatment. **B**, Radiograph of the tooth immediately after extraction, root-end preparation and filling, and replantation. **C**, At the 1-year follow-up visit, the tooth was asymptomatic and showed good periradicular healing. (Courtesy Dr. Matt Davis.)

INTENTIONAL REPLANTATION

Intentional replantation may be an option when surgical access is limited or presents unacceptable risks. Mandibular second molars are a common example for this technique because of the typically thick overlying buccal bone, shallow vestibular depth, and proximity of the root apices to the mandibular canal (Fig. 9-64). However, any tooth that can be atraumatically removed in one piece is a potential candidate for intentional replantation. Contraindications include teeth with flared or moderately curved roots and the presence of periodontal disease. Vertical root fracture has often been considered a contraindication,[401] although some investigators have demonstrated moderate success using a dentin-bonded resin and intentional replantation for the treatment of teeth with root fractures.[226,268,506] The prognosis was generally better for incisors and for teeth with fractures less than two thirds of the root length. Clinical success after 1 year was about 89% and decreased to 59% at 5 years.[226]

The tooth should be extracted with minimal trauma to the tooth and socket. Ideally, elevators are not used and the root surface is not engaged with the forceps. All instruments and materials for root-end preparation and filling should be arranged before extraction to minimize extraoral working time. The root surface must be kept moist by wrapping the root with gauze soaked in a physiologic solution, such as Hank's Balanced Salt Solution. After root-end preparation and filling (as described previously in this chapter), the tooth is replanted and the buccal bone is compressed. The patient may be instructed to bite on a cotton roll or other semisolid object to help position the tooth properly in the socket. Occlusal adjustment is indicated to minimize traumatic forces on the tooth during the initial stage of healing. A splint may be applied, but this is often not necessary. The patient should eat a soft diet and avoid sticky foods, candy, and chewing gum for at least 7 to 10 days. Based on clinical observations and several animal model studies, the prognosis for successful healing after replantation is most closely related to avoiding trauma to the PDL and cementum during extraction and minimizing extraoral time.[21,23,391]

POSTOPERATIVE CARE

As previously noted, NSAIDs generally are the preferred class of drugs for managing postoperative pain (also see Chapter 4).[9,41,54,143] Ibuprofen (400 to 800 mg) or an equivalent NSAID typically is given before or immediately after surgery and can be continued for several days postoperatively as needed. When additional pain relief is required, a narcotic such as codeine, hydrocodone, or tramadol may be added to the standard NSAID regimen. This strategy may result in a synergistic effect, and therefore greater pain relief, than would be expected with the separate analgesic value of each drug.[148] A useful short-term approach to the management of moderate to severe pain is a "by the clock" alternating schedule of an NSAID and an acetaminophen/narcotic combination.[250,348] Pain after periradicular surgery typically is only mild to moderate. Postoperative pain usually is managed quite well with NSAIDs only, especially when the previously recommended strategy of preoperative NSAID therapy and a long-acting local anesthetic is combined with a minimally traumatic surgical approach.

Sutures commonly are removed 2 to 4 days after surgery.[92,213] This recommendation is based on the current understanding of wound healing and the desire to remove any potential irritants from the incision area as soon as possible. Local anesthesia is rarely required, although application of a topical anesthetic may be helpful, especially to releasing incisions in nonkeratinized mucosa. Sharp suture scissors or a #12 scalpel blade can be used to cut the sutures before they are removed with cotton pliers or tissue forceps. A transient bacteremia can be expected after suture removal, even when a preprocedural chlorhexidine mouth rinse is used.[79] Antibiotic coverage should be considered only for patients at high risk of developing bacterial endocarditis.

If healing is progressing normally at the suture removal appointment, the patient does not need to be seen again in the office until the first scheduled recall examination, typically 3 to 12 months after surgery. However, phone contact with the patient approximately 7 to 10 days after suture removal is recommended to confirm the absence of problems. Patients with questionable healing at the suture removal appointment should be reevaluated in the office in 7 to 10 days or sooner if necessary.

MANAGEMENT OF SURGICAL COMPLICATIONS

Although serious postoperative surgical complications are rare, the clinician should be prepared to respond to patient concerns and recognize when additional treatment may be necessary. Careful case evaluation, adherence to a minimally traumatic surgical technique, and proper patient management, as described previously in this chapter, should result in a low incidence of postoperative complications. Even so, some patients experience mild to moderate postoperative pain, swelling, ecchymosis, or infection. In a prospective study of 82 patients undergoing endodontic surgical treatment, Tsesis and coworkers[545] reported that 76.4% were pain free 1 day after surgery and 64.7% did not report any swelling. Only 4% of the patients in this study experienced moderate pain, and this sequela was closely related to the presence of presurgical symptoms. Postoperative pain typically peaks the day of surgery, and swelling reaches its maximum 1 to 2 days after surgery.[294] As previously noted, good evidence supports the use of prophylactic NSAID therapy and a long-acting local anesthetic to reduce the magnitude and duration of postoperative pain.

Patients should be advised that some postoperative oozing of blood is normal, but significant bleeding is uncommon and may require attention. Most bleeding can be controlled by applying steady pressure for 20 to 30 minutes, typically with a piece of moist cotton gauze or a tea bag. Bleeding that persists requires attention by the clinician. Pressure to the area and injection of a local anesthetic containing 1:50,000 epinephrine are reasonable first steps. If bleeding continues, it may be necessary to remove the sutures and search for a small severed blood vessel. When located, the blood vessel can be crushed or cauterized to control bleeding. Cauterization may be performed with a heat source commonly used for warm obturation techniques. Local hemostatic agents, as previously described, may also be used. Occasionally, a patient may require hospitalization and surgical intervention to control bleeding, but this is an extremely rare event. Extraoral ecchymosis (Fig. 9-65) occurs when blood seeps through the interstitial tissues; although it may be alarming to the patient and clinician, this condition is self-limiting and does not affect the prognosis.[281] Moist heat applied to the area may be helpful, although complete resolution of the discoloration may take up to 2 weeks. Heat should not be applied to the face during the first 24 hours after surgery.

Sinus exposure during surgical root canal procedures on maxillary posterior teeth is not uncommon. Postoperative antibiotics and decongestants are often recommended[16,29,281,568]; however, this practice is controversial, and no evidence supports the routine use of antibiotics and decongestants in these cases. Walton[576] makes a persuasive argument that antibiotics are not routinely indicated for the management of sinus exposures during periradicular surgery when primary closure of the oral-antral communication is possible. Further support for this position is provided by other clinicians who have observed excellent healing and minimal complications after sinus exposure during periradicular surgery.[291,456,579] Clinical judgment should guide the use of antibiotics and decongestants on a case-by-case basis until more conclusive evidence on this practice is available.

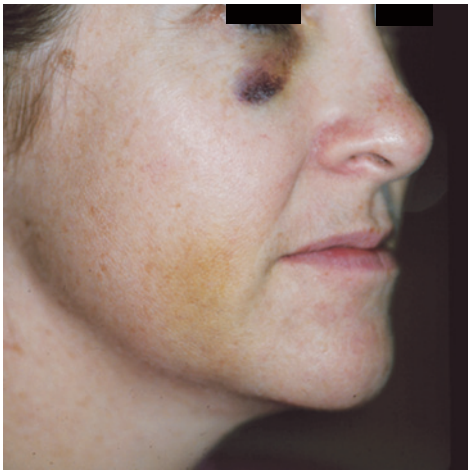

FIG. 9-65 Postoperative ecchymosis can be alarming to the patient but resolves spontaneously within 7 to 14 days.

No reliable data are available to provide an accurate estimate of the likelihood of paresthesia after surgical root canal treatment. The incidence of paresthesia after third molar removal is estimated to be 1% to 4.4%[443]; however, most reported cases of paresthesia after third molar extraction involved the lingual nerve, which is rarely encountered in mandibular periradicular surgery. The incidence of damage to the inferior alveolar nerve after third molar surgery is approximately 1.3%, with only about 25% of these cases resulting in permanent injury.[551] Unless the nerve is resected during surgery, most patients can be expected to return to normal sensation within 3 to 4 months. If the paresthesia does not show signs of resolving within 10 to 12 weeks, referral and evaluation for possible neuromicrosurgical repair should be considered.[167,431] Robinson and Williams[443] presented a useful method for charting and documenting paresthesias. The area of altered sensation is determined by pinching the skin or mucosa with cotton pliers; alternatively, a pinprick can be applied with a sharp instrument. The area of paresthesia is noted with a series of marks on a diagram of the face and mouth. This method provides a graphic and chronologic record of the paresthesia.

SUMMARY

Periradicular surgery today bears little resemblance to the surgical procedures commonly performed as recently as the 1990s. Enhanced magnification and illumination, microsurgical instruments, ultrasonics, new materials for hemostasis, root-end filling, and GTR, and a greater understanding of the biology of wound healing and the etiology of persistent periradicular disease all have contributed to the rapid evolution of periradicular surgery. With proper case selection and operator skill, periradicular surgery can be considered a predictable, cost-effective alternative to extraction and tooth replacement.

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CHAPTER 10

Regenerative Endodontics

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CHAPTER OUTLINE

Overview of Regenerative Dentistry
Overview of Regenerative Endodontics
Preclinical Studies on Regenerative Endodontics
Stem Cells
Growth Factors/Morphogens
Scaffolds
Delivery System
Translational Studies
Summary of Basic Research on Regenerative Endodontics
Clinical Studies on Regenerative Endodontics
Clinical Procedures Related to Regenerative Endodontics
Overview of Clinical Regenerative Endodontic Procedures (REPS)
Example of a Revascularization Protocol
Clinical Measures of Treatment Outcome
Summary

OVERVIEW OF REGENERATIVE DENTISTRY

Advancements in tissue engineering are dramatically changing medicine and dentistry. Tissue engineering is an interdisciplinary field that applies the principles of engineering and the life sciences to restoring, maintaining, or replacing biologic function.[127] It involves the interplay among stem cells, growth factors, and scaffolds (biologic matrices). It has become increasingly clear that the intentional manipulation of these three factors can lead to the regeneration of tissue function that would not otherwise occur if repair had taken place without intervention.[54] This relatively young field was first applied to medicine with many examples of regenerative medicine approaches used in the clinical practice.[125,188,240] Although the inclusion of tissue engineering in dentistry is more recent, it is also fundamentally changing the way clinicians are treating patients while providing a fertile research field that fosters future advancements and therapies.

Most of the history of dentistry is marked by the evolution of dental materials and techniques tailored to the replacement of lost or diseased tissues with inert materials. This prosthetic replacement of missing dental tissues has prevailed in dentistry since the primordial examples of dental treatments in ancient civilizations.[136,145,238] In contrast, the goal of regenerative dentistry is to induce biologic replacement of dental tissues and their supporting structures. The potential for regenerative dentistry is in large part due to advancements in biologic therapies that apply principles of tissue engineering with the spatial and temporal assembly of stem cells, growth factors, and scaffolds to achieve the functional regeneration of a missing tissue.

Pioneering work supporting the concept of regenerating dental tissues was reported in the 1960s when Dr. B.W. Hermann described the application of calcium hydroxide ($Ca[OH]_2$) for vital pulp therapy[219] and Professor Nygaard-Østby evaluated a revascularization method for reestablishing a pulp-dentin complex in permanent teeth with pulpal necrosis (discussed later).[172,173] The scope and clinical application of regenerative dental procedures have advanced to now include guided tissue or bone regeneration (GTR, GBR) procedures and distraction osteogenesis,[32,133,175] the application of platelet-rich plasma for bone augmentation,[85] Emdogain for regeneration of periodontal tissues and pulp,[7,45] recombinant human bone morphogenic protein (rhBMP) for augmentation of bone,[4,151] and clinical trials on the use of fibroblast growth factor 2 (FGF-2) for periodontal tissue regeneration.[253] The potential of regenerative procedures in endodontics has been emphasized by elegant studies demonstrating the regeneration of pulp, dentin, and enamel using scaffold materials and stem cells.[81,107,210,265] Thus, regenerative dental procedures are emerging as a vital, evolving field of dental care, creating a paradigm shift in many dental specialties, including endodontics.[152] This chapter reviews the current status of regenerative endodontic procedures with an emphasis on biologic principles and the advantages and limitations of currently available clinical procedures.

Overview of Regenerative Endodontics

The developing dentition is at risk for pulpal necrosis due to trauma, caries, and developmental dental anomalies such as dens evaginatus.[13,16,122,144,220,225,260] Loss of an immature permanent tooth in young patients with mixed dentition can be devastating, leading to loss of function, malocclusion, and inadequate maxillofacial development. These teeth were traditionally treated with apexification procedures using either long-term calcium hydroxide treatment[53,52] or immediate placement of a mineral trioxide aggregate (MTA) apical plug.[247] Although these treatments often resolve the signs and symptoms of pathosis, they provide little to no benefit for continued root development.[33] Thus, immature teeth treated with these procedures are considered in a state of arrested development,

and no further root growth, normal pulpal nociception, and immune defense should be expected.

Regenerative endodontic procedures (REPS) have been defined as biologically based procedures designed to replace damaged structures such as dentin, root structures, and cells of the pulp-dentin complex.[152] This new treatment modality has emerged as an alternative that, in addition to healing apical periodontitis, aims to promote normal pulpal physiologic functions. These include continued root development, immune competency, and normal nociception, as seen in some published cases.[62] Thus, the ultimate goal of these procedures is to regenerate the components and normal function of the pulp-dentin complex.

Regenerative endodontics is founded on the seminal work of Dr. Nygaard-Østby, completed in the 1960s. He hypothesized that a blood clot could be the first step in the healing of a damaged dental pulp, similar to the role of the blood clot in the healing process observed in other areas (e.g., alveolar bone following extraction).[171] To test the hypothesis that the presence of a blood clot within a root canal system promotes healing, mature teeth diagnosed with either vital or necrotic pulp received pulp space debridement followed by foraminal enlargement, medicament dressing for the necrotic cases, evoked intracanal bleeding, and a kloroperka obturation placed coronal to the formed blood clot. Patients (n = 17) were followed for various time periods (17 days to 3.5 years), and then the treated tooth was extracted and the newly formed tissues were histologically examined. The outcomes were similar for all teeth: (1) resolution of symptoms of inflammation related to foraminal enlargement and over-instrumentation in as early as 17 days; (2) resolution of signs and symptoms of pathosis for the necrotic cases; and, in certain cases, (3) radiographic evidence of apical closure. For the histologic analysis, it was observed that there was ingrowth of connective tissue into the canal space and varied levels of mineralized tissue found along the canal walls as well as "islands" of mineralized tissue embedded within the newly formed tissue (Fig. 10-1). Because dental pulp is a type of connective tissue with a rich supply of fibroblasts, this general finding was quite promising. However, the inclusion of undesired cell types (e.g., cementoblasts) and the lack of desired cell types (e.g., odontoblasts) indicate that this protocol did not lead to complete histologic regeneration of dental pulp. Despite its shortcomings, this pioneer study laid the foundation for the subsequent studies in the field of regenerative endodontics.

In 1966, a study was published reporting that disinfection could be established primarily with inter-appointment medication with polyantibiotic mixes (three different formulations used in five cases).[196] The investigators did not purposely evoke intracanal bleeding in this study, but instrumented canals short of what they thought to be vital tissue, determined by visualization of tissue and pain sensation upon instrumentation. Signs and symptoms of disease and continued root development were resolved for all reported cases. The study represented the first reported case in which polyantibiotic pastes were used in immature necrotic teeth for disinfection and to promote root development. Five years later, another study was published that included the use of antibiotics in the disinfection protocol and the intentional promotion of intracanal bleeding.[173] Resolution of symptoms and continued root development was a common finding. However, the histology of the extracted teeth demonstrated that connective tissue was formed in 28 out of 35 teeth, whereas cellular cementum was formed in 18 out of 35 teeth. Again, these protocols generated acceptable clinical outcomes (e.g., healing of apical periodontitis, lack of symptoms, etc.) with only partial evidence of dental pulp phenotype. Collectively, these findings laid the foundation for contemporary regenerative endodontics, demonstrating that repair could take place following root canal disinfection in immature teeth.

The first case report of a "contemporary" regenerative endodontic procedure occurred in 2001.[62] Since then, there has been an exponential increase in published cases reporting unprecedented clinical outcomes such as resolution of signs and symptoms of apical periodontitis, continued root development, and, in certain cases, normal nociceptive responses to vitality testing.[62] Despite the lack of randomized clinical trials, these published clinical observations support the hypothesis that patients with otherwise limited treatment options benefit from these procedures. Importantly, the field of regenerative endodontics has seen a dramatic increase in knowledge gained from translational basic science studies evaluating the interplay of the tissue engineering components (stem cells, growth factors, and scaffolds) applied to the clinical need and challenges.

PRECLINICAL STUDIES ON REGENERATIVE ENDODONTICS

Applying the principles of tissue engineering to the development of regenerative endodontic procedures requires research on the correct spatial assembly of distinct stem cells, growth factors/morphogens, and scaffolds to form a functional pulp-dentin complex.[95,107,127,161] In this section, we review each of these critical components in turn.

Stem Cells

Stem cells are defined as a distinct subpopulation of undifferentiated cells with self-renewal and differentiation potential. They can be classified as pluripotent or multipotent cells. Pluripotent stem cells have the capacity of becoming specialized cells and belong to all three germ layers. Embryonic stem cells are the best example of pluripotent cells. There is a significant body of research on embryonic stem cells, but ethical, legal, and medical (tissue-rejection) issues can render these cell types unsuitable for clinical applications.[152] True pluripotent stem cells can only be found in the developing embryo, and the harvesting of these cells requires destruction of the embryo, hence the legal and ethical concerns with such practice. Dr. Yamanka and colleagues reported the groundbreaking finding that somatic cells can be transformed into pluripotent stem cells—namely, induced pluripotent stem cells (iPSC).[177] The use of iPSCs does not have the same legal and ethical concerns as the use of embryonic stem cells, but iPSCs share the lack of control over their uninhibited proliferation and differentiation of embryonic stem cells. These cells tend to form teratomas after implanted into a host, a true testament of their high proliferative and differentiation capacities, but this makes them unsuitable for immediate common clinical practice.[177,221] On the other hand, all adult mesenchymal stem cells are more restricted in their capacity to differentiate, only forming tissues of mesenchymal origin, and therefore are classified as multipotent.[40] These cells can be found compartmentalized within tissues in "stem cell niches." The mesenchymal tissues (e.g.,

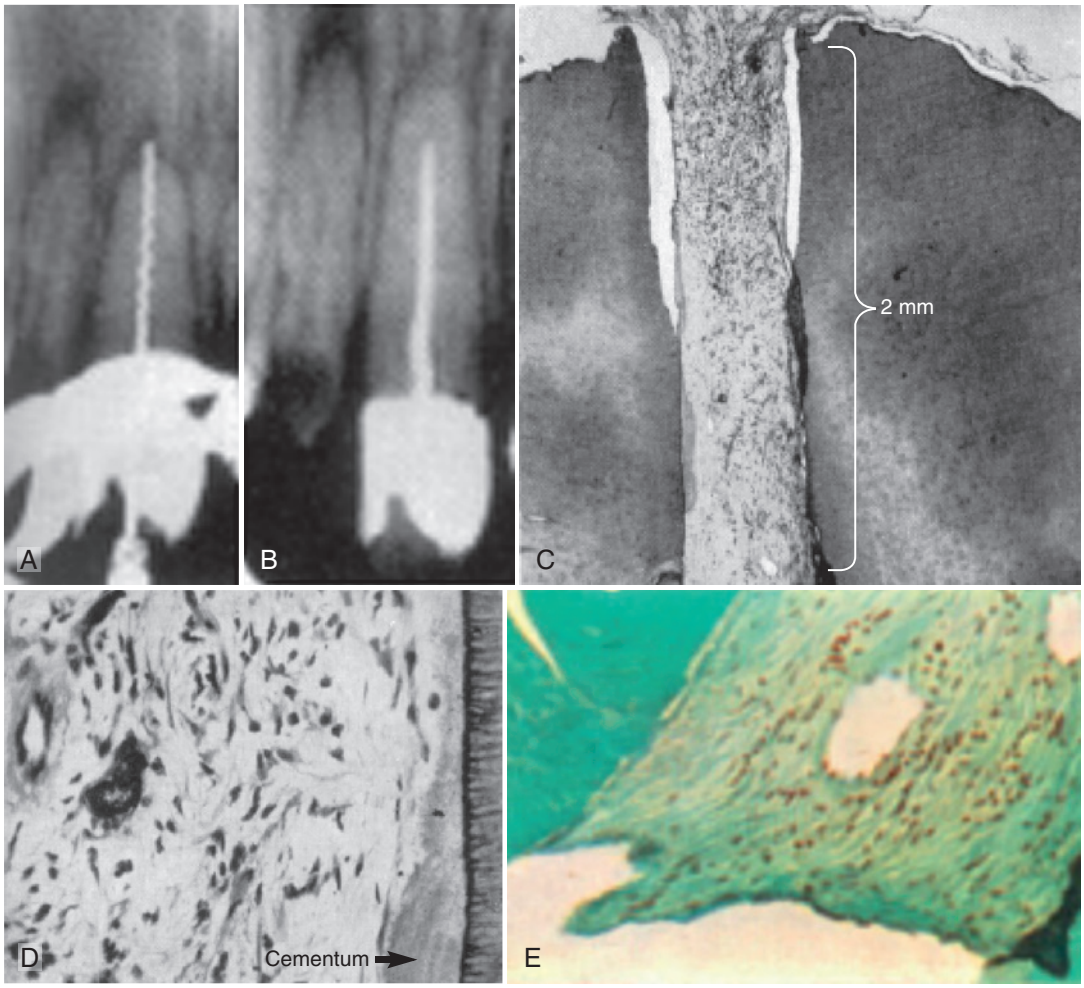

FIG. 10-1 Radiographic and histologic findings from a central incisor with a necrotic pulp, from Nygaard-Østby. **A,** Evidence of a file going beyond the apex. There is also evidence of a radiolucency in the apical area. **B,** Second radiograph at 14 months, taken shortly before tooth was extracted, showing the short fill. **C,** Histologic section of same tooth, showing fibrous connective tissue has grown into the apical 2 mm of the tooth. **D,** Higher magnification *(upper right)* shows evidence of what appears to be cementum deposition on the canal wall and fibrous connective tissue in the pulp space. **E,** Evidence of collagen bundles in the canal space. (From Nygaard-Østby B: The role of the blood clot in endodontic therapy: an experimental histologic study, *Acta Odontol Scand* 19:323, 1961.)

bone, dental pulp, periodontal ligament, etc.) appear to have an enriched population of adult stem cells.[40] These cells were first found in bone marrow decades ago and were characterized as self-renewing and plastic-adherent, and they formed cell colonies with a fibroblastic appearance.[79,80] They were initially called stromal stem cells but later received the now widely accepted name *mesenchymal stem cells (MSCs)*.[40] Most stem cells found in the orofacial region are MSCs.[66]

Different populations of adult stem cells have been identified in tissue compartments in the oral region. These include stem cells of the apical papilla (SCAP), inflammatory periapical progenitor cells (iPAPCs), dental follicle stem cells (DFSCs), dental pulp stem cells (DPSCs), periodontal ligament stem cells (PDLSCs), bone marrow stem cells (BMSCs), tooth germ progenitor cells (TGPCs), salivary gland stem cells (SGSCs), stem cells from human exfoliated deciduous teeth (SHED), oral epithelial stem cells (OESCs), gingival-derived mesenchymal stem cells (GMSCs), and periosteal derived stem cells (PSCs) (Fig. 10-2).[66,132] Although stem cells have been identified in most oral tissues, the stem cells more likely to be involved in REPS are localized around the periapical region. These include SCAP, PDLSCs, BMSCs, iPAPCs, and DPSCs (if vital pulp is still present apically).

The apical papilla and its residing stem cells (SCAP) were first characterized in 2006.[223] The apical papilla (Fig. 10-3) is a dense reservoir of undifferentiated MSCs with great proliferative and odontogenic differentiation capacity.[104,197] Importantly, SCAP are regulated by Hertwig's epithelial root sheath through a series of complex epithelial-mesenchymal interactions that dictate root development and shape.[249] Further, the close proximity of the apical papilla to the apices of teeth in continuum with the root canal space makes this rich source of stem cells readily available for regenerative endodontic therapeutics. The IPAPCs represent another important potential source of stem cells for regenerative endodontics in teeth with well-established apical periodontitis.[132,141] Lastly, stem cells of the periodontal

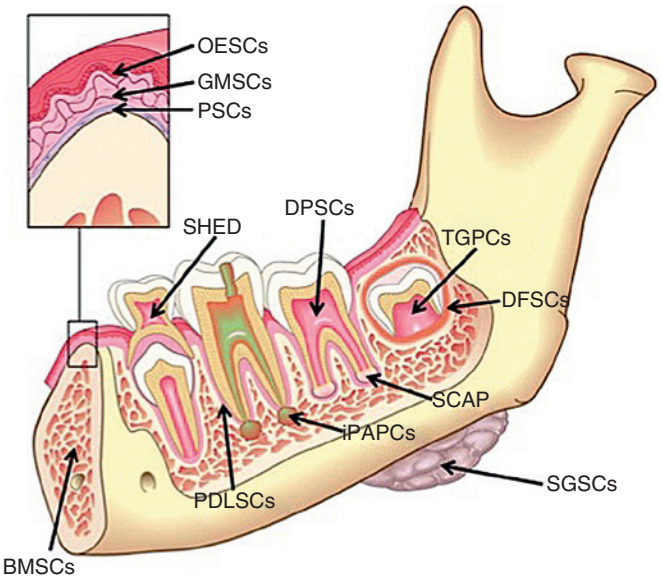

FIG. 10-2 Schematic drawing illustrating potential sources of postnatal stem cells in the oral environment. Cell types include tooth germ progenitor cells (TGPCS); dental follicle stem cells (DFSCS); salivary gland stem cells (SGSCS); stem cells of the apical papilla (SCAP); dental pulp stem cells (DPSCS); inflamed periapical progenitor cells (IPAPCS); stem cells from human exfoliated deciduous teeth (SHED); periodontal ligament stem cells (PDLSCS), bone marrow stem cells (BMSCS) and, as illustrated in the inset, oral epithelial stem cells (OESCS); gingival-derived mesenchymal stem cells (GMSCS); and periosteal stem cells (PSCS). (From Hargreaves KM, Diogenes A, Teixeira FB: Treatment options: biologic basis of regenerative endodontic procedures, *J Endod* 39:s30, 2013.)

ligament (PDL) and bone marrow should be also considered as stem cells sources for regenerative procedures because the action of mechanical disruption of the apical tissue (evoked bleeding) could also trigger the release of these cells, albeit their relative abundance is thought to be significantly less than SCAP and IPAPCs. In 2011, a study was conducted to evaluate the presence of mesenchymal stem cells following the evoked-bleeding step in regenerative procedures.[135] It was found that there is a substantial influx of mesenchymal stem cells into root canals during regenerative procedures resulting in an increase greater than 700-fold in the expression of MSC markers (Fig. 10-4). In addition, the cells could be harvested from clinical samples and examined under confocal microscopy (Fig. 10-5). This was the first demonstration that REPs are stem cell-based therapies.[62] Although this study did not evaluate whether the MSCs detected in REPs are derived from the apical papilla, it was assumed that these cells were SCAP because the evoked bleeding step lacerated the apical papilla. However, these MSCs are a heterogeneous population of cells that could come from any of the periradicular tissues after the mechanical step of evoking bleeding into the root canal system.

The delivery of substantial concentrations of MSCs into the root canal space, despite advanced apical periodontitis or abscess, points to an impressive survival capacity of these cells. In these clinical presentations, low oxygen tension, low pH, and a high concentration of endotoxins and inflammatory mediators are expected.[70,111,129,211] Indeed, the finding of high concentrations of the immune cell marker CD14 in those clinical samples indicates that there was still a substantial chronic inflammatory exudate present at the apical region of those teeth. These findings raise the question of how MSCs such as SCAP can survive during apical periodontitis where a complex microflora, an array of inflammatory mediators, immune cells, and presumably low oxygen tension are commonly encountered. The biologic reason for this apparent resilient survival,

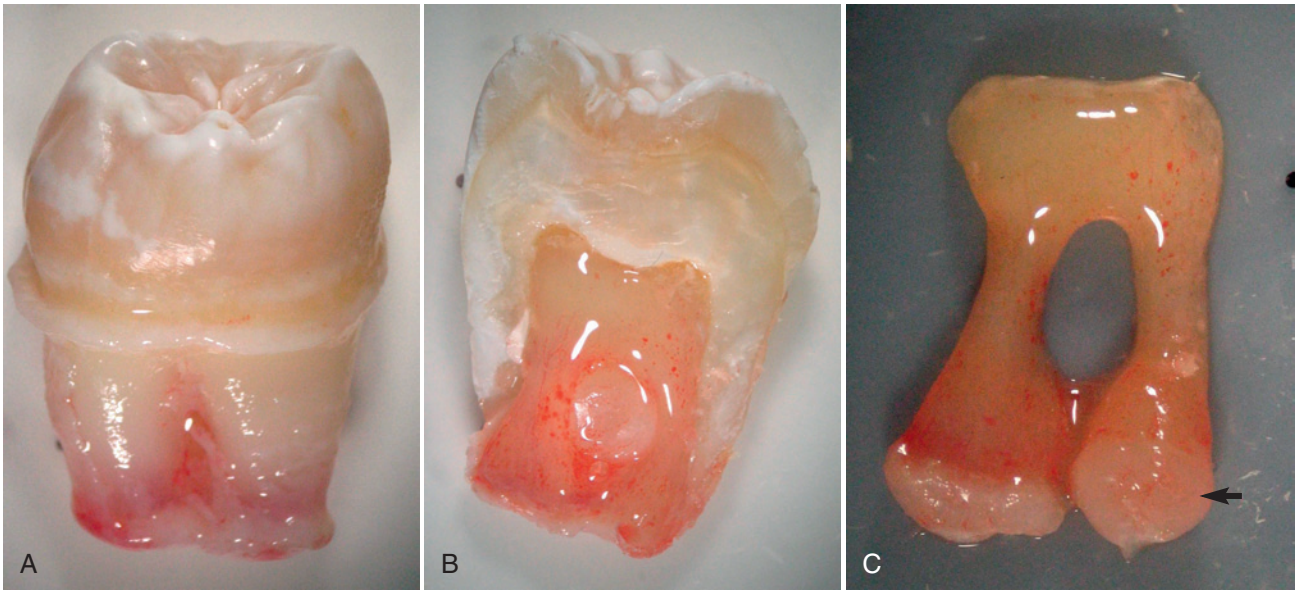

FIG. 10-3 **A-C,** Dissection of an immature permanent tooth indicating the extent of the apical papilla. Note that this structure is likely lacerated during the evoked bleeding step of revascularization cases and thus cells from this structure, including mesenchymal stem cells of the apical papilla (SCAP), are likely to be delivered into the root canal space. Arrow in **C** denotes junction of apical papilla and dental pulp. (Courtesy Dr. Michael Henry.)

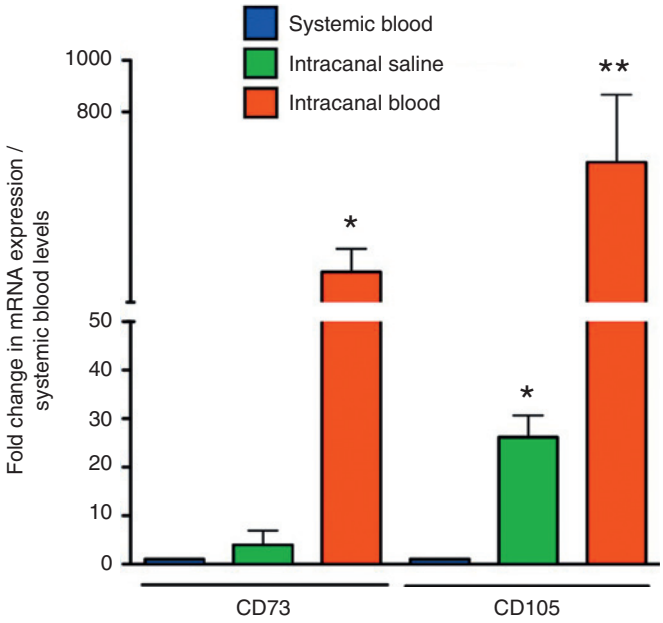

FIG. 10-4 Evoked-bleeding step in endodontic regenerative procedures in immature teeth with open apices leads to significant increase in expression of undifferentiated mesenchymal stem cell markers in the root canal space. Systemic blood, saline irrigation, and intracanal blood samples were collected during second visit of regenerative procedures. Real-time RT-PCR was performed by using RNA isolated from each sample as template, with validated specific primers for target genes and 18S ribosomal RNA endogenous control. Expression of mesenchymal stem cell markers CD73 and CD105 was upregulated after the evoked-bleeding step in regenerative procedures. Data were normalized to the housekeeping gene 18S levels and presented as mean ± standard deviation fold increase in relation to systemic blood levels for each gene and analyzed with one-way analysis of variance with Bonferroni post hoc test (n = 8; $*P < .05$; $**P < .01$; n.s., not statistically significant). (From Lovelace TW, Henry MA, Hargreaves KM, Diogenes A: Evaluation of the delivery of mesenchymal stem cells into the root canal space of necrotic immature teeth after clinical regenerative endodontic procedure, *J Endod* 37:133, 2011.)

despite challenging conditions, may be explained by the relatively low density of blood vessels in the apical papilla in comparison to the adjacent dental pulp, whereas the dental follicle surrounding the apical papilla is highly vascularized and may act as a capillary bed to supply nutrients to SCAP.[62] Indeed, the apical papilla was found to remain vital despite complete pulpal necrosis and advanced apical periodontitis in an animal model of endodontic infection.[169] Further, it has been demonstrated that hypoxic environments enhance the proliferation, survival, and angiogenic potential of dental stem cells.[5,19,57,106,201] Interestingly, similar enhancing effects were observed when dental stem cells were exposed to bacterial by-products such as endotoxin.[1] Thus, it appears that SCAP and surrounding stem cells are equipped to survive and maintain their potential for differentiation in adverse conditions such as apical periodontitis and apical abscesses. Nonetheless, stem cells delivered into root canals after bleeding is evoked from the periradicular tissues are likely from various apical sources or niches.

The dental pulp can be viewed as a core of innervated and vascularized loose connective tissue surrounded by a layer of odontoblasts. The major cell type of this core region is the fibroblast. Together with blood vessels, lymphatics, and neurons, this core tissue is embedded in an extracellular matrix consisting of collagen and other fiber types (see also Chapter 12). Dental pulp stem cells (DPSCs) can be found throughout the dental pulp but are known to accumulate in the perivascular region and the cell-rich zone of Hohl adjacent to the odontoblastic layer.[75,205] Thus, DPSCs from both sources are thought to be active participants in the process of reparative dentinogenesis.

Dental pulp stem cells are recruited to the site of injury following a gradient of chemotactic agents released by resident immune cells and from the damaged dentin.[2,227] The reparative dentin formed by these cells is distinct from the primary, secondary, and reactionary dentin that has been lost.[8,159,259] It is often called "osteodentin" when found to be disorganized, atubular, and having cellular inclusions. This process of cellular repair is enhanced by bioactive materials (e.g., MTA and Biodentine). These materials increase the inherent mineralizing potential of the dental pulp when used in both indirect and direct pulp applications.[187] However, the process of tertiary dentinogenesis requires a vital pulp and the resolution of the etiology (e.g., caries or trauma). This process becomes disrupted when the pulp succumbs to injury, resulting in liquefaction necrosis of the dental pulp. Regeneration in this case is only possible with the recruitment or delivery of autologous stem cells to the canal space following adequate disinfection.[62]

Odontoblasts are one of the most specialized cells of the pulp dentin complex with dentinogenic, immunogenic, and possibly sensorial functions.[34,67,226] Odontoblasts in the intact pulp-dentin complex are easily identified based on their location and distinct morphologic characteristics (i.e., columnar polarized cell body with cellular projections into the dentinal tubules). However, it is far more challenging to identify and characterize an odontoblast-like cell, mainly because these cells lack a primary odontoblast morphology and unique markers that could be used for identification.[102] Indeed, many markers used for the identification of odontoblast-like cells are also expressed in other mineralizing cell types such as osteoblasts. For example, both odontoblasts-like cells and osteoblasts are similar in the formation of mineralized nodules and in the expression of several proteins such as dentin sialoprotein (DSP), although DSP levels are nearly 400 times greater in odontoblasts than in osteoblasts.[244] Measuring only one or two characteristics of a cell might not conclusively identify whether the cell is a true odontoblast. Even among odontoblasts, the phenotype varies in cells located in the apical (squamous shape) versus coronal (tall columnar) pulpal tissue. Importantly, molecular studies have identified many of the genes selectively expressed in odontoblasts.[134,178-180] Lastly, an intermediate filament protein called nestin has been shown to be preferentially expressed in odontoblasts or odontoblast-like cells when in the active secretory function. Nestin expression could be used in conjunction with other markers to better identify odontoblast-like cells.[3,4] This knowledge is expected to aid future studies characterizing the conditions necessary for mesenchymal cells of multiple origins to differentiate into odontoblast-like cells. It is likely that definitive cellular identification depends on both the morphology of the cell and an assessment of the expression of multiple genes.

At least five different types of postnatal mesenchymal stem cells, in addition to DPSCs, have been reported to differentiate

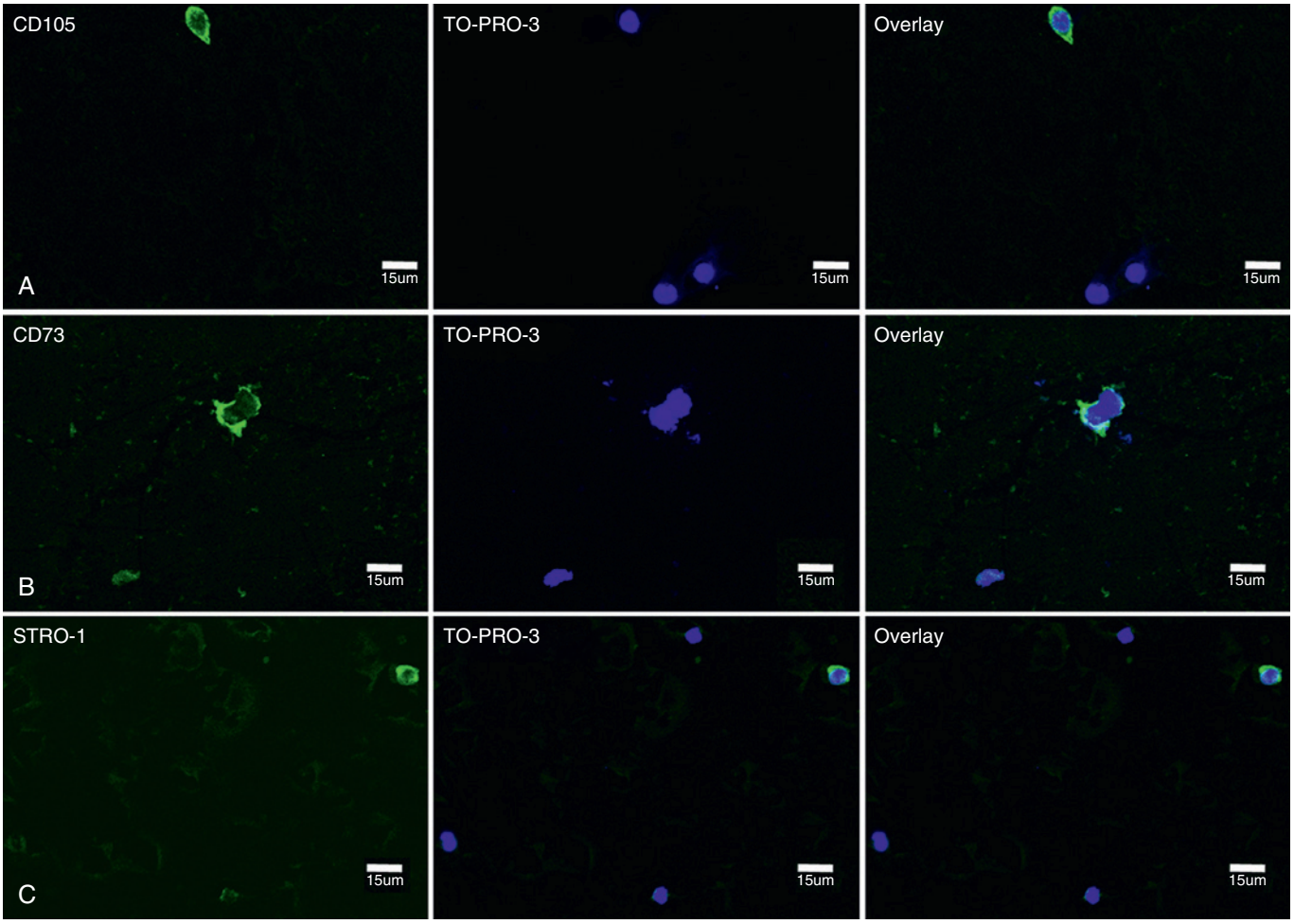

FIG. 10-5 Mesenchymal stem cells were delivered into root canal spaces during regenerative procedures in immature teeth with open apices. Cells collected from intracanal blood samples after the evoked-bleeding step or from systemic blood were stained with antibodies against CD105, CD73, or Stro-1 and evaluated with a laser-scanning confocal microscope. Cells in intracanal blood samples collected after the evoked-bleeding step showed expression of mesenchymal stem cell marker (**A**) and CD105 (green in **A**), CD73 (green in **B**), and STRO-1 (green in **C**), whereas nuclei appear blue as stained with TO-PRO-3. (From Lovelace TW, Henry MA, Hargreaves KM, Diogenes A: Evaluation of the delivery of mesenchymal stem cells into the root canal space of necrotic immature teeth after clinical regenerative endodontic procedure, *J Endod* 37:133, 2011.)

into odontoblast-like cells, including SHED,[147] SCAP,[124,125] IPAPCs,[132] DFPC,[149] and BMMSC.[22] One study demonstrated that over-instrumentation into the periradicular tissues followed by bleeding into the canal space results in a robust influx of cells with mesenchymal stem cell markers in fully mature teeth, similar to that seen in immature teeth.[135] Thus, it appears that MSCs from the apical region can be delivered into the root canal spaces in both immature and mature teeth. However, there is growing evidence that MSCs have decreased proliferative and differentiation potential with aging.[117,132,162] Further research is required to elucidate the age limit for the use of autologous dental-derived stem cells, but these findings suggest that regenerative procedures may be applicable to mature fully formed teeth in adults. In fact, in a proof-of-concept case report, resolution of apical periodontitis followed by narrowing of the canal space and apical closure was seen in two fully mature teeth in adult patients treated with regenerative endodontic procedures.[182] Thus, more research and development is required to make regenerative procedures more predictable for immature teeth, and these procedures may transition to be applicable to fully formed teeth.

Growth Factors/Morphogens

Dentine is composed of collagen fibers (90%, collagen type I) and noncollagenous matrix molecules (proteoglycans, phosphoproteins, and phospholipids). The collagen fibers act as a grid or matrix, and this structure behaves as a scaffold upon which mineralization can occur. Dentine phosphoprotein (DPP) and dentine sialoprotein (DSP) are the most abundant dentine-specific proteins among the noncollagenous proteins of organic matrix.[38] DSP resembles other sialoproteins such as bone sialoprotein, but its precise function is still unclear; it may have a role in matrix mineralization.[92] Both DSP and DPP make up part of the small integrin-binding, ligand N-linked glycoproteins (SIBLINGS), which include dentine matrix acidic phosphoprotein 1 (DMP-1), bone sialoprotein,

TABLE 10-1

List of Growth Factors Found into the Mineralized Dentin Matrix

Growth Factors in Dentin Matrix	
Transforming growth factor beta-1 (TGFβ-1)	Cassidy et al., 1997[42]
Transforming growth factor beta-2 (TGFβ-2)	Cassidy et al., 1997[42]
Transforming growth factor beta-2 (TGFβ-3)	Cassidy et al., 1997[42]
Bone morphogenic protein –2 (BMP-2)	Thomadakis et al., 1999[230]
Bone morphogenic protein –4 (BMP-4)	About et al., 2000[4]
Bone morphogenic protein –7 (BMP-7)	Thomadakis et al., 1999[230]
Insulin growth factor-1 (IGF-I)	Finkleman et al., 1990[74]
Insulin growth factor-2 (IGF-II)	Finkleman et al., 1990[74]
Hepatocyte growth factor (HGF)	Tomson et al., 2013[232]
Vascular endothelial growth factor (VEGF)	Roberts-Clark and Smith, 2000[194]
Adrenomedullin (ADM)	Musson et al., 2010[155]

osteopontin, osteocalcin, and osteonectin. These proteins are only a small part of the whole cocktail of noncollagenous proteins that form components of the dentine.[87]

Research on dentine structure and composition has highlighted that the matrix contains some components that may be important in regulating tissue due to their bioactive properties. For this reason, dentine is today considered a reservoir of growth factors and cytokines.[212] These growth factors/cytokines are secreted by the odontoblasts during primary dentinogenesis, becoming sequestered and "fossilized" into the dentine after biomineralization (Table 10-1). However, they may become solubilized by demineralization of the matrix, bacterial acid (caries decay), chemical treatment (EDTA rinsing solution, calcium hydroxide or acid etching for bonded restorations), or restorative materials such as mineral trioxide aggregate and Biodentine.[213,231]

These growth factors and their receptors have been shown to be present at the enamel organ-dental papilla interface by immunohistochemistry and in situ hybridization during tooth development and have been implicated in odontoblast differentiation:

- Growth hormone (GH) plays a paracrine or autocrine role in dental development.[263]
- IGF-1 and -2 (of the family of IGF: insulin-like growth factor).[25,42,112]
- TGFβ-1, -2, and -3[56,228] and BMP-2, -4, and -6[239] play a role in the polarization and the differentiation of odontoblasts.[25] Notably in adult pulp, TGFβ-1 plays an important role in the regulation of the inflammatory response and tissue regenerative processes.[131]

The dental pulp has well recognized regenerative potential observed in the process of reparative dentinogenesis.[153,212] In this process, dentin-derived growth factors are thought to play a key role to be deciphered into the regulation of progenitor cell recruitment, cell proliferation, and differentiation of new dentine-secreting cells.[153,212] Indeed, the differentiation of new odontoblast-like cells has also been reported following pulp capping with basic fibroblast growth factor (FGF), TGF-β1,[131] and BMP-7.[86]

The sequestration of these growth factors in the dentine matrix and their subsequent "fossilization" during the mineralization process appears key to the pulp healing process where their release from the matrix may be responsible for various signaling events. These growth factors are extremely potent and have a variety of cell signaling properties. However, their precise localization in the dentine[214] and their various biologic roles remain to be elucidated.

It is possible to imagine opportunities for therapeutic stimulation, inducing a targeted release of these proteins. For example, treatment of dentin with EDTA solution has been shown to dissolve the mineral phase, liberating growth factors that orchestrate the stimulation of progenitors or stem cell differentiation.[216-218] Etching with orthophosphoric acid, used for conditioning the dentine in bonding procedures, also promotes demineralization of the dentine and liberation of biologic factors.[64,72] For a long time, calcium hydroxide has been used as a protective lining, especially beneath amalgams fillings, or as a canal disinfection medication. It has been shown to have the ability to release bioactive components from the dentine, including growth factors.[91] Unlike dentin-etching acids, which only have brief contact with the dentine, calcium hydroxide remains in place beneath restorations or in canals allowing for a gentle and continued dissolution, thus releasing growth factors; its action is prolonged and potentially controllable depending on the form of the product. Lastly, calcium hydroxide, a by-product of the use of MTA and Biodentine, appears to underlie release of bioactive dentin-derived growth factors by these two bioactive materials.[231] Thus, clinicians may take advantage of potent growth factors stored within dentin with the use of chemical treatments and materials that promote the release of these factors.

Morphogens

It is also important to clearly keep in mind that a second level of regulation exists during dental development (and thus during pulp regeneration process)—transcription factors. Notably, Msx1 is expressed in polarized preodontoblasts, whereas Msx2 is present in mature odontoblasts.[25] Protein and transcripts for Msx1 have been identified in the pulp mesenchyme at early stages of tooth development and their concentrations decreased at the bell stage.[49] The expression of these transcription factors is under the control of growth factors, and they can ultimately have broad-ranging effects. Significantly, BMP4 upregulates Msx1 and Msx2 expression. In turn, transcription factors regulate further growth factor expression; for example, Msx1 upregulates BMP4 synthesis in the mesenchyme, and Msx2 regulates Runx2 and osteocalcin gene expression during odontogenesis.[30,31]

Growth factors and transcription factors are central to the cascade of molecular and cellular events during tooth development and are responsible for many of the temporospatial morphologic changes observed in the developing tooth germ. For these reasons, they are also likely involved in the regeneration process.

It is also important to consider the nature of the signaling process between the injurious agent and the pulp cells. Bacteria and their toxins are key candidates in the direct stimulation of pulp cells.[65] Lipopolysaccharides (LPS) and other bacterial toxins initiate intrapulpal inflammatory processes by activation of Toll-like receptors (e.g., TLR4 activation by LPS).[65,226,242] Importantly, both progenitor and dental stem cells have been shown to express these receptors.[34,35] Thus, stem cells within the dental pulp or periradicular tissues are equipped to detect microorganisms. Exposure of these cells to microbial antigens has been shown to directly modulate the proliferation and differentiation potential of these cells.[34,46,139,213,237] Lastly, cytokines commonly found in the inflammatory milieu (including that of the dental pulp) have a profound effect on stem cells. For example, it has been shown that TNF-alpha stimulates differentiation of dental pulp cells toward an odontoblastic phenotype via MAP kinase pathway activation and p38 phosphorylation.[183,209] Therefore, stem cell fate within the dental pulp is ultimately dictated by a complex cascade of intracellular signaling pathways activated by agents released from microorganisms, dentin, and immune cells.

Interestingly, morphogens are not only naturally occurring factors found within teeth. Several growth factors have also been evaluated for their ability to trigger the differentiation of selected mesenchymal stem cell populations into odontoblast-like cells (Table 10-2). Interestingly, several case studies have reported that patients taking long-term corticosteroids often present with dramatic reduction of the radiographic size of the pulp chamber and up to a fivefold increase in the thickness of the predentin layer.[164,165,248] Although these were medically complex patients (e.g., those experiencing renal failure) taking multiple drugs, the use of corticosteroids appeared to be associated with the observed increased activity of human odontoblasts. Further, these unexpected "side-effects" were also observed in a retrospective study evaluating the association of pulp calcifications and the long-term use of statins.[186] These incidental effects of commonly prescribed medications were further evaluated in translational studies that have extended this general observation by demonstrating that the application of dexamethasone or statins greatly increased the differentiation of human dental pulp cells into odontoblast-like cells.[102,176] This was particularly evident when dexamethasone was combined with 1,25-dihydroxyvitamin D_3.[102] Merely changing the composition of growth factors completely altered the differentiation of these cells, with the same population of cells able to express markers of odontoblasts, chondrocytes, or adipocytes, depending on their exposure to different combinations of growth factors.[244] Such findings emphasize the importance of growth factors in guiding the differentiation of these cells. Other studies have evaluated growth factors administered alone or in various combinations for promoting differentiation of odontoblast-like cells.

TABLE 10-2

Effects of Selected Growth Factors on the Differentiation of Odontoblast-Like Cells

Growth Factors	Cell Source	Phenotype	Condition	Authors
Dexamethasone	Human dental pulp	Odontoblast-like	In vitro × 8 weeks	Huang et al., 2006[102]
Dexamethasone and Vitamin D_3	Human dental pulp	Odontoblast-like	In vitro × 8 weeks	Huang et al., 2006[102]
Dexamethasone and Ascorbate-2-phosphate and β-Glycerophosphate	Human or rat dental pulp	Odontoblast-like	In vitro × 3 weeks	Wei et al., 2007[244] Zhang et al., 2005[266]
Insulin and Indomethacin and 3-Isobatyl-1-methylxanthine (IMBX)	Human dental pulp	Adipocyte	In vitro × 19 days	Wei et al., 2007[244]
Dexamethasone and Insulin and Ascorbate-2-phosphate and Sodium pyruvate and TGF-β1	Human dental pulp	Chondrocyte	In vitro × 8 weeks	Wei et al., 2007[130]
Growth/differentiation factor 11 (Gdf11)	Dental pulp	Odontoblast-like	In vitro/in vivo 10 days	Nakashima et al., 2004[75]
Simvastatin (statins)	Human dental pulp	Odontoblast-like	In vitro/in vivo	Okamoto et al., 2009[88]
LIM mineralization protein 1 (LMP-1)	Human dental pulp	Odontoblast-like	In vitro/in vivo	Wang et al., 2007[129]
Bone morphogenetic proteins	Dental pulp	Odontoblast-like	In vitro	Saito et al., 2004[99] Sloan et al., 2000[107] Chen et al., 2008[19]
TGF-β1-3	Rat/monkey dental pulp	Odontoblast-like	In vitro	Sloan et al., 1999[109]
Demineralized dentin	Human or rodent pulp	Odontoblast-like	In vitro/in vivo	Smith et al., 1990[111] Smith et al., 2001[110] Tziafas, 2004[123]
Nerve growth factor (NGF)	Immortalized apical papilla	Odontoblast-like	In vitro	Arany et al., 2009[11]
Fibroblast growth factor 2	Human dental pulp	Odontoblast-like	In vitro	He et al., 2008[43]
Dentin matrix protein 1	Rat dental pulp	Odontoblast-like	In vivo	Almushayt et al., 2006[1]

Several of the approaches using compounds later found to have growth factor–like effects have immediate clinical implications. First, it is unlikely that a single growth factor will result in maximal differentiation, so combinations of growth factors may be required for evaluation in clinical trials. Related to this point, many of the studied growth factors (e.g., dexamethasone, insulin) are drugs already approved for human use in other medical/dental applications. Second, the demonstration that statins promote the differentiation of an odontoblast-like phenotype suggests that patients clinically taking statins may also have narrowing of the pulp chamber space, similar to the findings previously described for corticosteroids. This would be an important future area of research. Third, clinicians have long used demineralized human bone to augment healing after surgical procedures.[190] Demineralized human bone is thought to contain a natural combination of appropriate growth factors and scaffolds, thereby providing an appropriate environment for osteoblast differentiation or function. Extending this concept, several research groups have demonstrated that demineralized human dentin has significant benefit for promoting the differentiation of odontoblast-like cells. Importantly, translational studies in regenerative endodontics have demonstrated that irrigation of dentin with 17% EDTA increases the survival of stem cells[105,142] and odontoblastic differentiation,[81,142] possibly due to the release of bioactive molecules from dentin.[215] Collectively these findings suggest that EDTA irrigation of the dentinal walls as part of an REP could improve clinical outcomes.

Scaffolds

An important component of tissue engineering is a physical scaffold.[160,245] Tissues are organized as three-dimensional structures, and appropriate scaffolding is necessary to (1) provide a spatially correct position of cell location and (2) regulate differentiation, proliferation, or metabolism while promoting nutrient and gaseous exchanges. Extracellular matrix molecules are known to control the differentiation of stem cells,[189,254] and an appropriate scaffold might selectively bind and localize cells, contain growth factors,[251,252] and undergo biodegradation over time.[261] Thus, a scaffold is far more than a simple lattice to contain cells, but instead can be viewed as the blueprint of the engineered tissue.

Scaffolds can be classified as either natural or synthetic. Examples of natural scaffolds include collagen,[103,159] glycosaminoglycans, hyaluronic acid (HA), demineralized or native dentin matrix,[24,93,158,236,255] and fibrin.[82] On the other hand, examples of synthetic scaffolds include poly-L-lactic acid (PLLA),[60] polyglycolic acid (PGA), polylactic-coglycolic acid (PLGA),[68] polyepsilon caprolactone,[256] hydroxyapatite/tricalcium phosphate,[11] bioceramics, and hydrogels such as self-assembly peptide hydrogels.[71,140] The great majority of currently published regenerative endodontic procedures involve evoked bleeding and the formation of a blood clot to serve as a scaffold.[62] Although it is relatively straightforward as it does not require ex vivo manipulation, this simplistic approach is not without challenges. The blood clot is often difficult to achieve, and it does not have many of the properties of the ideal scaffold. These properties include easy delivery, adequate mechanical properties, controllable biodegradation, and incorporation of growth factors.[82] In addition, the blood clot contains a great number of hematopoietic cells that eventually undergo cell death, releasing their toxic intracellular enzymes into the microenvironment, which may be detrimental to stem cell survival.

Another approach for creating a scaffold involves the use of autologous platelet-rich plasma (PRP). It requires minimal ex vivo manipulation, being fairly easy to prepare in a dental setting. PRP is rich in growth factors, degrades over time, and forms a three-dimensional fibrin matrix.[17,18,109,174] Platelet rich fibrin (PRF) is an alternative to PRP, as it has a three-dimensional architecture conducive with stem cell proliferation and differentiation and contains bioactive molecules.[58,61] These autologous scaffolds have been used successfully in regenerative cases.[21,222,264,267] However, it should be emphasized that, despite their reported use, there are some drawbacks to their clinical use: the process requires collection of intravenous blood that can be challenging in children, the diversity and concentration of growth factors within PRP and PRF preparations are not controllable,[20,116,241] and they lack temporal degradation control and the mechanical strength to support a coronal restoration. Thus, despite some desirable characteristics, other scaffold alternatives to PRP and PRF should be carefully considered.

Hydrogels are a class of scaffolds composed of three-dimensional hydrophilic polymers that absorb water or tissue fluids up to several times their weight.[73,250] These water-swollen materials are easily injectable in their colloidal form, undergoing gelation by chemical (e.g., changes in pH and osmolarity) or physical (e.g., temperature change) cues. These materials are highly tunable, biocompatible, and can be designed to resemble naturally occurring extracellular matrices.[250] They are of particular interest for regenerative endodontics because they can be easily injected into narrow root canal spaces and can be modified to deliver chemotactic and angiogenic agents to drive stem cell homing and supportive angiogenesis.[62,89] Hydrogels made of self-assembly peptides (e.g., Puramatrix)[43] show great potential to be used in endodontic tissue engineering because their sequence includes short peptide sequences, similar to those naturally occurring in tissues, enhancing cell attachment and proliferation.[84]

Delivery System

Even with selection of the appropriate cell source, growth factors, and scaffold, the resultant mixture must be delivered in a spatially appropriate fashion into the space of the root canal system. For example, nearly all cells of the body are within 0.1 to 1 mm of a blood vessel in order to maintain adequate diffusion of oxygen and nutrients.[90,98] This represents a challenge still to be overcome in the currently performed regenerative endodontic procedures that recruit stem cells[135] to a canal space devoid of lateral vascularity and several millimeters away from apical blood vessels. If one were to inject cells, in a cell-based approach, along the entire coronal-apical extent of a root canal system, the majority of cells would be expected to succumb to tissue hypoxia. Interestingly, it has been demonstrated that under hypoxic conditions, stem cells proliferate faster and release greater levels of angiogenic factors such as vascular endothelial growth factor 1 (VEGF) that promote targeted angiogenesis into the engineered space.[19] Thus, an alternative approach would be to inject a scaffold with chemotactic factors into the root canal. This approach is called cell homing, as cells are attracted to the scaffold along with supportive blood vessels in a progressive manner[121]; instead of being abruptly delivered to an avascular space (i.e., similar to

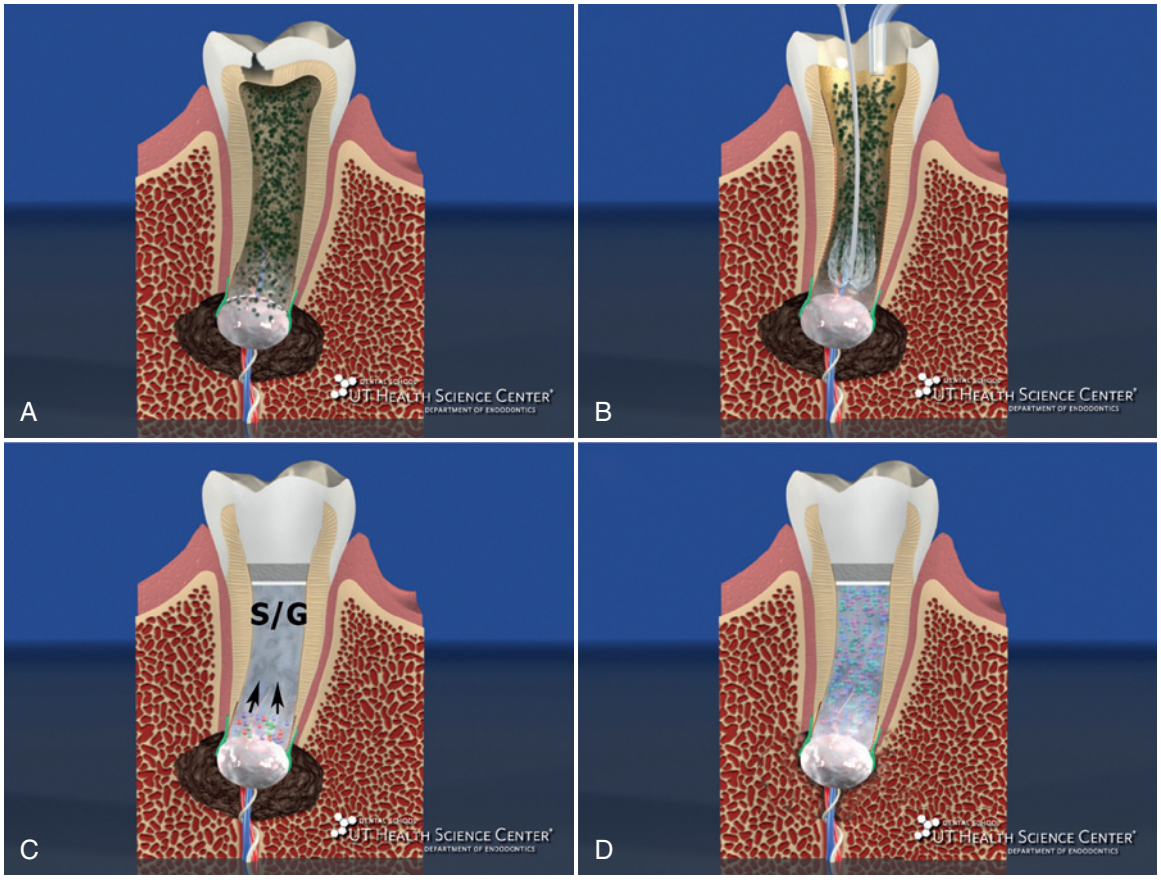

FIG. 10-6 Schematic drawing illustrating bioengineering using a cell homing approach. An immature premolar with a necrotic pulp and apical lesion (panel A) is disinfected (panel B), followed by placement of a biodegradable scaffold (s) containing growth factors and chemotactic factors (g) to allow progressive proliferation and migration of apical stem cells into the canal space (panel C) leading to the population of the canal space with stem cells concomitantly with vascular supply and tissue organization (panel D). (From Diogenes A, Henry MA, Teixeira FB, Hargreaves KM: An update on clinical regenerative endodontics, *Endod Topics* 28:2, 2013.)

the current revascularization procedures), the cell-homing approach can be applied in a cell-free[83] (no cells implanted along the chemotactic factors, see Fig. 10-6) or cell-based approach (cells are delivered in the chemotactic-containing scaffold).[152] Because dental pulp can be approximated as a loose connective tissue core surrounded by a layer of odontoblasts, the spatial arrangement of cells and growth factors within the scaffold may be particularly important to promote odontogenesis without having complete calcification of the root canal system. Complete recapitulation of the pulp-dentin complex architecture requires additional research effort.

Translational Studies

Several elegant studies in regenerative endodontics have used various translational methodologies including evaluation of clinical samples,[135] organotypic root canal models,[142,235] tooth slice models,[60,88,102] whole tooth culture, and animal models.[81,119,121,162,229,243] These studies have been crucial to provide a strong scientific foundation for the field of regenerative endodontics while allowing for clinical treatment protocol optimization and the development of new treatment strategies such as inclusion of scaffold and growth factors in regenerative procedures.[71,157]

One study demonstrated that new dentin and pulplike tissue could be generated in human root segments implanted subcutaneously in immune-compromised mice.[130] In this study, root segments had one of the openings sealed with MTA to mimic the coronal restoration of regenerative endodontic cases. The canal space was filled with either SCAP or DPSCs in a PLGA-based scaffold. The implants were harvested 3 months later and processed for immunohistochemical analysis. The results indicated that there was a dramatic circumferential apposition of dentin-like material along the dentinal walls. The new mineralized tissue was lined with polarized cells expressing odontoblastic markers. In addition, the dentin-like tissue was largely atubular and displayed cellular inclusions similar to the histologic presentation of osteodentin. Importantly, the cells from the engineered pulp were positive for human mitochondria, demonstrating that they originated from the implanted human stem cells and not from the host (mouse). Lastly, the root segments that were implanted without stem cells had only connective tissue that did not resemble a pulplike tissue, nor did it have mineralized tissues and odontoblast-like cells. Thus, a pulp-dentin complex could be engineered in human roots implanted subcutaneously in immune-deficient mice.

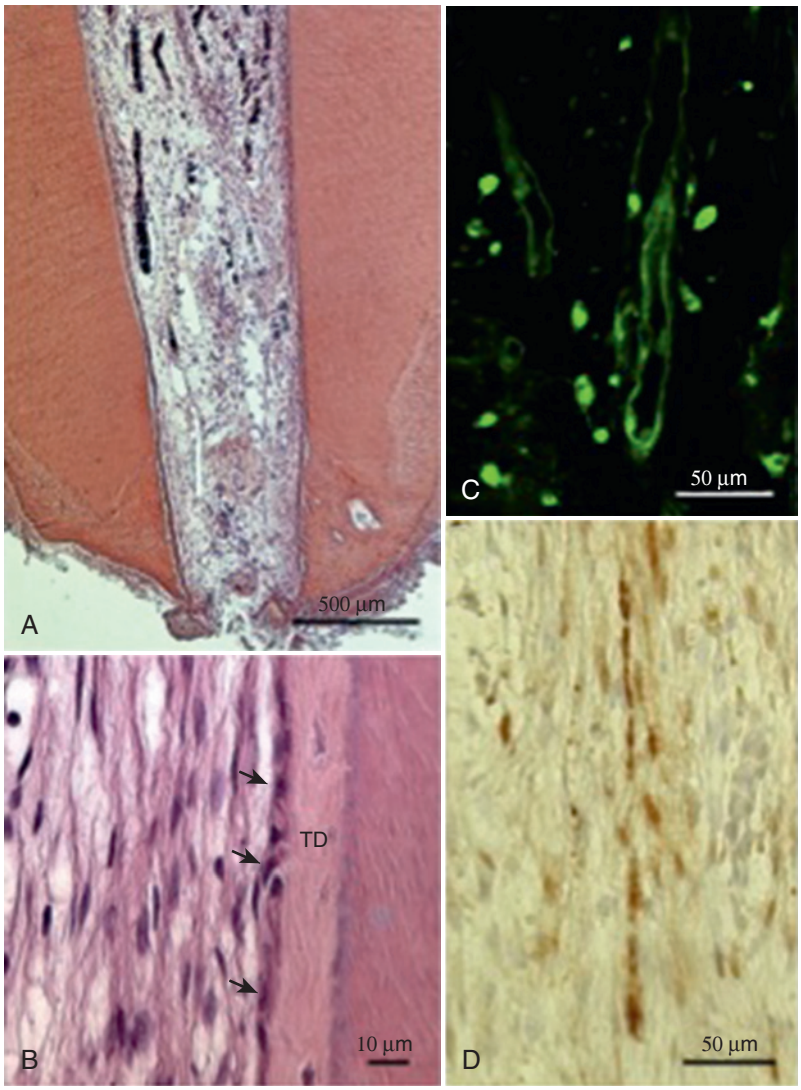

FIG. 10-7 Regeneration of pulp tissue after autologous transplantation of mobilized dental pulp stem cells (MDPSCs) with G-CSF in pulpectomized teeth of young dogs on day 14. **A-B,** Regenerated pulp tissue. **B,** Odontoblastic cells *(black arrows)* lining to newly formed osteodentin/tubular dentin (TD) along with the dentin. **C,** Immunohistochemical staining of BS-1 lectin. **D,** Immunohistochemical staining of PGP9.5. (From Nakashima M, Iohara K: Mobilized dental pulp stem cells for pulp regeneration, *J Endod* 40:S29, 2014.)

Nakashima and colleagues accomplished complete pulpal regeneration in dogs.[107] In this elegant study, dental pulp was removed via a sterile pulpectomy procedure, followed by placement of sorted CD105+ DPSCs in a collagen gel to the midroot. The remaining coronal part of the canal was back-filled with the collagen gel containing the chemotactic factor stromal derived factor 1 (SDF-1). Subsequent histology demonstrated the formation of new pulp tissue with innervation, vascularization, and odontoblast-like cells lining the dentinal walls. In addition, the engineered pulp had protein and RNA expression similar to the native dental pulp.[107]

In another important study, mobilized DPSCs were generated by selecting DPSCs that migrated toward a concentration gradient of granulocyte-colony stimulating factor (G-CSF).[108,150,163] These selected cells were implanted in a collagen gel into pulpectomized root canals.[108] Complete pulpal regeneration was observed, with evidence of new dentin formation, blood vessels, and innervation in the engineered tissue (Fig. 10-7). The impressive results of this cell-based approach have laid the foundation for the use of this technology in emerging clinical trials.[163]

It is noteworthy that all of the demonstrations of pulp regeneration in animal models were in root canals without any history of infection and pulpal necrosis.[107,108,130,159,200] Previously infected root canals must be adequately disinfected in order to suppress chronic inflammation that is detrimental to regeneration.[48] However, many irrigants and medicaments have detrimental effects on stem cell survival and differentiation.[63] Several studies using organotypic root canal models and animal models evaluated the combination and concentrations of irrigants and medicaments that allowed for stem cell proliferation and differentiation.[81,142,235] Thus, adequate disinfection and resolution of inflammation appears to be a limiting factor in complete pulpal regeneration, despite exciting results from current advances in dental pulp tissue engineering.

Summary of Basic Research on Regenerative Endodontics

Regeneration of a functional pulp-dentin complex relies on the foundation of tissue engineering and can be viewed as a function of the spatially correct delivery of appropriate stem cells and growth factors embedded within a scaffold. Although considerable research has used in vitro cell-culture methods to identify key factors regulating the differentiation of odontoblast-like cells, emerging studies conducted in animal models are promising for regeneration of this pulp tissue. Preclinical studies involving surgical placement of a human tooth filled with a human stem cell/growth factor/scaffold combination into immunocompromised mice[119,130] have permitted histologic analysis of neovascularization as well as the differentiation and mineralization activity of newly formed odontoblasts. The use of human cells in a mouse model permits histologic confirmation that the resulting odontoblast-like cells were of human origin. These novel findings provide strong evidence that either human SCAP or DPSC cell sources, on a PLGA scaffold, were able to regenerate a vascularized tissue that had histologic evidence of odontoblast-like cell differentiation and the spatially appropriate formation of dentin-like material onto the root canal walls. Although no specific growth factors were added to this mixture, it is important to note that the root canal walls were treated with 17% EDTA, an irrigant known to expose endogenous growth-factor proteins embedded in the dentinal walls.[268] This and other related studies provide strong impetus for clinical translational research evaluating various potential regenerative endodontic therapies.

CLINICAL STUDIES ON REGENERATIVE ENDODONTICS

To date, most case reports, case series, and retrospective studies published in regenerative endodontic have not fully incorporated the tissue-engineering concepts described. Instead, most of these reports present cases with variations of revascularization techniques.[62] These procedures were initially performed empirically with a strong focus on disinfection and the intentional bleeding into the root canal. However, it became obvious that these were in fact stem-cell based procedures with all three components of the tissue engineering triad present: stem cells,[135] growth factors,[26] and scaffolds.[23,234] Important preclinical studies previously described in this chapter have provided the foundational framework for a paradigm shift. This shift represents a clear departure from the traditional "disinfect the canals at all cost" to "disinfect while creating a microenvironment conducive for tissue engineering." Varied terminologies have been given to these procedures, including *revascularization,*[23] *revitalization,*[234] and *maturogenesis.*[6,101,115] Of these terms, the most popularly used has been *revascularization*. This term is largely based on the trauma literature observation that immature teeth could become *revascularized* after trauma. However, *revascularization* is a term better used for the reestablishment of the vascularity of an ischemic tissue, such as the dental pulp of an avulsed tooth. From this perspective, a focus on revascularization would ignore the potential importance of growth factors and scaffolds that are required for histologic recapitulation of the pulp-dentin complex. Although we appreciate that angiogenesis and the establishment of a functional blood supply are key requirements in the maintenance and maturation of a regenerating tissue, it is noteworthy that some of the published cases report positive responses to pulp sensitivity tests such as cold or electric pulp tests.[62] This is evidence that a space that was previously vacant (debrided root canal) may become populated with an innervated tissue supported by vascularity. Taken together, the core concepts of tissue engineering distinguish a regenerative treatment philosophy from a revascularization philosophy derived from certain trauma cases (which only occur in a low percentage of replanted teeth). Lastly, a number of procedures have been performed with intentional manipulation of the principles of tissue engineering such as use of autologous platelet-rich plasma (PRP),[234] platelet-rich fibrin (PRF),[208] and exogenous growth factors and scaffolds.[157] Thus, instead of using different terms for each variant of these procedures, we will refer to them simply as regenerative endodontic procedures (REPS), which include past, present, and future procedures that aim for the functional regeneration of the pulp dentin–complex.

Clinical Procedures Related to Regenerative Endodontics

Clinicians face several challenges when presented with an incompletely formed root in need of endodontic treatment.[62] Because the apex is not fully developed and often has a blunderbuss shape, cleaning and shaping of the apical portion of the root canal system can be difficult. The process is further complicated by the presence of thin, fragile dentinal walls that may be prone to fracture during instrumentation or obturation. In addition, the open apex increases the risk of extruding material into the periradicular tissues. Traditionally, an immature tooth with an open apex is treated by apexification, which involves creating an apical barrier to prevent extrusion. In many cases, this entails an involved, long-term treatment with $Ca(OH)_2$, resulting in the formation of a hard-tissue apical barrier.[30-52,78,77,96,97,247] However, a disadvantage of the traditional apexification procedures is that the short-term[195] or long-term[14,15,257,258] use of $Ca(OH)_2$ has the potential to reduce root strength.[14,257,258] This finding is consistent with a large case series using the traditional apexification protocol; it showed that a major reason for tooth loss following apexification was root fracture.[53] In a retrospective study, the use of calcium hydroxide in apexification procedures resulted in the fracture of 23% of the teeth treated during the follow-up period of up to 18 months.[110] The advent of one-step apexification, by the creation of artificial barriers using materials such as MTA,[146,181,233] has greatly decreased the number of appointments and time to completion. Importantly, one-step apexification has been shown to have as high a success rate as apexification with calcium hydroxide in resolving apical periodontitis (both symptoms and radiographic presentation).[247] However, apexification procedures do not generally result in further root development. A primary advantage of regenerative endodontic procedures in these cases is the greater likelihood there will be an increase in root length and root wall thickness, in addition to the possibility that the patient will regain vitality responses.

There have been numerous published cases of regenerative endodontic procedures. Investigators and clinicians have used a variety of medicaments to disinfect the canal space.[62] Approximately 51% of the cases included the use of a triple antibiotic paste (a 1:1:1 mixture of ciprofloxacin/

metronidazole/minocycline), whereas 37% used $Ca(OH)_2$ as an intracanal medicament.[62]

The development of the triple antibiotic paste was led in large part by Hoshino and colleagues.[100,202] They demonstrated the effectiveness of combinations of antibiotics (and in particular the high efficacy of the combination of ciprofloxacin, metronidazole, and minocycline) in eradicating bacteria from the infected dentin of root canals.[202] Astute practitioners realized that the triple antibiotic paste could be a valuable adjunct for revascularization procedures, because it could be used to create an environment favorable for the ingrowth of vasculature and regenerative cells by reducing or eradicating bacteria in the canal space of teeth with necrotic pulps and incompletely formed apices. The efficacy of the triple antibiotic paste in disinfecting necrotic root canal systems has been demonstrated in a preclinical model.[246] In this dog study, 60 teeth were accessed and infected by sealing dental plaque and sterile saline on a cotton pellet into the pulp chamber for 6 weeks. By the end of this period, each premolar was radiographically confirmed to have apical periodontitis. The canals were then sampled at three time points: before and after irrigation with 1.25% NaOCl, and 2 weeks after the delivery of the triple antibiotic paste into the root canal system using a Lentulo spiral. Before irrigation, all of the teeth had positive cultures for anaerobic bacteria, with a mean colony-forming unit (CFU) count of $1.7 \times 10.^8$ After irrigation with 1.25% NaOCl, 10% of the teeth sampled cultured bacteria free. The mean CFU count was 1.4×10^4, or an approximate 10,000-fold reduction in viable bacteria. After dressing with the triple antibiotic paste for 2 weeks, 70% of the teeth sampled cultured bacteria free. The mean CFU count was only 26, which is about another 1000-fold drop in bacteria. These findings were confirmed in another related dog study.[229] This study provides strong support for the effectiveness of triple antibiotic paste in disinfection of immature teeth with apical periodontitis.

As stated previously, calcium hydroxide has been the second most used intracanal medicament in published cases. This application represents a new use of a long established intracanal medicament in endodontics. Although $Ca(OH)_2$ appears to be less effective against some intracanal bacterial species than antibiotic paste formulations,[199] its use is associated with lower cytotoxicity to stem cells,[126,198] release of important bioactive growth factors from the treated dentin,[94] and greater survival and proliferation of stem cells in the presence of the conditioned dentin.[167] Also, the relatively short-term use of this medicament in regenerative procedures does not appear sufficient to reduce fracture resistance.[258] Another factor to consider when choosing an intracanal medicament is the ability to remove the medicament from the canal space. One study that addressed this question incorporated radioactive tracers in both calcium hydroxide paste (Ultracal, Ultradent, Inc.) and triple antibiotic paste (Champs Pharmacy, San Antonio, TX).[28] The radiolabeled medicaments were placed in extracted teeth with standardized root canals. After 28 days of incubation, canal spaces were irrigated with a standardized protocol using different techniques. Surprisingly, greater than 80% of the triple antibiotic paste could not be removed from the tooth (Fig. 10-8), and it was found not in the canal lumen, but greater than 350 µm into the dentinal tubules. In contrast, greater than 80% of calcium hydroxide was removed (see Fig. 10-8) with the remaining medicament present superficially within dentin.[28] This is an important finding, given that drugs

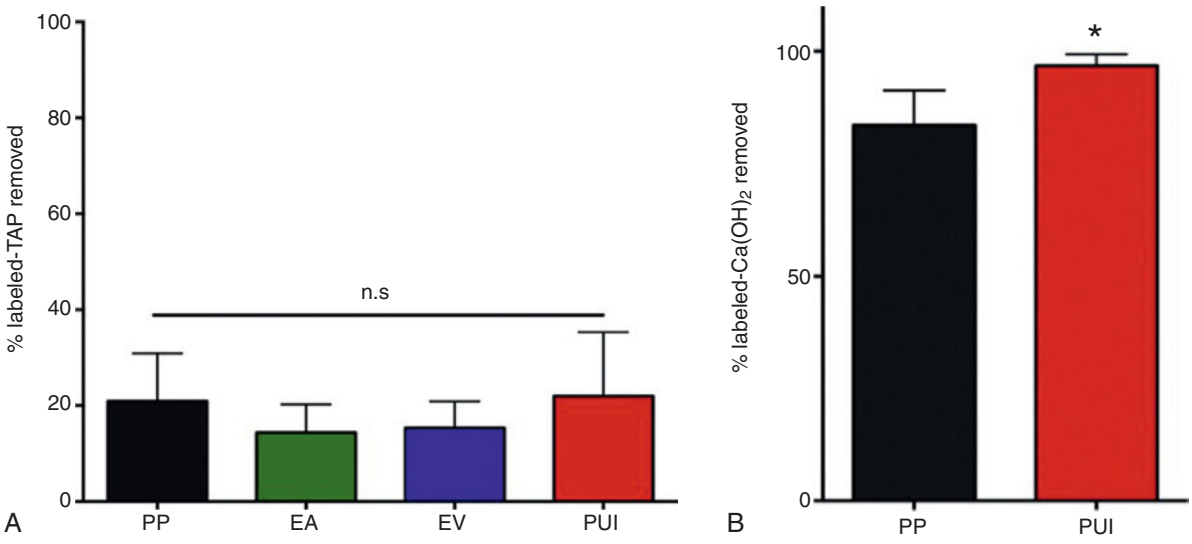

FIG. 10-8 TAP remains in dentin, whereas most $Ca(OH)_2$ is eliminated after endodontic irrigation. Radiolabeled TAP or $Ca(OH)_2$ was placed within canals of standardized root segments and incubated for 28 days at 37° C. The canals were flushed with standardized volumes of EDTA and saline using either positive pressure with a side-vented needle (PP) or positive pressure with ultrasonic activation of irrigants (PUI). There was no difference in labeled TAP removal among groups with only approximately 20% of the medicament being removed by the irrigation protocols **(A).** In contrast, > 80% of $Ca(OH)_2$ was removed, with more efficient removal observed in canals irrigated with PUI **(B).** Data are presented as the mean percentage of total radiolabeled medicament removal ± standard error of the mean. *$P < .05$ tested by the Student *t* test ($n = 12$/group). (Modified with permission from Berkhoff JA, Chen PB, Teixeira FB, Diogenes A: Evaluation of triple antibiotic paste removal by different irrigation procedures, *J Endod* 40:1172, 2014.)

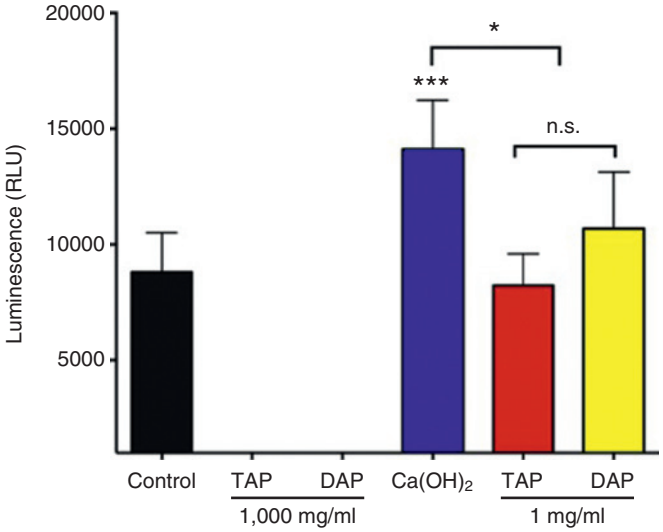

FIG. 10-9 Dentin conditioning for 7 days with medicaments used in REPs has a profound effect on SCAP survival. Standardized dentin disks were treated for 7 days with TAP or double antibiotic paste (DAP) (concentrations of 1000 mg/mL or 1 mg/mL), $Ca(OH)_2$ (Ultracal), or sterile saline (control). SCAP in a Matrigel scaffold (BD Biosciences, Bedford, MA) was seeded into the lumen of the disks after the medicaments were removed and cultured for 7 days. Cell viability (survival) was determined using a luminescent assay. SCAP culture on dentin treated with TAP or DAP at the concentration of 1000 mg/mL resulted in no viable cells. Conversely, dentin conditioning with TAP or DAP at the concentration of 1 mg/mL supported cell viability with no difference from untreated dentin disks (control). Greater survival and proliferation were detected in the group treated with $Ca(OH)_2$. Data are presented as mean ± standard deviation of relative luminescence units ($n = 12$/group). $^*P < .05$. $^{***}P < .001$. n.s., no statistical difference as tested by 1-way analysis of variance. (From Althumairy RI, Teixeira FB, Diogenes A: Effect of dentin conditioning with intracanal medicaments on survival of stem cells of apical papilla, *J Endod* 40:521, 2014.)

remaining within dentin are likely to have an effect on the fate of stem cells in contact with the treated dentin. One study revealed that triple antibiotic paste (TAP), when used at concentrations typically used in case reports (1 g/mL) and removed with standardized irrigation protocol, resulted in no SCAP survival.[9] In contrast, dentin treated with calcium hydroxide promoted SCAP survival and proliferation, and dentin treated with TAP at a 1 mg/mL had no effect on SCAP survival (Fig. 10-9). Thus, clinicians must carefully evaluate the advantages and disadvantages of each intracanal medicament while observing the ideal therapeutic concentration.[63]

Regardless of the intracanal medicament used, regenerative endodontic procedures have common features. Most of these published procedures reported minimal to no instrumentation.[62] This might be due, at least in part, to the concern of further weakening fragile dentinal walls and the difficulty of mechanically debriding canals of such large diameters. Because of the lack of mechanical debridement, clinicians relied on copious irrigation for maximum antimicrobial and tissue dissolution effects.[62] Canals were then medicated for a period that varied from days to several weeks. At the second visit, if signs and symptoms of disease had subsided, the medicament was removed, the canal was dried, and intracanal bleeding was evoked in most, but not all, cases.[62] In most cases, a coronal plug of MTA was placed over the blood clot or a collagen-based internal matrix, followed by a bonded coronal restoration. Each of these steps will be discussed later in light of the current American Association of Endodontists (AAE) considerations for REPs.

Overview of Clinical Regenerative Endodontic Procedures (REPS)

To date, most published clinical studies on REPS are composed of case reports and case series, with only one retrospective cohort study[110] and one prospective randomized clinical trial[157] on regenerative endodontics treatment available; no randomized controlled clinical trials have been published. Although case series do not provide definitive evidence to support a given treatment modality, they do have the advantage of being conducted on actual patients and thus provide a higher level of evidence than do preclinical studies. Although techniques for regenerative endodontics have varied in published case reports and case series,[62] there have been some consistent features worth noting. Nearly all reported cases involve patients 8 to 18 years old and teeth with immature apices with the exception of two published cases performed in mature, fully formed teeth.[182] Thus, the patient age appears to be an important factor in case selection because some studies suggest that younger patients have a greater healing capacity or stem cell regenerative potential.[10,12,55,128,154] Another important factor related to age is the stage of root development, because the large diameter of the immature (open) apex may foster the ingrowth of tissue into the root canal space and may indicate a rich source of mesenchymal stem cells of the apical papilla (SCAP; see Fig. 10-3).[104,197,224] These tissues are likely lacerated during the evoked bleeding step and constitute a likely source of mesenchymal stem cells delivered into the root canal space.[135] Another consistent finding reported in nearly every case is the lack of instrumentation of the dentinal walls related to concerns about the potential fracture of these thin, incompletely developed roots. The lack of instrumentation would be expected to have the benefit of avoiding generation of a smear layer that could occlude the dentinal walls or tubules. On the other hand, the lack of instrumentation could result in remaining bacterial biofilms within dentinal tubes. This is an issue that has not been evaluated in many cases, but the lack of canal wall instrumentation (and subsequent identification of bacteria in the apical dentin) has been suggested as a reason for failure of a regeneration case.[183] Nonetheless, these procedures are marked by robust disinfection protocols. Sodium hypochlorite, either alone or in combination with other irrigants, has been used to disinfect the canal space in most cases. In a majority of cases, a combination of triple antibiotic (minocycline, metronidazole, and ciprofloxacin) was left in the canal space for a period of days to weeks, so the disinfection protocol was primarily a chemical method rather than the chemomechanical approach used in conventional nonsurgical endodontic therapy.[62] In most cases, a blood clot formed in the canal.[62] The formation of a blood clot might serve as a protein scaffold, permitting the three-dimensional ingrowth of tissue.

Nearly all of these reports noted continued thickening of the root walls and subsequent apical closure. Examples of increased root length and thickening of root walls following regenerative endodontic procedures are seen in Figs. 10-10 through 10-13. Because of the lack of histology in most clinical

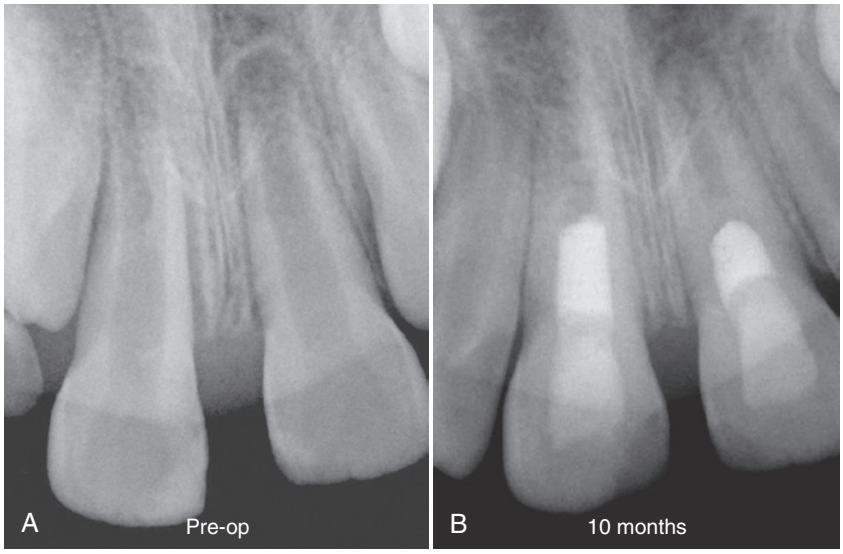

FIG. 10-10 Revascularization case illustrating treatment delivered to a 9-year-old male patient with a diagnosis of pulpal necrosis secondary to trauma, with a class 3 fracture in tooth #8 and a class 2 fracture in tooth #9 **(A)**. The patient reported moderate to severe pain in both teeth. The teeth were isolated, accessed, and irrigated with 5% sodium hypochlorite, followed by placement of a mixture of ciprofloxacin, metronidazole, and minocycline for 55 days. Upon recall, the teeth were isolated, and the triple antibiotic paste was removed by irrigation. Bleeding was established in tooth #9 but not in tooth #8, where CollaCote was placed prior to mineral trioxide aggregate (MTA). The root canal systems were sealed with white MTA and a composite restoration **(B)**. (Courtesy Dr. Alan Law.)

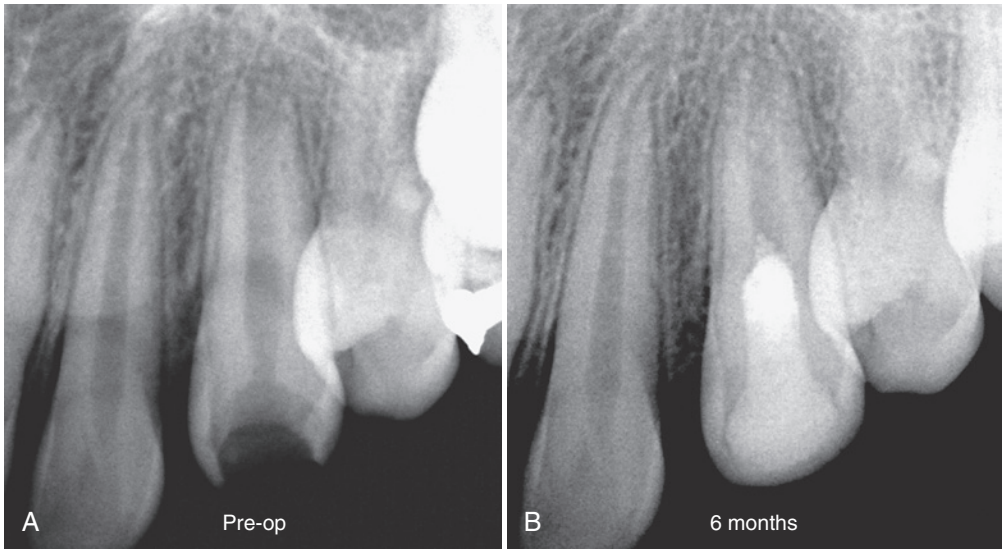

FIG. 10-11 Regenerative endodontic case illustrating treatment delivered to a 13-year-old female patient with a diagnosis of pulpal necrosis secondary to caries, with an unspecified prior history of trauma **(A)**. The tooth was isolated, accessed, and irrigated with 5% sodium hypochlorite, followed by placement of a mixture of ciprofloxacin, metronidazole, and minocycline for 21 days. Upon recall, the tooth was isolated, and the triple antibiotic paste was removed by irrigation. Bleeding was established in both teeth, and the root canal system was sealed with white mineral trioxide aggregate and a composite restoration **(B)**. (Courtesy Dr. Alan Law.)

cases, it should be recognized that radiographic findings of continued root wall thickness do not necessarily indicate that dentin was formed. Based on histologic results from preclinical studies, it is possible that the radiographic appearance of increased root wall thickness might be due to the ingrowth of cementum, bone, or a dentin-like material. Histologic evidence from human extracted teeth following REPS suggests that regenerated pulp tissue may be in the canal space,[123,206] and that the mineralized tissue along the dentinal walls appears to be cementum-like or osteodentin.[143,207] It should also be noted that in some of the case reports,[11,118] although the teeth were nonresponsive to pulp testing, the authors suggested that vital tissue was identified in the apical portion of the canal space. In these cases, necrotic tissue was removed until bleeding was

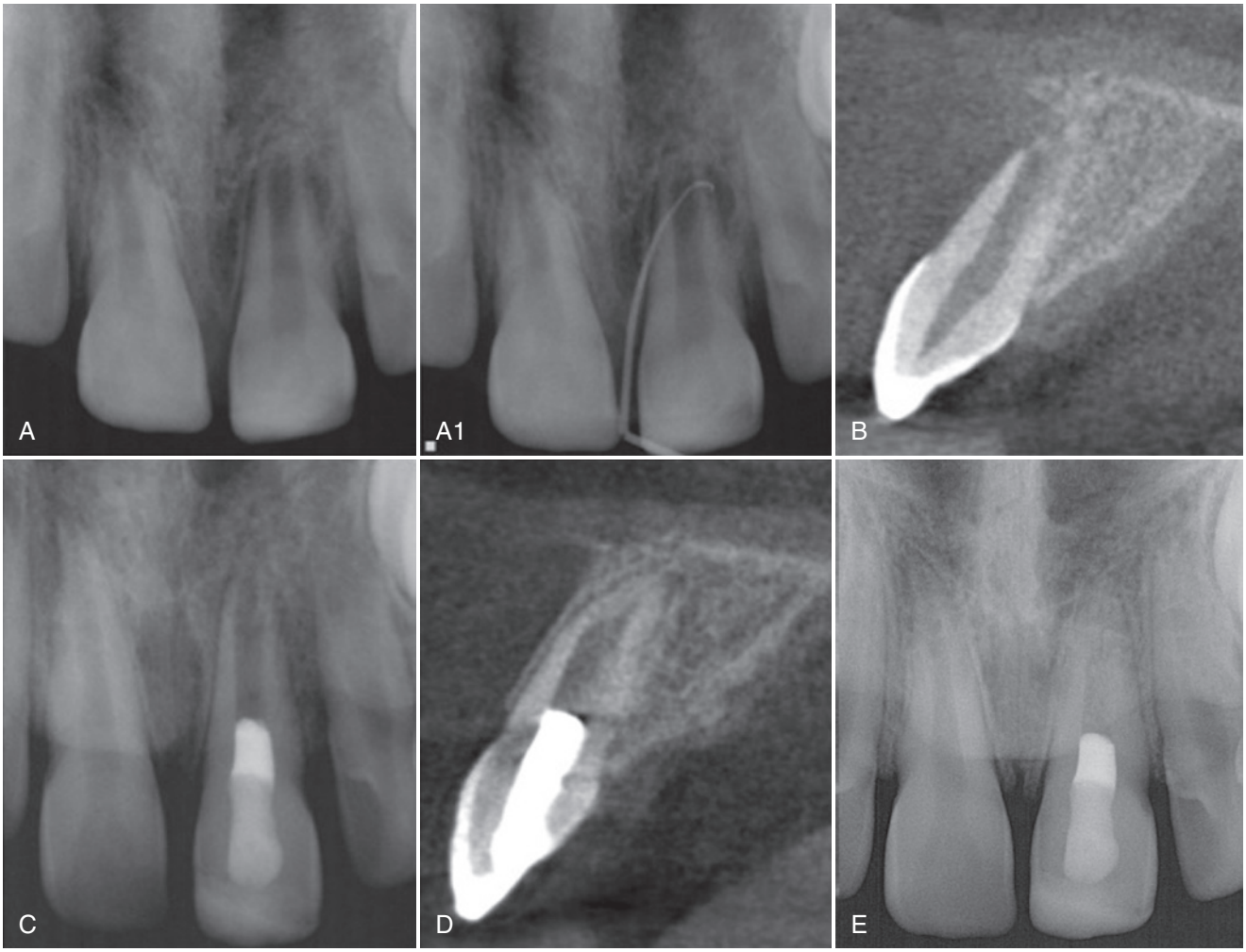

FIG. 10-12 Regenerative endodontic case illustrating treatment delivered to a 9-year-old male patient with a diagnosis of pulpal necrosis secondary to trauma and periradicular chronic apical abscess on tooth #9 seen on preoperative periapical radiograph **(A)** and CBCT **(B)**. The patient was asymptomatic and has draining facial sinus tract. The tooth was treated with a regenerative procedure using double antibiotic paste (DAP), a mixture of ciprofloxacin and metronidazole for 1 month. At the second appointment, the sinus tract had resolved. Then, the tooth was isolated, and the double antibiotic paste was removed by irrigation with 20 mL 17% EDTA. Intracanal bleeding was evoked from the apical tissues using a precurved #25 hand-file extending approximately 2 mm beyond the root apex. A Collaplug barrier was placed at the midroot level, then covered with 3 mm white MTA. Fuji II LC glass ionomer was used as a coronal seal of the MTA. The access was then restored with a composite resin and polished **(C)**. At the 1-year follow-up, the patient was asymptomatic, responsive to electrical pulp test (EPT), periodontal probings were no greater than 3 mm, and the tooth exhibited grade I mobility without coronal discoloration. Importantly, there was appreciable root maturation in addition to resolution of apical radiolucency seen on CBCT **(D)** and periapical radiograph **(E)**. (Courtesy Dr. Obadah Austah.)

observed, then the canals were disinfected with antibiotic paste or $Ca(OH)_2$.[62] One could argue that by leaving vital pulp tissue in the apical segment of the canal, the resulting progression of root formation is more similar to apexogenesis than revascularization. Although the biologic process may differ between the revascularization and apexogenesis cases presented in these case reports, the goals of the procedures are similarly advantageous and significant. In both procedures, there is healing of the periradicular tissues and a progression of root development in a tooth that would otherwise have had a progression of pulpal and periradicular pathosis.

A retrospective study has compared the radiographic changes in 48 revascularization cases to 40 control cases.[33] Although published revascularization cases have been treated with varying clinical protocols, they can be grouped by method of canal disinfection—namely, triple antibiotic paste, $Ca(OH)_2$ treatment, or formocresol treatment. This study applied a mathematical image-correction procedure that permitted the comparison of nonstandardized radiographs with subsequent statistical analysis of radiographic outcomes. The percentage change in root dimensions was first compared in two negative control groups (nonsurgical root canal treatment [NSRCT] and MTA apexification) predicted to have little to no change in root dimensions. This provides an internal test that the mathematical analysis was appropriate. The results indicate that these two negative control groups had minimal measured changes in root

FIG. 10-13 Regenerative endodontic therapy case illustrating treatment delivered to a 9-year-old female patient with a diagnosis of previously initiated endodontic therapy (pulpectomy) due to pain from pulpal inflammation on #29. Bilateral examination revealed presence of a talon cusp (dens evaginatus), which may have been the etiology of pulpal inflammation on #29. Radiographic examination revealed large periapical radiolucency on a periapical radiograph **(A)** and CBCT **(B)**. The tooth was treated with a regenerative procedure using double antibiotic paste as an intracanal medicament for 48 days. On the second visit, the tooth was isolated, and the double antibiotic paste removed by saline irrigation followed by a final rinse with 17% EDTA. The canal was dried and bleeding was evoked followed by placement of an internal matrix of Collaplug 3 to 4 mm below the CEJ. Mineral trioxide aggregate (MTA) was then placed over it and the tooth was restored with Fuji II LC and Build It. There was complete resolution of apical radiolucency on 1-month recall **(C)**, appreciable root development at 5-month recall **(D)**, and complete root maturation seen at 1-year recall on both perirapical radiograph and CBCT (**E** and **F**, respectively). Greater root maturation is observed at 2.5-year recall periapical radiograph **(G)** and CBCT (H and I). In addition, the tooth responded to EPT on the 1- and 2.5-year recall visits. (Courtesy Dr. Nikita Ruparel.)

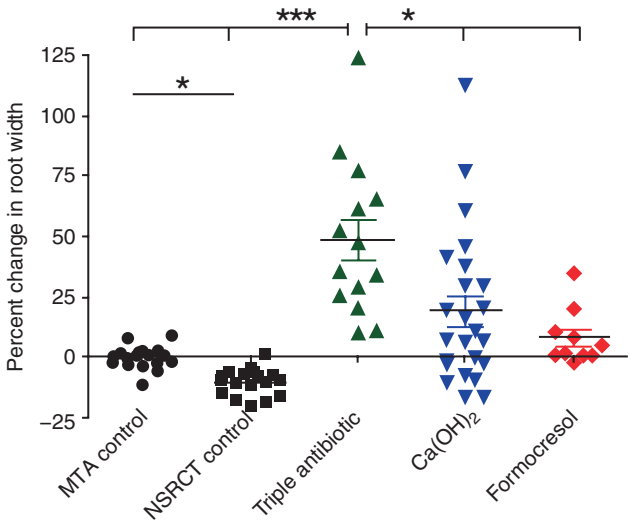

FIG. 10-14 Retrospective analysis of the percentage of change in root length from preoperative image to postoperative image, measured from the cementoenamel junction (CEJ) to the root apex in 40 control patients and in 48 patients following a revascularization procedure. ***P < 0.001 versus mineral trioxide aggregate (MTA) apexification control group (n = 20) and NSRCT control group (n = 20). P < 0.05 versus MTA control group only. Median values for each group are depicted by horizontal line, and individual cases are indicated by the corresponding symbol. (From Bose R, Nummikoski P, Hargreaves K: A retrospective evaluation of radiographic outcomes in immature teeth with necrotic root canal systems treated with regenerative endodontic procedures, *J Endod* 35:1343, 2009.)

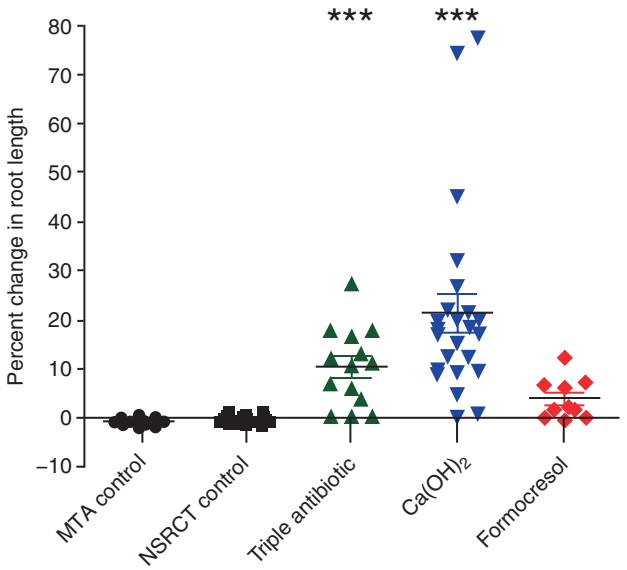

FIG. 10-15 Retrospective analysis of the percentage of change in dentinal wall thickness from preoperative image to postoperative image, measured at the apical third of the root (position of apical third defined in the preoperative image) in 40 control patients and in 48 patients following a revascularization procedure. ***P < 0.001 versus mineral trioxide aggregate (MTA) apexification control group and NSRCT control group. P < 0.05 versus NSRCT control group only. P < 0.05 versus calcium hydroxide ($Ca[OH]_2$) and formocresol groups. P < 0.05 versus NSRCT control group only. (From Bose R, Nummikoski P, Hargreaves K: A retrospective evaluation of radiographic outcomes in immature teeth with necrotic root canal systems treated with regenerative endodontic procedures, *J Endod* 35:1343, 2009.)

width (Fig. 10-14) or root length (Fig. 10-15), with the anticipated finding that instrumentation with files of greater taper resulted in a slight but detectable loss of apical root wall width. The results indicated that revascularization treatment with either the triple antibiotic paste or $Ca(OH)_2$ medicament produced significantly greater increases in root length compared with either the MTA or NSRCT control groups. Also, treatment with the triple antibiotic paste produced significantly greater increases in root wall thickness compared with the MTA and NSRCT control groups. Treatment with $Ca(OH)_2$ resulted in significantly greater changes in root wall thickness compared with the NSRCT group, but no differences were observed between these medicaments and the MTA apexification group. Finally, the triple antibiotic paste produced significantly greater differences in root wall thickness compared with either the $Ca(OH)_2$ or formocresol groups. In general, the formocresol group showed the smallest improvement in root length and wall thickness. Secondary analyses indicated that a 12- to 18-month recall is probably the minimal time to judge radiographic evidence of root development, although later time points (36 months) often demonstrate continued root development. Jeeruphan and colleagues (2012) used a similar method to measure changes in root length and width following apexification versus revascularization.[110] They reported that teeth treated with revascularization showed a significantly greater percentage increase in root length (14.9%) compared with teeth treated by either MTA apexification (6.1%) or calcium hydroxide apexification. They also reported that the revascularization protocol produced significantly greater percentage increases in root width (28.2%) compared with teeth treated by either MTA apexification (0.0%) or calcium hydroxide apexification. In addition to measuring changes in root dimensions, the authors reported survival (defined as retention of the tooth in the arch at the time of the postoperative recall) rate of 100% at an average of 14 months after revascularization treatment. This compared favorably with survival rates of 95% for MTA apexification and 77% for calcium hydroxide apexification. Although prospective randomized clinical trials with standardized radiographic assessment are clearly required, the results of this retrospective study are consistent with a robust outcome of revascularization procedures, particularly when medicated with either a triple antibiotic paste or a $Ca(OH)_2$ medicament. The demonstration of continued root development does not reveal whether this radiopaque material is dentin, cementum, or bone, so given the known reliance of stem cells on an appropriate scaffold and growth factor combination, this will be an important focus for future research efforts.

Example of a Revascularization Protocol

Based on current research, several factors can be reviewed when considering a protocol for revascularization treatment. The first issue is case selection; the best available evidence indicates that this treatment should be considered for the incompletely developed permanent tooth that has an open apex and is negative to pulpal responsiveness testing. Although the ultimate goal of this approach may include a tissue engineering–based method of pulpal regeneration in the fully developed permanent tooth, it should be recognized that

current revascularization protocols have not been developed or evaluated for these more challenging cases. Informed consent should include the number of appointments (at least two), the possible adverse effects (primarily potential staining of the crown), the potential lack of response to treatment and alternative treatments, and possible posttreatment symptoms. Because the canal space will not be accessible following revascularization, teeth requiring retention in the canal space for the restoration are not good candidates for REPs. Clinical staining of the crown and any root structure above the gingival margin appears to be due to the presence of minocycline.[120,185,191] This outcome can be minimized by using a delivery system that restricts the drug below the cementoenamel junction (CEJ).[191] When it does occur, it can often be reduced or eliminated by a walking bleach method with sodium perborate. The use of mineral trioxide aggregate (MTA; ProRoot, Dentsply Tulsa Dental, Tulsa, OK), in both gray and white form variants,[27,36,39] may also cause tooth discoloration, which can also be reduced or eliminated by a walking bleach technique.[36] Alternative treatments that should be discussed with the patient and guardian would include MTA apexification, no treatment, or extraction.

At the first appointment (Fig. 10-16, *A-E*), the treatment alternatives, risks, and potential benefits should be described to the patient and guardian after collecting clinical information and establishing pulpal and apical diagnoses. Following informed consent, the tooth is anesthetized, isolated, and accessed. Minimal instrumentation should be accomplished, but the use of a small file to "scout" the root canal system and determine working length is important. If sensation is experienced within the canal system, this may suggest that some residual vital pulp tissue remains.[59] The root canal system should be copiously and slowly irrigated. Because of its proven efficacy as a canal disinfectant and a tissue dissolution agent, sodium hypochlorite (NaOCl) has been the irrigant of choice in most of the revascularization case reports.[62] However, studies have demonstrated that NaOCl is cytotoxic to stem cells.[62,142,235] Importantly, a minimally deleterious effect was seen with the use of 1.5% NaOCl followed by 17% EDTA. Thus, a lower concentration (1.5%) of NaOCl should be considered as the standard irrigant for REPs. Moreover, the use of chlorhexidine should be limited or avoided because it does not have tissue dissolution capability and has also been shown to be cytotoxic to stem cells.[235] Because canal disinfection relies considerably on chemical irrigants, it is important to place the needle into the apical third and irrigate using needles with a closed end and side-port vents (e.g., Max-I-Probe needles), together with a slow rate of infusion, to help to reduce any irrigants passing through the open apex.

The root canal system is then dried with sterile paper points, and the antimicrobial medicament is delivered into the root canal space. The best available evidence would support the use of either a triple antibiotic paste or $Ca(OH)_2$. Triple antibiotic paste (TAP) and $Ca(OH)_2$ have been shown to be effective (see Figs. 10-9 and 10-10). The TAP has the advantage of being a very effective antibiotic combination against endodontic microorganisms[203]; its efficacy is supported by its use in most published cases.[62] This combination is not approved by the U.S. Food and Drug Administration (FDA), however, and carries a potential for minocycline staining of the crown. In addition, triple antibiotic paste has been shown to be cytotoxic to stem cells. It is important to note that Ruparel and colleagues (2012)[197] have shown that higher TAP at concentrations used in many published case reports has a profound detrimental effect on stem cell survival. Importantly, it had minimal to no deleterious effect when used at the concentrations of 0.1 or 1 mg/mL, whereas these concentrations are several orders of magnitude greater than those required to eliminate bacteria from infected dentin.[203] In addition, the same report showed that all concentrations of calcium hydroxide promoted stem cell survival. Moreover, TAP has an indirect detrimental effect on stem cell survival, as cells do not survive when in contact with dentin previously treated with TAP at the concentration of 1 g/mL.[167] This effect is largely avoided if TAP is used at the lower concentration of 1 mg/mL.[167] Thus, The use of TAP at the concentration of 1 mg/mL or calcium hydroxide pastes as intracanal medicaments seems warranted; however, further investigation is needed to determine appropriate formulations and lower concentrations of antibiotic pastes, if necessary, to be used. When performing REPs on teeth in aesthetic areas, practitioners should consider eliminating minocycline from the antibiotic paste, sealing the coronal dentin with a dentin bonding agent or composite,[120] or using calcium hydroxide paste. After antimicrobial medicament is placed, the tooth is then sealed with a sterile sponge and a temporary filling (e.g., Cavit), and the patient is discharged for 3 to 4 weeks.

At the second appointment (see Fig. 10-16, *F-M*), the patient is evaluated for resolution of any signs or symptoms of an acute infection (\swelling, sinus tract pain, etc.) that may have been present at the first appointment. The antimicrobial treatment is repeated if resolution has not occurred.[47,113] In most reported cases, the acute signs and symptoms resolved after treatment with the intracanal medicament.[62] Because revascularization-induced bleeding will be evoked at this appointment, the tooth should not be treated with a local anesthetic containing a vasoconstrictor. Instead, 3% mepivacaine can be used, which will facilitate the ability to trigger bleeding into the root canal system.[185] Following isolation and reestablishment of coronal access, the tooth should be copiously and slowly irrigated, possibly together with gentle agitation with a small hand file to remove the antimicrobial medicament. When choosing an irrigant at the second appointment, it is worth considering that irrigants such as NaOCl and chlorhexidine may be cytotoxic to stem cells either directly[69] or indirectly after dentin has been exposed to the irrigants.[69,142,235] It has been demonstrated that exposure of dentin to 5% to 6% sodium hypochlorite leads to decreased stem cell survival and odontoblastic differentiation.[81,142,235] This indirect effect is likely related to various deleterious effects of sodium hypochlorite on the dentin matrix leading to reduced cell attachment[192] and a decrease in dentin matrix–derived growth factors such as TGF-β1.[268] Thus, it is prudent to avoid sodium hypochlorite on the second visit. Instead, irrigation with 17% EDTA may be advantageous because it has been shown to release growth factors from dentin[26,268] and promote stem cell survival and differentiation.[142] In addition, Galler and colleagues (2011), using dentin cylinders transplanted into immunocompromised mice, showed that dental pulp stem cells seeded onto EDTA-conditioned dentin differentiated into odontoblast-like cells and had cellular processes extending into the dentin.[81] On the other hand, stem cells differentiated into osteoclasts/odontoclasts caused resorption on the dentinal walls if dentin was previously treated with sodium hypochlorite. Thus, the use of 17% EDTA as the last irrigant promotes

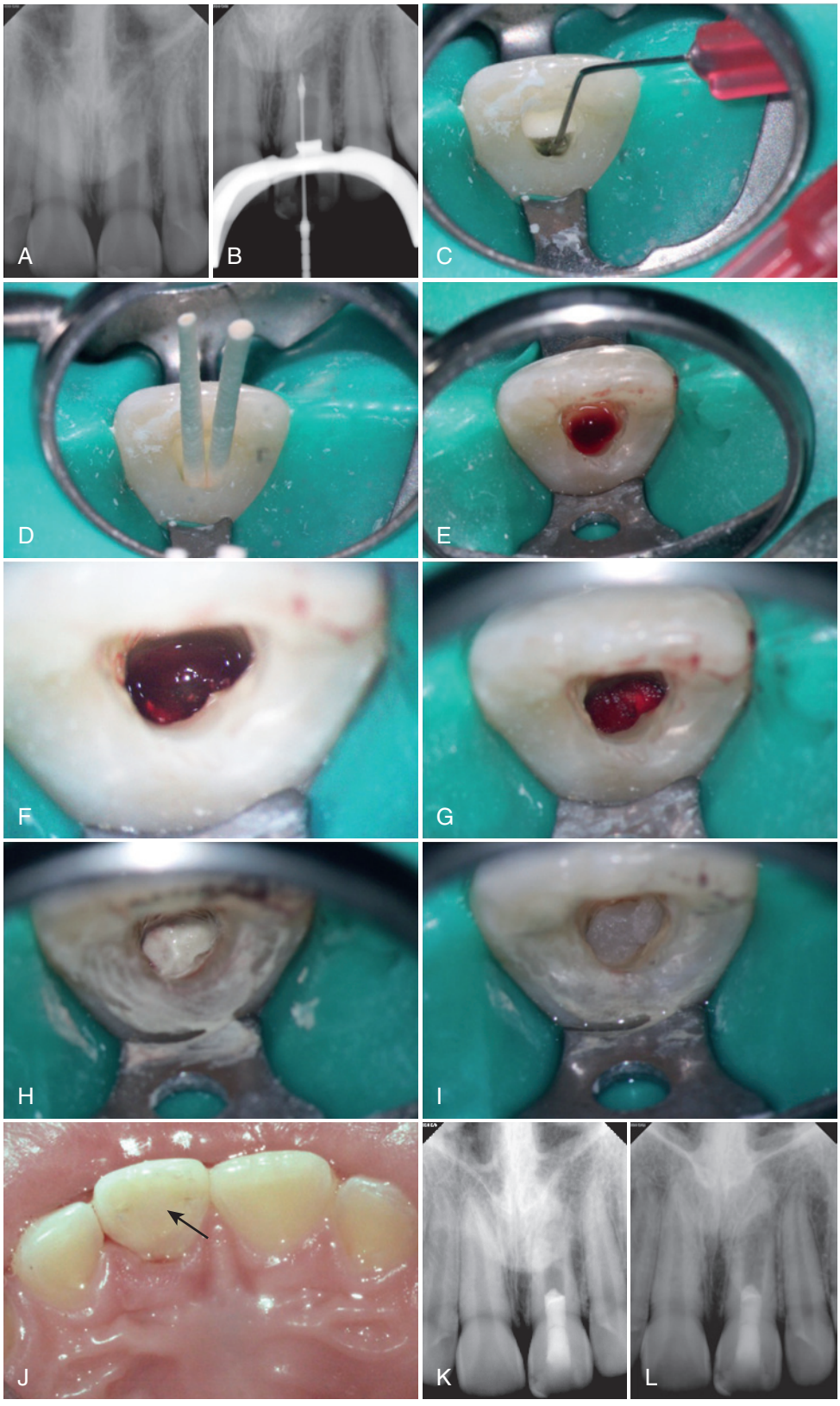

FIG. 10-16 Example of a revascularization case. Treatment was delivered to a 12-year-old boy who suffered trauma on tooth# 9 2 years prior to the appointment. First appointment: Clinical examination revealed a pain upon percussion and palpation **(A)**. A working length file was placed into the root canal system **(B)**. The tooth was slowly irrigated with 20 mL of 1.5% sodium hypochlorite (NaOCl) followed by 20 mL of saline using a Max-I-Probe needle inserted to the apical third **(C)**. The canals were dried and medicated with calcium hydroxide paste (Ultracal) **(D)**. The patient returned asymptomatic 1 month later. The tooth was isolated, accessed, and the medicament removed by slow irrigation with 1.5% NaOCl followed by 17% EDTA. After the canal was adequately dried, intracanal bleeding was achieved by lacerating the apical tissues **(E)**. CollaPlug as seen in access **(F)**. CollaPlug was placed below the CEJ **(G)** to serve as a matrix to position the white mineral trioxide aggregate coronal to the blood clot **(H)**. The tooth was then sealed with a layer of Fuji IX **(I)**, etched and restored with a composite **(J)**. A final radiograph was taken **(K)**. The tooth responded to electrical pulp tested at the 1-year recall, and it demonstrated apical closure and moderate dentinal wall thickening **(L)**. (Courtesy Dr. Anibal Diogenes.)

the attachment, proliferation, and odontoblastic differentiation of stem cells.

After drying the canal system with sterile paper points, a file is placed a few millimeters beyond the apical foramen, and the apical tissue is lacerated with bleeding up to 3 mm from the CEJ. A small piece of Colla-Plug (Zimmer Dental, Carlsbad, CA) may be inserted into the root canal system to serve as a resorbable matrix to restrict excessive apical positioning of the MTA. About 3 mm of MTA is then placed. MTA has been used in many of the case reports and may have advantages over other materials because it creates a bacteria-tight seal, is biocompatible, and has conductive and inductive properties.[118,148,166,170] However, the use of MTA, including white MTA, has been associated with tooth discoloration; thus, its use should be avoided in aesthetic areas. Biodentin is a bioactive material with desirable handling characteristics and less susceptibility to staining.[124] Although this material is relatively new, there have been encouraging studies demonstrating that it induces stem cell proliferation and odontoblastic differentiation.[137,138,262] Indeed, the use of Biodentine in pulpotomies and direct pulp capping cases is associated with an adequate dentin bridge and continued root development.[29,59,204] Thus, MTA and Biodentine appear suitable to be used in regenerative endodontic procedures.

In addition to follow-up visits in the first several months after REPs to assess for signs of healing, a 12- to 18-month recall should be considered as a reasonable time point to radiographically assess apical healing and root development.[33]

Box 10-1 summarizes the steps in a regenerative endodontic procedure. Importantly, these considerations are based on the best available evidence and are likely to change as the field progresses.

Clinical Measures of Treatment Outcome

The goal of conventional endodontic therapy is to maintain or restore the health of periradicular tissues by preventing or healing apical periodontitis. The goals of regenerative procedures extend beyond the goals of conventional endodontic therapy and include continued root development and reestablishment of pulpal vitality. As mentioned earlier, contemporary regenerative endodontic procedures are being modified and evolving toward accomplishing these additional objectives, which are not achievable with conventional nonsurgical root canal therapy. Accordingly, the definition of success for regenerative endodontic procedures has not been fully determined. Also, assessments of clinical outcomes of REPs are not always easily achievable. Although the resolution of signs and symptoms of disease can be easily assessed by a clinical and radiographic examination, evidence of root development and vitality responses are more challenging.

It is well established that the primary goal of endodontics is the prevention and healing of pulpal and periradicular inflammation (i.e., resolution of signs and symptoms of disease). Immature teeth with pulpal necrosis have been traditionally treated with apexification procedures.[78] A retrospective study reported a success rate of 93.5% for apexification procedures performed in one visit and 90.5% for those performed in two-visit appointments.[247] Another retrospective study reported that apexification procedures resulted in complete healing of apical periodontitis in 85% of the treated teeth.[99] For teeth treated with regenerative procedures, the healing of apical periodontitis has been reported to vary from 90% to 100%.[44,110,156] Most of these studies did not directly compare apexification to regenerative endodontic procedures with the exception of one prospective clinical trial[157] and one retrospective study.[110] Thus, the success rate for both apexification and regenerative endodontic procedures are similarly high (i.e., above 90%) based on most current evidence. Nonetheless, larger randomized clinical trials with adequate follow-up periods are warranted to provide greater insight in the long-term outcome for both apexification and regenerative procedures.

BOX 10-1

Treatment Procedures for Regenerative Endodontics

First Treatment Visit for Regenerative Endodontics

1. Informed consent, including explanation of risks and alternative treatments or no treatment.
2. After ascertaining adequate local anesthesia, rubber dam isolation is obtained.
3. The root canal systems are accessed and working length is determined (radiograph of a file loosely positioned at 1 mm from root end).
4. The root canal systems are slowly irrigated first with 1.5% NaOCl (20 mL/canal, 5 min) and then irrigated with saline (20 mL/canal, 5 min), with irrigating needle positioned about 1 mm from root end.
5. Canals are dried with paper points.
6. Calcium hydroxide or an antibiotic paste or solution (combined total of 0.1 to 1 mg/mL) is delivered to canal system.
7. Access is temporarily restored.

Final (Second) Treatment Visit for Regenerative Endodontics (Typically 2 to 4 Weeks after the First Visit)

1. A clinical exam is first performed to ensure that that there is no moderate to severe sensitivity to palpation and percussion. If such sensitivity is observed, or a sinus tract or swelling is noted, then the treatment provided at the first visit is repeated.
2. After ascertaining adequate local anesthesia with 3% mepivacaine (no epinephrine), rubber dam isolation is obtained.
3. The root canal systems are accessed; the intracanal medicament is removed by irrigating with 17% ethylenediaminetetraacetic acid (EDTA) (30 mL/canal, 5 min) and then a final flush with saline (5 mL/canal, 1 min).
4. The canals are dried with paper points.
5. Bleeding is induced by rotating a precurved K-file size #25 at 2 mm past the apical foramen with the goal of having the whole canal filled with blood to the level of the cementoenamel junction.
6. Once a blood clot is formed, a premeasured piece of Collaplug (Zimmer Dental Inc., Warsaw, IN) is carefully placed on top of the blood clot to serve as an internal matrix for the placement of approximately 3 mm of white MTA (Dentsply, Tulsa, OK) or Biodentin (Septodont).
7. A (3- to 4-mm) layer of glass ionomer layer (e.g., Fuji IX, GC America, Alsip, IL, or other) is flowed gently over the bioactive coronal barrier and light cured for 40 secs.
8. A bonded reinforced composite resin restoration (e.g., Z-100, 3M, St Paul, MN, or other) is placed over the glass ionomer.
9. The case needs to be followed-up at 3 months, 6 months, and yearly after that for a total of 4 years.

Continued root development is another desirable outcome of regenerative endodontic procedures. Most published cases of regenerative endodontics report continued root development or apical closure.[62] It is important to note that there were highly varied treatment protocols in the case reports and case series.[62] In addition, the reports of continued root development are often subjective, without an attempt to quantify root development. This shortcoming is due, at least in part, to the difficulty in acquiring standardized radiographs in young patients undergoing rapid cranioskeletal development. A study reported the methodology to perform computational digital correction and the quantification of root length and width on nonstandardized regenerative endodontics radiographs.[33] Studies that have used this methodology to quantify root development have collectively reported that apexification procedures did not promote any root development, whereas regenerative procedures allowed mean increases in root width of 25% to 35.5%[33,110,114] and in root length of 11.3% to 14.9%.[33,110,114] This methodology has been modified to measure changes in radiographic root area (RRA).[76] Regenerative endodontic procedures resulted in a 31.6% increase in RRA, whereas apexification procedures, as with the other studies, had no effect.[76] Thus, regenerative endodontic procedures allowed continued root development not seen with other treatment alternatives.

Although achieving regeneration of pulp tissue continues to be a preferred objective, an alternative acceptable outcome—retention of a tooth with healed apical tissue—could be considered satisfactory. A retrospective study by Jeeruphan and colleagues[110] compared survival rates (defined as retention of the tooth in the arch at the time of the postoperative recall) of teeth that underwent revascularization procedures versus teeth that underwent calcium hydroxide or MTA apexification. They reported a 100% survival rate for revascularization-treated teeth versus 95% for MTA apexification cases and 77.2% for calcium hydroxide apexification cases.[110] Survival of teeth treated with these procedures is noteworthy, because premature loss of these permanent teeth would likely have led to a loss of alveolar bone and compromised future replacement of the tooth. Retention of a tooth following a revascularization procedure, even in the absence of continued root development, would potentially allow for implant placement (if necessary) following alveolar bone growth[37,184] and should be considered an acceptable outcome for patients, parents, and practitioners.

Vitality responses have been reported in approximately 50% of the published cases.[62] Responses to electric pulp tester (EPT) are more commonly reported than cold responses. These responses to vitality testing (with either cold or EPT), as well as the lack of signs and symptoms of pathosis, suggest the presence of functioning tissue in the canal space. Vitality responses, in addition to continued root development, are a desirable outcome because "normal" nociception is protective and suggest immune-competence of the vascularized tissue. However, the lack of responses should not be interpreted as failure because there have been several reports of cases demonstrating healing of apical periodontitis and appreciable root development in the absence of a positive response to vitality testing. Although vitality response is a desirable outcome because it suggests a "functional regeneration," its presence likely depends on several factors, including the depth level of coronal restoration placement and the degree of intracanal mineralization. Thus, from a clinical perspective, the ideal clinical outcome is an asymptomatic tooth that does not require retreatment and demonstrates continued root development and perhaps vitality responses.

It is worth noting that the published revascularization cases have shown increased root wall thickness that is limited to the midroot and apical root.[62] There has been no demonstration of increased root thickness in the cervical area, an area shown to be prone to fracture in immature teeth with a history of trauma and subsequent endodontic treatment. Future clinical studies should focus on not only on making these procedures more predictable, but extending regeneration into the cervical area, potentially strengthening this area and decreasing the risk of root fracture.

SUMMARY

The field of regenerative endodontics is rapidly advancing. This progress is based on the principles of tissue engineering—namely, the spatial delivery of appropriate cells, scaffolds, and growth factors. Similar to most rapidly developing fields, the preclinical area of research has outpaced translational clinical studies. Preclinical studies have identified several mesenchymal stem cell sources capable of differentiating into odontoblast-like cells, as well as candidate scaffolds and growth factors capable of guiding this development. The initial preclinical animal studies indicate that using all three components of the tissue engineering triad (stem cells, growth factors, and scaffolds) can result in complete regeneration of the pulp-dentin complex. There is a delicate balance between disinfection of the root canal system and the interplay of these three components that warrants more research. It is likely that this combined approach of in vitro and in vivo preclinical research will greatly advance our understanding of the conditions necessary to regenerate a functional pulp-dentin complex.

The translational nature of regenerative endodontic research is allowing for changes to take place in the clinical practice in a relatively short time. This cross-talk between basic and clinical sciences is largely fueled by the realization that all three components of the tissue engineering triad are already present in revascularization procedures: stem cells, scaffold (blood clot), and growth factors (from dentin and blood). Preclinical studies that evaluated the effect of irrigants[81,142,235] and medicaments[167,197] on the survival of stem cells, release of growth factors from dentin,[231,268] and odontoblastic differentiation[41,81] are shaping the future generations of regenerative procedures. Further, the incorporation of other scaffolds such as PRP, PRF, and gelatin sponges has been used in the clinical practice with encouraging results.[123,143,157,208,234] Discussion about the requirements of an appropriate scaffold and growth factor was begun in the 1960s by Nygaard-Østby,[173] who had no access to our contemporary instruments, materials, and knowledge base of tissue engineering. Although clinical revascularization procedures do not constitute the ideal regenerative treatment, it is important to note that they do generate a scaffold (fibrin) and growth factors (from platelets and access to proteins embedded in the dentinal walls), and the clinical outcome results in continued radiographic root development of the immature permanent tooth with a diagnosis of pulpal necrosis. Thus, REPs provide a treatment of high value in cases with an otherwise poor prognosis. Histologic analyses of REPs conducted in patients[123,143,171,206] or animals[168,193,243] suggest that the increase

in root dimensions is often due to deposition of cementum-like material, osteodentin, dentin, or bone.

Future clinical research will likely focus on further translating basic research findings into improved regenerative procedures. For example, the formation of a cementum-like material on the dentinal walls may lead to studies evaluating benefits of REPs for overall tooth resistance to fracture. In addition, it is clear that the multipotent nature of many mesenchymal stem cell types could contribute to the finding of cementum deposition. Controlled differentiation of stem cells into odontoblasts is an important area of research and amenable to tissue-engineering concepts. The development of delivery systems that permit structural reinforcement of the cervical area (or, ideally, the pulp chamber) might provide clinical opportunities to regenerate lost tooth structure, thereby permitting natural teeth to be retained instead of possible fracture or extraction. Finally, the ultimate and long-term goal for regenerative endodontic procedures should be to treat the fully formed permanent tooth. Although this situation is more complex than the immature tooth with an open apex and a ready source of stem cells, it provides the unique potential of saving the natural dentition while restoring the sensory, immunologic, and defensive properties of the pulp-dentin complex.

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CHAPTER 11

Evaluation of Outcomes

YUAN-LING NG | KISHOR GULABIVALA

CHAPTER OUTLINE

Context of Evaluating Endodontic Outcomes
Types of Disease and Their Treatment
What Are Surrogate Outcome Measures?
Types of Outcome Measures
What Is the Purpose of Evaluating Outcomes?
Effectiveness of Procedures
Factors Affecting Outcomes
Value for Prognostication
Outcome Measures for Endodontic Treatment
Outcome Measures for Vital Pulp Therapy Procedures
Outcome Measures for Nonsurgical Root Canal Treatment and Retreatment
Outcome Measures for Periapical Surgery
Outcomes of Vital Pulp Therapy Procedures
Indirect Pulp Capping (One-Step versus Stepwise Excavation)
Direct Pulp Capping
Pulpotomy
Summary of Prognostic Factors for Vital Pulp Therapy
Outcomes of Nonsurgical Root Canal Treatment
Factors Affecting Periapical Health or Healing Following Root Canal Treatment
Factors Affecting Tooth Survival Following Root Canal Treatment
Impact of Root Canal Treatment on Quality of Life
Outcome of Nonsurgical Retreatment
Outcome of Surgical Retreatment
Factors Affecting Periapical Health or Healing Following Periapical Surgery and Root-End Filling
Factors Affecting Periodontal Incisional Wound Healing
Factors Affecting Tooth Survival Following Periapical Surgery and Root-End Filling
Impact of Periapical Surgery on Quality of Life
Concluding Remarks

CONTEXT OF EVALUATING ENDODONTIC OUTCOMES

The development of medical and dental practices has been guided by prevailing philosophies and consensus of expert opinions. The foundations of modern endodontics were shaken when Billings[97] brought the apparent relationship between oral sepsis and bacterial endocarditis to the attention of dentistry and medicine. He had strengthened the concept further from Miller[134] who had coined the term *focus of infection* to highlight a possible link between "mouth germs" and systemic disease. The disastrous consequences of the focal infection era were sealed when Hunter[96] delivered his famous address at McGill University. Fears of fatal oral sepsis from deficient root canal treatments led to widespread extraction of pulpless teeth. Endodontics virtually disappeared from many dental schools, whereas in some areas, treatment was restricted to anterior teeth. The focal infection theory reigned for about 50 years[83] until around 1940.

The discipline of endodontics was rescued by individual, diligent practitioners in Europe and USA who meticulously recorded their treatments, as well as their outcomes, to demonstrate the effectiveness of the procedures in controlling root canal infection. It was through these individual endeavors of merit that the reputation of endodontic procedures was restored and the discipline granted its specialist status in 1952 in the United States.[59]

Toward the 1990s, the improvements in general health and longer survival of Western populations as well as their teeth, coupled with the rising costs of health care for longer-living individuals, prompted a host of reevaluations of the altered economic burdens on society. Among them, the *cost-effectiveness of treatment procedures* for management of diseases loomed large. Thus began the era of evidence-based medicine and dentistry, with its emphasis on costs, benefits, and outcomes of treatment. Attempts to pool outcome data for greater power brought with it the realization that studies originating from different centers varied in diverse ways, leading to very heterogeneous data that challenged formal attempts at drawing definitive conclusions. Characterization of data types and its quality prompted calls for standardization of approaches in measuring outcomes to enable more meaningful pooling.

Because of the better established science of evidence assessment, a modern revisitation of the "focal infection era"[94] did not lead to the same threat to the endodontic discipline that was evident at the beginning of the 20th century. In fact, it was suggested as an opportunity to secure research funding for assessing and managing the importance of dental care on systemic health.

Interestingly, the modern threat to endodontics was posed by the cost-economic pressures exerted on treatment planning decisions centering on the question of whether to "save the tooth" or extract it with replacement by an implant-supported crown.[238] Once again, the science of evidence-based practice

has helped to avert irrational treatment recommendations leaning toward extraction of savable teeth.[54,100,103]

Types of Disease and Their Treatment

The statement, "Endodontists provide endodontic treatment to manage endodontic disease" is a gross oversimplification that masks important subtleties in recognition of the nature of the diseases and how best to address them. Endodontists manage inflammation of the specialized connective tissues within and surrounding the teeth; more specifically, they deal with inflammation that generally commences in the pulp tissue and progresses to the periradicular tissues via portals of communication that convey the neurovascular supply. Incipient or established pulpitis may be managed by vital pulp therapy when judged reversible, largely because a sufficient body of pulp tissue remains healthy and unaffected. More advanced pulpitis that approaches the foramina into the periodontal ligament may require root canal treatment. When pulpal inflammation, necrosis, or infection encroaches on the apical periodontal ligament, root canal treatment is required. Apical periodontitis that persists despite technically adequate root canal treatment may require root canal retreatment, periradicular surgery, or extraction to manage the source of the persistent disease. *Endodontic treatment* is therefore a collective and nonspecific term for a range of procedures directed at managing the spread of pulpal inflammation or infection. Endodontic treatment encompasses the following procedures:

1. Vital pulp therapy (indirect pulp therapy, direct pulp capping, pulpotomy, regenerative pulp therapy)
2. Nonsurgical root canal treatment
3. Nonsurgical root canal retreatment
4. Surgical retreatment

The ideal outcome for endodontic treatment consists of controlled reduction of inflammation, accompanied by healing through regeneration, although sometimes repair may follow instead. Unfortunately, none of the involved tissues are within the direct sight of the clinician, hidden as they are by their housings of hard tooth structure or alveolar bone and their gingival/mucosal coverings. Consequently, surrogate measures must be employed to evaluate the presence or absence of the disease process and its resolution. The evaluation process is further complicated by the lack of direct correlation between measures of the disease process and its clinical manifestation.

WHAT ARE SURROGATE OUTCOME MEASURES?

Signs of acute inflammation are classically described in the "triple response" exhibited by mechanically injured skin, which includes altered color (redness), texture/contour (swelling), and sensation (pain), which are directly accessible and viewable. These changes have a direct correlation with histopathologic and molecular alterations. Chronic inflammation does not necessarily exhibit the same highly visible signs and symptoms of its histopathologic character. When hidden from view, as are pulpal and periradicular tissues, the task of recognizing the presence or absence of chronic inflammation is even more challenging. The clinician is therefore left with indirect or associated (proxy) changes by which to judge the presence or absence of disease; these are called "surrogate" measures. Some of these may have to be inferred through associations, some may be directly visualized, and others are only indirectly "viewable" through various imaging techniques (such as radiography). The process therefore calls upon the clinician to assimilate various sources of information to form a judgment about the presence or absence of disease.

Types of Outcome Measures

In its broadest sense, an outcome measure for a treatment intervention may constitute any consistently anticipated and measurable consequence of the treatment. The prepared shape of the root canal system, bacterial load reduction, and technical quality of the root filling may all be regarded as outcome measures by this definition. But the ultimate *clinical* measure of a treatment outcome is assessing the prevention and resolution of disease.

The outcome of endodontic treatment may be assessed in four dimensions as in other medical disciplines.[9] The first dimension is physical/physiologic and related to presence or absence of pulpal/periapical health/disease, pain, and function. The second dimension assesses longevity or tooth survival. The third dimension relates to economics and assesses direct and indirect costs. Finally, the fourth dimension examines psychologic aspects involving perceptions of oral health–related quality of life (OHRQoL) and aesthetics.

WHAT IS THE PURPOSE OF EVALUATING OUTCOMES?

Apart from the importance of developing a solid foundation of evidence-based practice, it is important to evaluate treatment outcomes for a number of reasons.

Effectiveness of Procedures

First, treatment procedures must be effective. Otherwise, there is no reason to recommend them to patients as a treatment option. The patient must be properly informed as to the risks, benefits, and potential outcomes of the offered treatment. The availability of pooled outcome data and consensus guidelines offers both patients and endodontists reassurance and confidence in the validity and predictability of the offered procedure. However, the pooled average outcome data may not pertain if the clinician does not have the requisite experience, skill, and personal outcomes matching at least these average figures of performance. Personal outcome data offer the patient more precise measures for comparison and expectation. It also aids practitioners in refining their technique and knowledge to further improve their outcomes and, ultimately, to help improve the overall pooled data.[34] The treating clinician must assess his or her own personal outcome data and personal expectation of success. If the clinician is uncertain of a result or feels that the outcome could be enhanced by treatment from another clinician, then referral to someone else more qualified is paramount.

Factors Affecting Outcomes

Pooling data (that is preferably homogeneous) offers the potential to evaluate and prioritize the factors that exert a dominant influence on outcomes. In this way, protocols for treatment may be improved in a progressive fashion, bringing about the perfect meld of technical, clinical, and biologic insight necessary for the highest performance and most predictable outcome. Evaluation of factors affecting treatment outcomes arguably provides the strongest influence for making

changes that will improve the clinical outcomes. Weighing the relative importance of individual factors should help to identify the key biologic factors at play and how best to manage them from a clinical or technical perspective.

Value for Prognostication

Prognostication, which could be defined as the prediction, projection, prophesizing, or foretelling the likely outcome of treatment, is not often well defined in endodontics. The overall prognosis of a tooth depends on the interaction among three individual and often independent variables, including endodontic, periodontal, and restorative prognoses. Each has a set of subsidiary factors that must be considered to derive an overall prognosis. Finally, the tooth in question must be considered from a strategic perspective, relative to its position in the dental arch and the contribution it makes in the dynamic occlusion.

A deeper analysis of the factors affecting outcomes as prognostic indicators is merited in order to clarify the degree of complexity inherent in the problem. A comparison may be made with the randomized controlled trial of a drug as a treatment intervention. The drug therapy is delivered as a clearly prescribed and standardized dose regimen delivered by specified timings to effect an anticipated blood or target tissue concentration for a specified duration. Data recording is confined to compliance with the prescription and possibly checking of actual blood levels achieved as well as the final outcome effect.

In stark contrast, a surgical intervention, regardless of any standardization of the described procedural protocol, is subject to enormous variation in its delivery dependent on interpretation and execution of the protocol by each operator. To add to the levels of complexity and variation, surgical protocols are often multistep, sequential procedures, each prospective step dependent on the previous for its efficacy. Even characterizing and accurately recording any variations in treatment protocols becomes a challenge because many aspects and steps must be documented. This leads to the next challenge, that of demonstrating compliance with the accuracy of such complex data gathering. Furthermore, it becomes important from an analytic perspective to not only consider the effect of individual steps (factors) alone but also any interactive effect between the multiple steps inherent in the procedure. Even when such comprehensive prospective outcome data are available, utilizing these data to individually calculate the prognosis in a particular scenario may be challenging. Clinically, there may be two distinct ways to estimate prognosis: (1) to apply heuristic principles and intuitively gauge the effect of dominant factors or, at the other end of the extreme, (2) to mathematically input comprehensive data into an algorithmic model to calculate the estimated outcome. An even more sophisticated approach may be to use mathematical modeling to calculate iterations on variations within the system. At present, the evidence base is insufficient to allow such a sophisticated approach.

It therefore follows that evaluating outcomes and the factors that affect them are fundamental to the creation of a suitable data foundation for future application to endodontic prognostication. The endodontic treatment option is in competition with other alternative therapies and therefore, for the discipline to survive and thrive, a suitable blend of efficacy, efficiency, utility, predictability, and cost-effectiveness data must be generated for its principal therapies. Achievement of such a goal will require a diligent and conscientious collection of data from many sources. Analyses of such powerful datasets will ultimately yield proper insight into outcomes of the procedures we use. Coupled with biologic insight of the problem, it will become possible to derive new therapeutic solutions necessary to deliver them.

OUTCOME MEASURES FOR ENDODONTIC TREATMENT

Before determining the success or failure of an endodontic procedure, the parameters as to what is considered a success or a failure have to be discussed. These are termed the *outcome measures* of the procedures.

Outcome Measures for Vital Pulp Therapy Procedures

The techniques used to maintain the vitality and health of pulps in teeth with extensive caries or those with traumatic/mechanical exposures include (1) indirect pulp capping with one-step or stepwise caries excavation; (2) direct pulp capping of exposed pulps; and (3) partial/full pulpotomy procedures for more extensively involved pulps. The surrogate outcome measures adopted in studies include (1) clinical success (pulp sensitivity to cold test and absence of pain, soft-tissue swelling, sinus tract, periradicular radiolucency, or pathologic root resorption), (2) patient satisfaction, (3) adverse events (pain, swelling, tooth fracture), and (4) tooth extraction.[89,136] Although not providing a specific follow-up strategy, the quality guidelines of the European Society of Endodontology[61] suggests "initial review at no longer than 6 months and thereafter at further regular intervals." They also provide the criteria for judging the favorable outcome of vital pulp therapy (Table 11-1). There are substantial differences in the criteria adopted

TABLE 11-1

Criteria for Assessing the Outcome of Pulp Therapy

Quality Guidelines for Endodontic Treatment: Consensus Report of the European Society of Endodontology (2006)	Guidelines on Pulp Therapy for Primary and Immature Permanent Teeth (The American Academy of Paediatric Dentistry 2014)
1. Normal response to pulp sensitivity tests (when feasible) 2. Absence of pain and other symptoms 3. Radiologic evidence of dentinal bridge formation 4. Radiologic evidence of continued root formation in immature teeth 5. Absence of clinical and radiographic signs of internal root resorption and apical periodontitis	1. Tooth vitality maintained 2. Absence of posttreatment signs or symptoms such as sensitivity, pain, or swelling 3. Occurrence of pulp healing and reparative dentin formation 4. Absence of radiographic evidence of internal or external root resorption, periapical radiolucency, abnormal calcification, or other pathologic changes 5. Teeth with immature roots should show continued root development and apexogenesis

in published clinical studies on outcomes of vital pulp therapy[89,136] with few studies adopting all of the criteria. The frequency of radiologic review adopted in published studies also varies substantially, with some recommending the first review at 1 month followed by subsequent 3 monthly reviews.[172,242] The relative benefit of acquired information versus unnecessary radiation to patients has been questioned.[66] An initial assessment at 6 to 12 weeks, followed by a review 6 and 12 months after treatment, seems to have been accepted and is recommended. Some studies reported up to 10 years follow-up on case series demonstrating lower success rates, inferring the possibility of slow spread of pulpal inflammation and late failures. The conclusion is that longer follow-up periods are merited.

The process at each review consists of obtaining a history of symptoms, coupled with an examination to determine the presence or absence of tenderness to palpation of adjacent soft tissues, tenderness to pressure and percussion of the tooth, signs of radiographic pulpal, and periapical changes, and responses to pulp tests. The accuracy of pulp tests may be limited in pulpotomized teeth because of the distance of the remaining pulp tissue from the tooth's surface. In the case of pulp capping and pulpotomy, additional tests include radiographic verification of the presence of the calcific barrier (Fig. 11-1) and its integrity by removal of the dressing and direct probing. Although an initial examination at 6 weeks has been suggested, this can be modified for the radiographic assessment. If there is no evidence of complete bridge formation, the treatment is considered failed and root canal treatment should be considered. In addition, in the case of incompletely formed roots there should be radiographic evidence of continued root development (Fig. 11-2).

Outcome Measures for Nonsurgical Root Canal Treatment and Retreatment

Root canal treatment may be employed either to prevent or resolve periapical disease. Given that periapical lesions develop as a result of *interaction* between bacteria (and their by-products) and the host defenses, it is clear that prevention or resolution of the disease process depends on preventing or terminating this interaction.

Prevention of apical periodontitis applies to the clinical situation where it is judged that the pulp is irreversibly inflamed, necrotic, or infected to the extent that vital pulp therapy would not resolve the problem, which therefore requires pulpectomy. The implication is that the procedure demands asepsis as a prime requirement. The precise technical details of the protocol may not be so important, as long as they are focused on effective and aseptic removal of the pulp tissue. This is validated by the high chance of retaining periapical health (judged by conventional radiography), regardless of the clinical protocol used.[153]

Once the periapical lesion has become established, the challenge is a different one because the purpose now is to remove the bacterial biofilm and effect switching-off the periapical host response. The challenge seems to be greater still if the periapical lesion is larger as it is associated with a more diverse infection. A number of approaches (protocols) have been used to achieve this general aim. The periapical healing process that occurs after root canal treatment is less clear. Nevertheless,

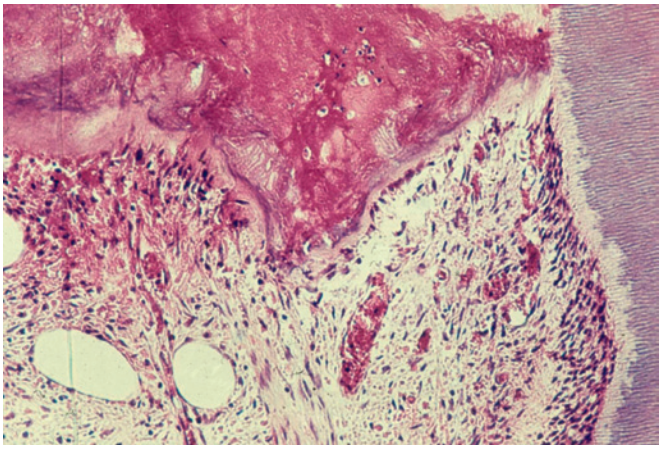


FIG. 11-1 Histologic view of intact calcific bridge formation following pulpotomy.

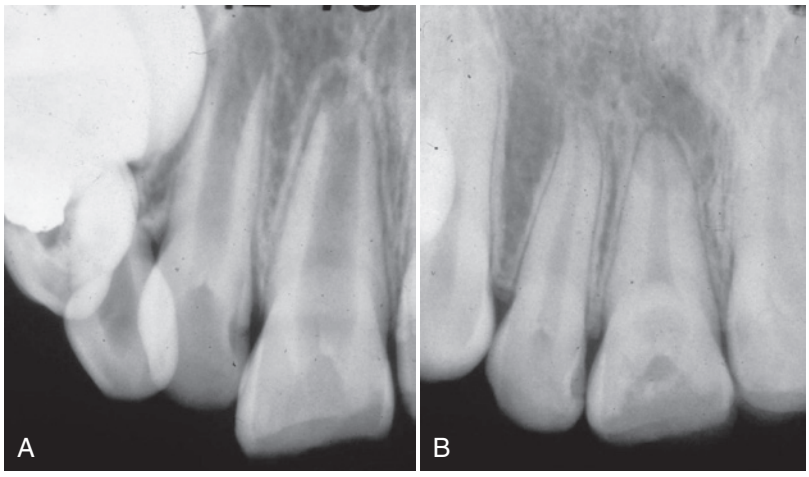


FIG. 11-2 Immature maxillary right lateral and central incisors (**A**) showing continued root development following pulpotomies (**B**).

ideal healing would eventually result in regeneration and the formation of cementum over the apical termini, isolating the root canal system from the periapex (Fig. 11-3); but this is not an inevitable result. Incomplete removal of the infection will reduce but not eliminate the periradicular inflammatory reaction, and in fact this is generally what happens.[147] This implies that residual infection in the apical anatomy is typical following completion of root canal treatment and that an ongoing interaction continues between the residual infection, root filling material, and host defenses, which plays a definitive role in determining the final healing outcome.

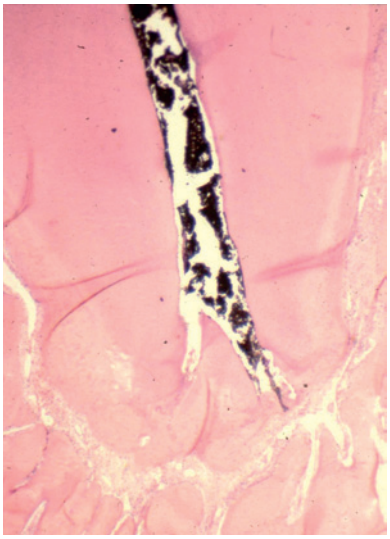


FIG. 11-3 Low-power view of healing by cementum formation when Sealpex is used (black particles are residual Sealapex and root filling material). (Courtesy Prof. M. Tagger.)

The culture test has been used during root canal treatment as an interim measure of the efficacy of the chemomechanical procedure; however, it has fallen out of favor in contemporary practice for a variety of reasons.[139] The outcome measures that quantitate healing subsequent to root canal treatment are the absence of clinical signs and symptoms of persistent periapical disease.[17,18] The definitive outcome measure (in conjunction with the absence of signs and symptoms), however, is periapical healing, because the treatment is aimed at resolution of periapical disease (Fig. 11-4).[152] Clinical judgment of the outcome of treatment is based on the absence of signs of infection and inflammation, such as pain, tenderness to pressure/percussion of the tooth, tenderness to palpation of the related soft tissues, absence of swelling and sinus tract, and radiographic demonstration of reduction in the size of the periapical lesion (if sufficient time has lapsed), with a completely normal development of the periodontal ligament space. Although the majority of periapical lesions heal within 1 year, healing may continue for up to 4 years or longer.[229]

Absence of signs and symptoms of periapical disease with radiographic evidence of a persistent periapical radiolucency may indicate either fibrous repair (Fig. 11-5) or persistent chronic inflammation or infection. Only time and acute exacerbation would identify the latter, whereas the former should remain asymptomatic.

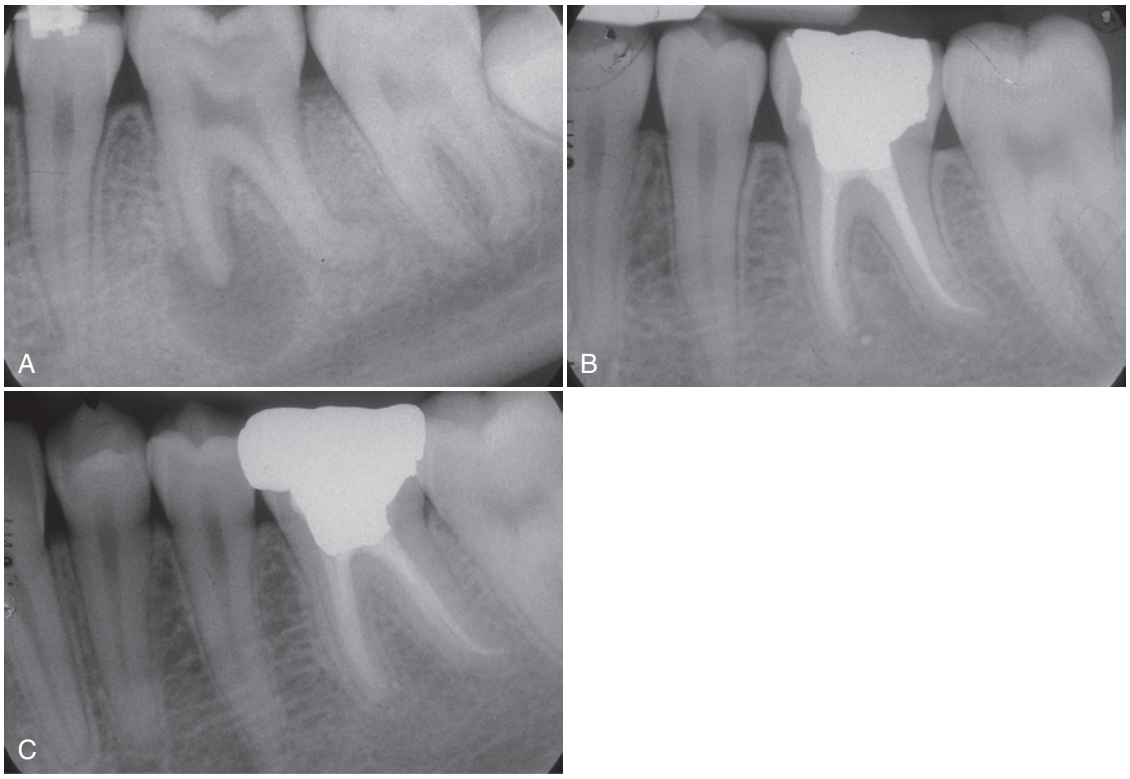


FIG. 11-4 Resolution of periapical disease after root canal treatment of the mandibular left molar with preoperative periapical radiolucency associated with mesial and distal roots (**A**), delayed healing caused by extruded filling material from the distal canal (**B**), and nearing complete healing as extruded material is resorbed (**C**).

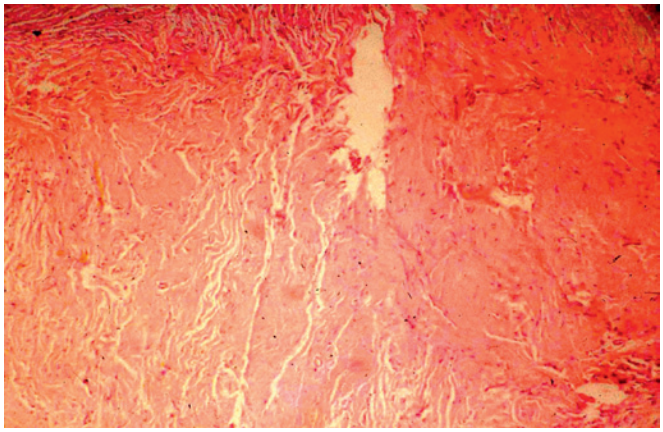


FIG. 11-5 Histologic view of fibrous periapical healing.

Longevity measures include survival of the root canal fillings or treatment[118,228,236] and tooth retention or survival.[111,149,190] The term *functional retention* was coined by Friedman in 2004[69] to mean retention of the tooth in the absence of signs and symptoms regardless of the radiographic presence of a lesion. The term *functionality* should further and more specifically cover the functional utility of the tooth—that is, some patients may complain that despite the absence of specific signs and symptoms of infection or inflammation, they find it impossible to use the tooth because it "feels" weak.

Modern definitions of health embrace a broader spectrum of measures including psychological well-being.[116] Instruments to assess these aspects have been developed in general medicine and adapted for application to dentistry, such as the Oral Health Related Quality of Life instrument.[55,87,115] There is no definitive instrument for measuring these aspects in endodontics as yet. Studies published so far have mainly adapted versions of the Oral Health Impact Profile (OHIP).

The periapical status of root-treated teeth has traditionally been assessed using two-dimensional conventional radiographic imaging. The use and limitations of two-dimensional radiographic images for assessing treatment outcome has been reviewed thoroughly and reported to suffer from low sensitivity, particularly in the posterior parts of the mouth.[98] The development of digital imaging technology[80] brought the possibility of image manipulation, including digital subtraction,[50,158,243,259] densitometric analysis,[156] correction of gray values,[32] and the manipulation of brightness and contrast.[75] However, none of these approaches has addressed the main deficiencies of accurately elucidating and quantitating the actual existence or progression of periapical bone loss.

Cone-beam computed tomography (CBCT), a new three-dimensional imaging technique requiring only 8% of the effective dose of conventional computed tomography,[119] has been proposed as a means of overcoming the problem of superimposition of tissue layers and structures. The diagnostic values of periapical radiography and cone-beam computed tomography were compared on dogs' teeth using histology as the comparative method of determining apical periodontitis 180 days after root canal treatment.[44] CBCT was found to be significantly more accurate in detecting minor bone lesions compared with two-dimensional radiology.[44,174] Other evidence to support the superior sensitivity of CBCT is available from studies using a variety of simulated defects created in bone in pig[226] or human[165,221] jaws. Although the validity of clinical outcome data derived using conventional radiographic techniques has been questioned,[256] the *routine use* of CBCT is not recommended[62,91,197] owing to its higher radiation dosage.[5,91] The use of CBCT, which may be more sensitive for detection of periapical healing,[114,167] may give lower healing rates and longer durations to complete healing.

Many studies consider the treatment successful only when both radiographic and clinical criteria are satisfied.[69] It has been documented that a small proportion of cases present with persistent symptoms despite complete radiographic resolution of the periapical lesion.[176] Comparison of success rates estimated with or without clinical examination revealed no[93] or only a very small difference (1%).[150]

The widely accepted definition for endodontic success and failure by Strindberg[229] embraces both radiographic and clinical findings (Table 11-2). Friedman and Mor[69] preferred the terms *healed, healing,* and *diseased* instead of *success* and *failure* because of the potential of the latter to confuse patients. The "healed" category corresponds to "success" as defined by Strindberg,[229] whereas "healing" corresponds to "success" as defined by Bender and colleagues[17,18] (see Table 11-2).

The long duration of the periapical healing process coupled with the reduced recall rates at longer follow-up periods has provided opportunity for setting the thresholds of success at either complete healing or partial healing (reduced lesion size). The success criteria used for complete resolution have been described either as "strict"[152] or "stringent,"[69] whereas the criteria established for reduction in periapical radiolucency size has been described as either "loose"[152] or "lenient."[69] The frequency of adoption of these two thresholds has been similar in previous studies; the expected success rates using "strict" criteria would be expected to be lower than those based on "loose" criteria. The literature finds the difference to vary from 4% to 48%.[153]

A periapical index (PAI) consisting of five points on the scale was devised for measuring the periapical status and used in some studies.[157,159] However, the studies only reported the increase or decrease in mean scores for the clinical factors under investigation coupled with the proportion of successful cases. This approach precluded direct comparison of their data with studies reporting conventional dichotomized outcomes. Other studies using the index dichotomized the scores into "healthy" (PAI 1 or 2) or "diseased" (PAI 3 to 5) categories,[160] thus allowing the data to be compared directly with the more traditionally used binary outcomes of success or failure. In this system of designation, given that the PAI score 2 represents periodontal ligament widening, it effectively signals adoption of the "loose" threshold. The longitudinal follow-up[86] of 14 cases presenting with widened apical periodontal ligament (PAI score at 2) for 10 years revealed unfavorable future healing in only a small proportion of the cases (28%, 4/14).

Outcome Measures for Periapical Surgery

Nonsurgical root canal treatment alone may fail to resolve apical periodontitis in a small proportion of cases because it may not allow access to the infection or resolution of the cause of the infection (i.e., root fracture or crestal bone

TABLE 11-2

Criteria for Determination of Periapical Status

Strindberg (1956)	Bender et al. (1966, a and b)	Friedman & Mor (2004)
Success	**Success**	**Healed**
Clinical: No symptoms *Radiographic:* The contours, width, and structure of the periodontal margin were normal OR The periodontal contours were widened mainly around the excess filling	*Clinical:* Absence of pain/swelling; disappearance of fistula; no loss of function; no evidence of tissue destruction *Radiographic:* An eliminated or arrested area of rarefaction after a posttreatment interval of 6 months to 2 years	*Clinical:* Normal presentation *Radiographic:* Normal presentation
Failure		**Diseased**
Clinical: Presence of symptoms *Radiographic:* A decrease in the periradicular rarefaction OR Unchanged periradicular rarefaction OR An appearance of new rarefaction or an increase in the initial rarefaction		Radiolucency has emerged or persisted without change, even when the clinical presentation is normal OR Clinical signs or symptoms are present, even if the radiographic presentation is normal
Uncertain		**Healing**
Radiographic: There were ambiguous or technically unsatisfactory control radiographs that could not, for some reason, be repeated OR The tooth was extracted prior to the 3-year follow-up owing to the unsuccessful treatment of another root of the tooth		*Clinical:* Normal presentation *Radiographic:* Reduced radiolucency

communication to the periapical infection). Where the cause is microbial persistence, the infection may be localized intraradicularly (in the apical canal anatomy [Fig. 11-6] or in the apical dentinal tubules [Fig. 11-7] or extraradicularly [Fig. 11-8]). In these instances, a surgical approach to the periapex may be required in addition to the nonsurgical approach to removing such microbes (see Fig. 11-7).

The success of periapical surgery has been assessed with the same clinical and radiographic criteria as for nonsurgical root canal treatment. However, the radiographic criteria for successful periapical healing are different from those for nonsurgical root canal treatment[140,186] (Table 11-3; Figs. 11-9, 11-10, and 11-11). Moreover, periodontal attachment loss in the form of marginal gingival recession is an additional criterion for measuring the outcome of periapical surgery.

Now that the parameters have been discussed as to the variables that need to be considered when assessing healing or nonhealing outcomes, the following discussion will describe the expected outcomes of the various endodontic procedures.

OUTCOMES OF VITAL PULP THERAPY PROCEDURES

The vital pulp therapy procedures discussed in this chapter include those for managing extensive caries with a high risk of pulpal exposure or pulps exposed through caries or traumatic injuries.

Indirect Pulp Capping (One-Step versus Stepwise Excavation)

The most conservative management for extensive caries is indirect pulp capping using either the one-step or the stepwise excavation approach. The stepwise excavation approach with initial partial caries removal was advocated to reduce the risk of pulpal exposure and is generally considered to be associated with poorer outcomes. The clinical outcomes of the one-step or stepwise approaches have been compared in three randomized controlled trials on permanent teeth with deep caries.[19,113,121,122] Stepwise excavation was found to be associated with a lower rate of pulp exposure and higher chance of long-term clinical success than the one-step excavation approach[19,113] (Table 11-4). Maltz and colleagues,[121,122] however, found the contrary, and they attributed the low long-term clinical success rate of the stepwise approach to patients being noncompliant in returning for scheduled completion of treatment. Interestingly, comparison of the cost-effectiveness of the one-step versus the stepwise excavation techniques using health-economic modeling involving simulated treatment of a molar tooth with deep caries in a 15-year-old patient revealed the one-step procedure to accrue lower long-term costs as well as longer tooth retention and pulp vitality.[200]

Meta-analyses of data from studies[19,20,65,81,89,113,120-122,141] listed in Table 11-4 revealed that the weighted pooled success rate for indirect pulp capping using the one-step approach (81.7%; 95% confidence interval [CI]: 72.7%, 90.6%)

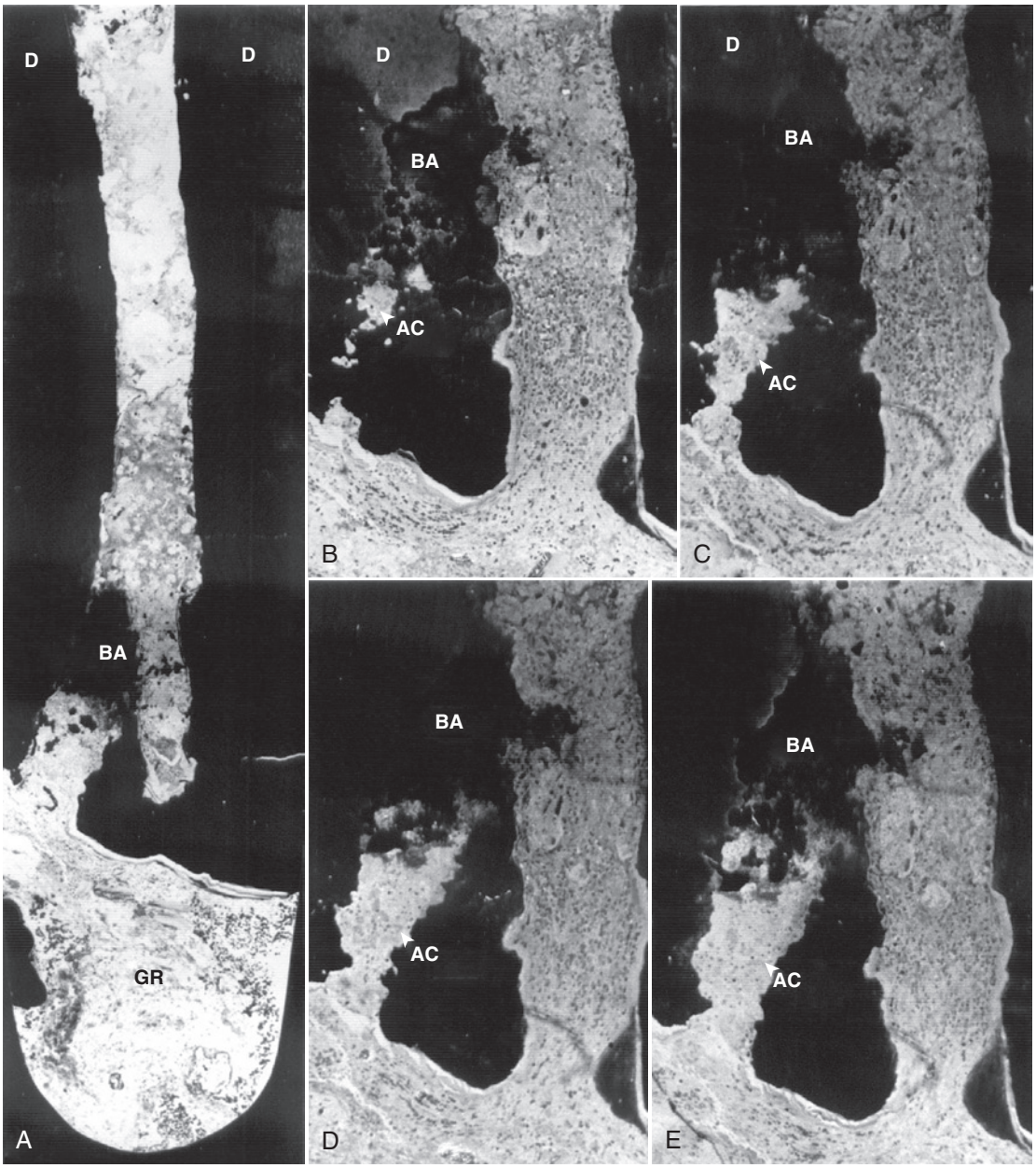


FIG. 11-6 Axial sections through the surgically removed apical portion of the root with a therapy-resistant periapical lesion (GR). Note the cluster of bacteria (BA) visible in the root canal. Parts **B–E** show serial semithin sections taken at varying distances from the section plane of **(A)** to reveal the emerging **(B)** and gradually widening **(C–E)** profiles of an accessory root canal (AC). Note that the accessory canal is clogged with bacteria (BA). Original magnification: **A** ×52; **B–E** ×62. (From Nair PN, Sjogren U, Krey G, Sundqvist G: Therapy-resistant foreign body giant cell granuloma at the periapex of a root-filled human tooth, *J Endod* 16:589, 1990.)

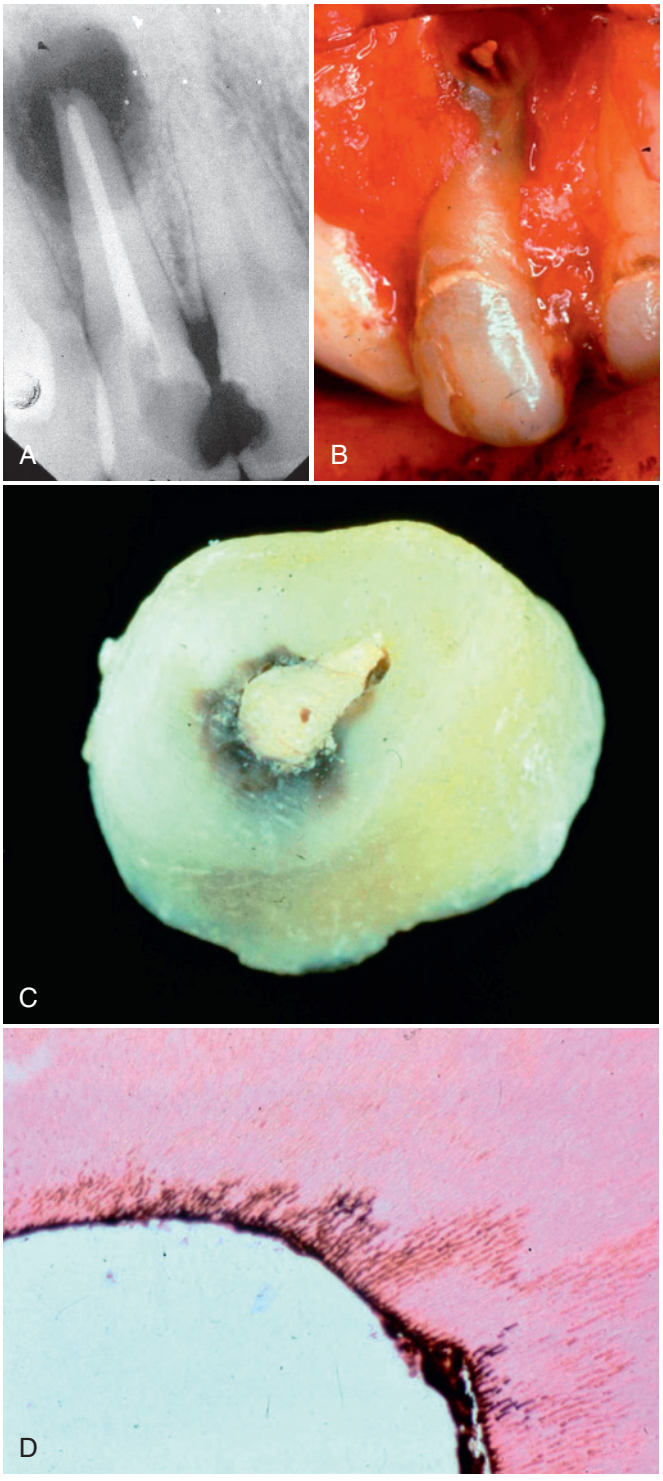


FIG. 11-7 A, Adequate root filling demonstrated on radiograph. B, View of the periapical surgery and root resection of the tooth shown in (A) shows stained root dentine. C, Resected root showing stained/infected dentine. D, Histologic view of the root end shown in (C) showing infected dentinal tubules (S).

TABLE 11-3

Classification of Periapical Healing Following Apical Surgery

Rud et al. (1972) and Molven et al. (1987)
Complete Healing
Reformation of a Periodontal Space with
Normal width and lamina dura to be followed around the apex
Slight increase in width of apical periodontal space but less than twice the width of noninvolved parts of the root
Tiny defect in the lamina dura (maximum 1 mm) adjacent to the root filling
Complete Bone Repair with
Bone bordering the apical area does not have the same density as surrounding noninvolved bone
No apical periodontal space can be discerned
Incomplete Healing (Scar Tissue)
The Rarefaction Has Decreased in Size or Remained Stationary with
Bone structure recognized within the rarefaction
The irregular periphery of the rarefaction and demarcation by a compact bone border
The rarefaction located asymmetrically around the apex
Angular connection between the rarefaction and the periodontal space
An Isolated Scar Tissue in the Bone with Findings Above
Uncertain Healing
The Rarefaction Has Decreased in Size with
The size larger than twice the width of the periodontal space
Lamina-dura like bone structures around the border
A circular or semicircular periphery
Symmetric location around the apex as a funnel-shaped extension of the periodontal space
Bone structure discernible within the bony cavity
A collar-shaped increase in width of lamina dura coronal to the radiolucency
Unsatisfactory Healing (Failures)
The rarefaction has enlarged or is unchanged
If a case still demonstrated "uncertain healing" 4 years postoperatively, the treatment should be considered a failure[186]

(Fig. 11-12) was similar to that for the stepwise approach (81.9%; 95% CI: 72.1%, 91.7%) (Fig. 11-13).

Calcium hydroxide cement was the preferred lining material for the pulpal surface, whereas zinc oxide–eugenol–based cement was the preferred base material in most studies. Recently, resin-modified-glass-ionomer liner has also been used, but the type of lining material did not influence the outcome (see Table 11-4). The age of patients, presence of preoperative pain, and pulpal exposure during excavation were significant negative prognostic factors.[19]

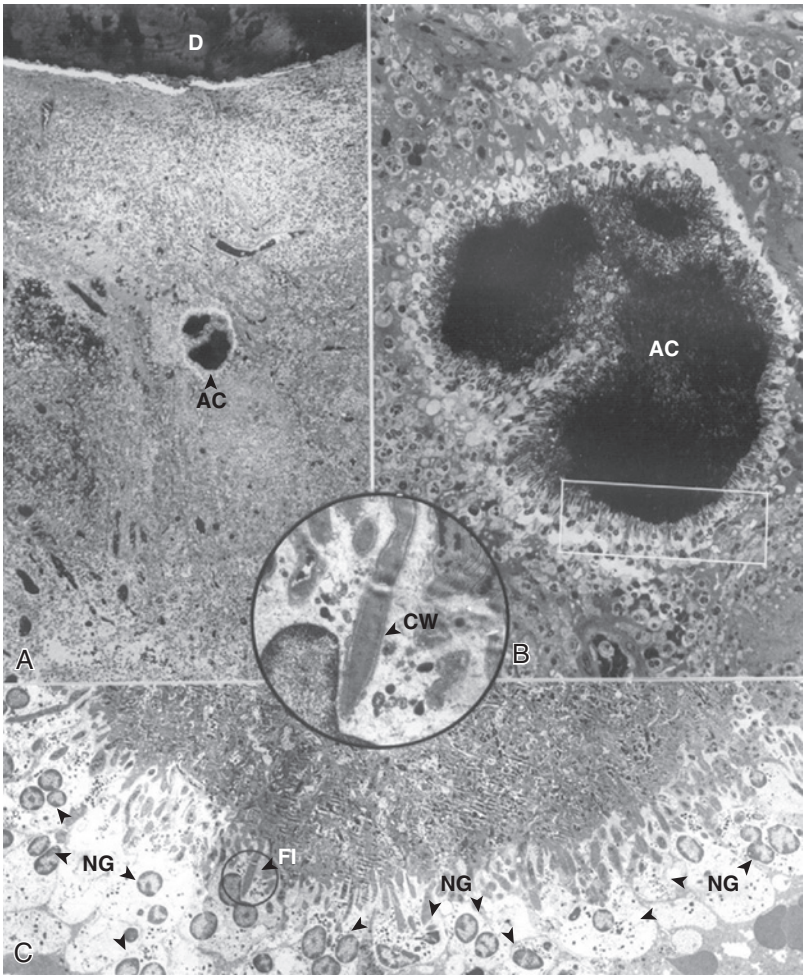


FIG. 11-8 Actinomyces in the body of a human periapical granuloma. The colony (AC in B) is magnified in B. The rectangular area demarcated in B is magnified in C. Note the starburst appearance of the colony with needle-like peripheral filaments surrounded by few layers of neutrophilic granulocytes (NG), some of which contain phagocytosed bacteria. A dividing peripheral filament (Fl) is magnified in the inset. Note the typical Gram-positive wall (CW): D = dentine. Original magnification: A ×60; B ×430; C ×1680; inset ×6700. (From Nair PR, Schroeder H: Periapical actinomycosis, *J Endod* 10:567, 1984.)

Direct Pulp Capping

Direct pulp capping is performed on teeth with pulp exposures caused by caries, caries excavation, or traumatic injuries. Most studies investigating the clinical outcome of direct pulp capping on permanent teeth had excluded teeth with signs and symptoms of irreversible pulpitis and apical pathosis (Table 11-5). Saline, sodium hypochlorite, and chlorhexidine have been reportedly used to irrigate the exposed pulp and to achieve hemostasis. Calcium hydroxide paste and mineral trioxide aggregate (MTA) were the commonly used capping materials (see Table 11-5).

A meta-analysis of data from studies* listed in Table 11-5 revealed the weighted pooled success rate to be 70.1% (95% CI: 59.9%, 80.2%) (Fig. 11-14). The patient's age and sex; tooth location and type; pulp exposure type, size, and its location; and the restoration type, size, and quality did not have a significant influence on success. Although the outcome of direct pulp capping of teeth with immature versus mature apices has not been systematically compared in individual studies, indirect comparison of pooled data from different studies[1] revealed that teeth with immature roots were associated with significantly more successful outcomes.

The type of capping material was another significant prognostic factor,[90,197,207] with MTA performing superiorly to calcium hydroxide in a randomized controlled trial[90] and in a systematic review.[1]

Pulpotomy

Earlier studies on the outcome of *partial pulpotomy* (specifying varying degrees of coronal pulp removal) had only included teeth with vital traumatic pulp exposures; however, more recent studies have also included teeth with carious pulp exposures. Teeth with spontaneous or severe pain and signs and

*Please see the following references: 12, 19, 21, 64, 65, 72, 88, 90, 92, 128, 132, 133, 193, 207, and 254.

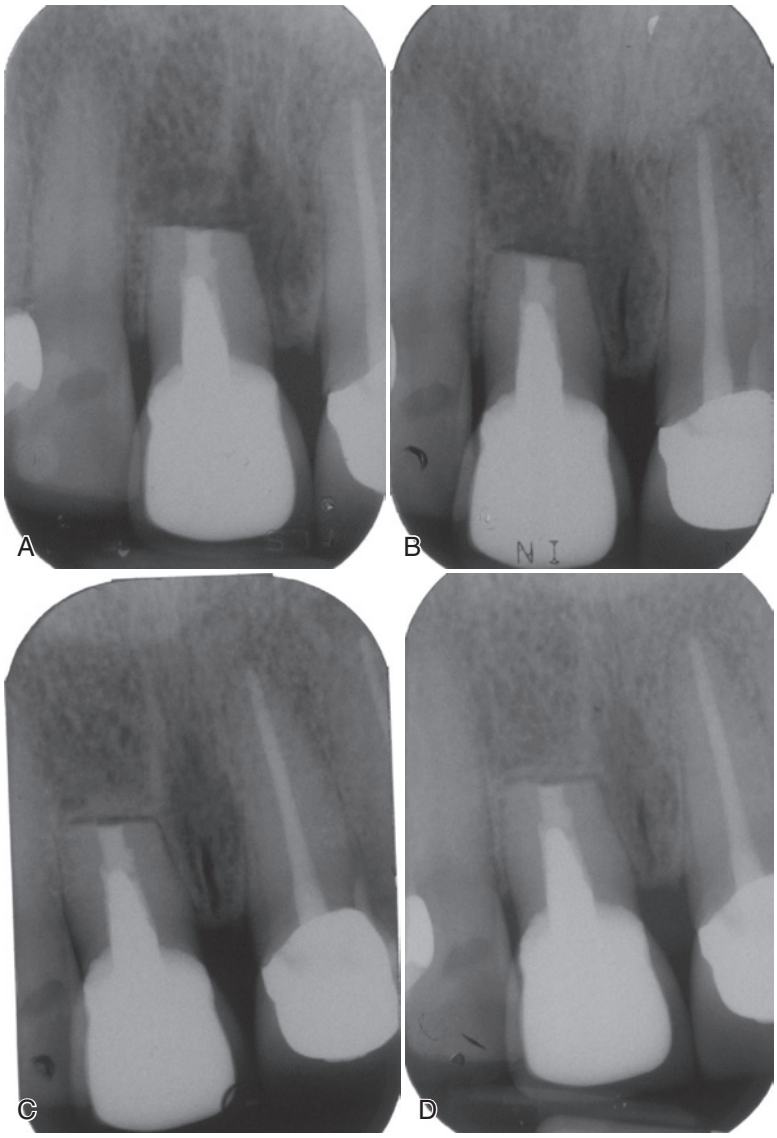


FIG. 11-9 A, A maxillary right central incisor having undergone periapical surgery. B, Incomplete periapical healing at 1 year postoperatively. Complete healing at 3 (C) and 4 years (D) postoperatively.

FIG. 11-10 Example of incomplete healing with isolated scar tissue.

symptoms of apical pathosis were excluded (Table 11-6). On the other hand, the outcomes of *full pulpotomies* (completely removing the coronal pulp and retaining the radicular pulp) had only been investigated in teeth with carious exposures (Table 11-7). Saline irrigation has been preferred for achieving hemostasis, whereas calcium hydroxide and more recently MTA were the preferred pulp capping materials (see Table 11-6).

A meta-analysis of data from studies listed in Tables 11-6[10,11,19,40,71,125,131,177] and 11-7[6,30,31,52,58,126,187,192,235,251,255] revealed the weighted pooled success rate to be 79.3% (95% CI: 66.7%, 91.8%) for partial pulpotomies (Fig. 11-15) and 82.4% (95% CI: 69.3%, 95.4%) for full pulpotomies (Fig. 11-16).

The effects of potential prognostic factors for pulpotomy have not been explored systematically except for the pulp capping material. Randomized controlled trials revealed that MTA achieved similar outcomes in partial[177] or full pulpotomies[58] when compared with calcium hydroxide.

Text continued on p. 495

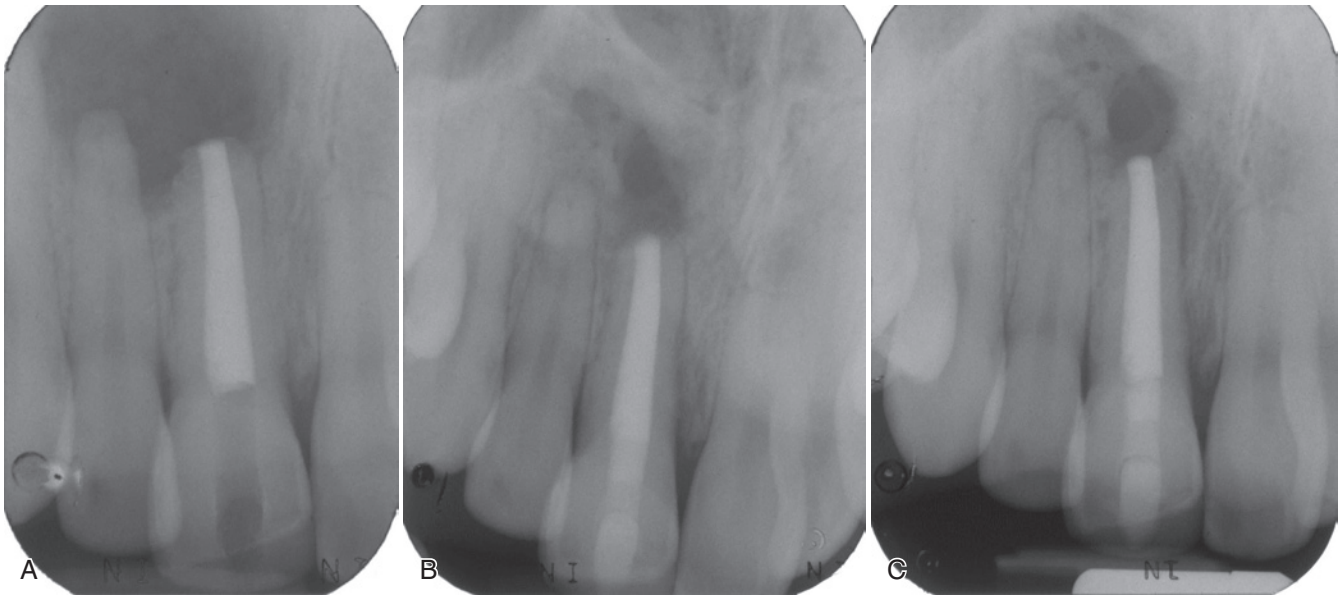


FIG. 11-11 Example of uncertain healing. Periapical radiographs of a maxillary left central incisor taken immediately after periapical surgery (A), at 2 years postoperatively (B), and at 3 years postoperatively (C).

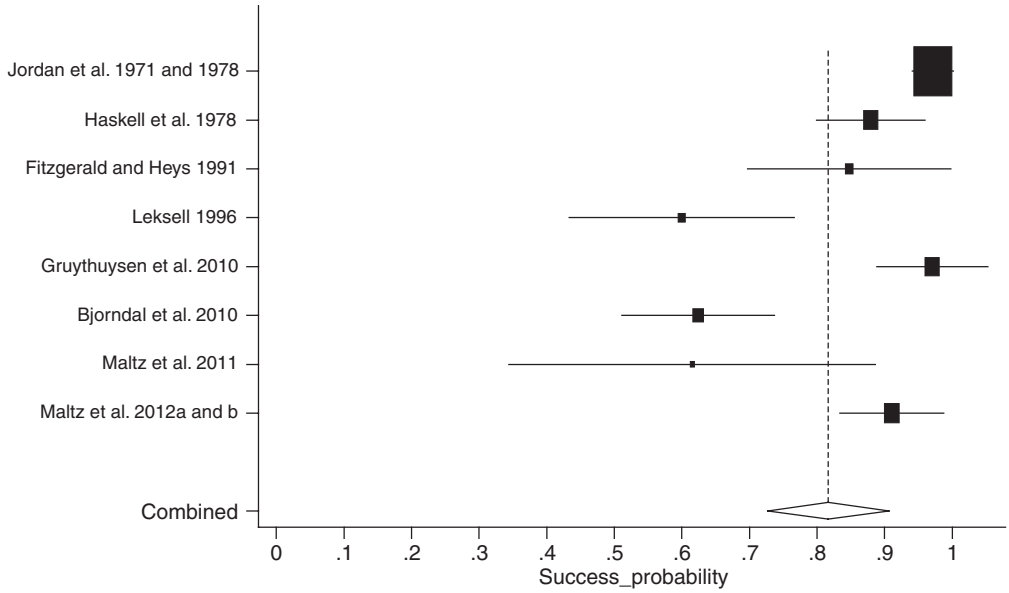


FIG. 11-12 Forest plot showing individual study and weighted pooled probabilities of success for one-step indirect pulp capping.

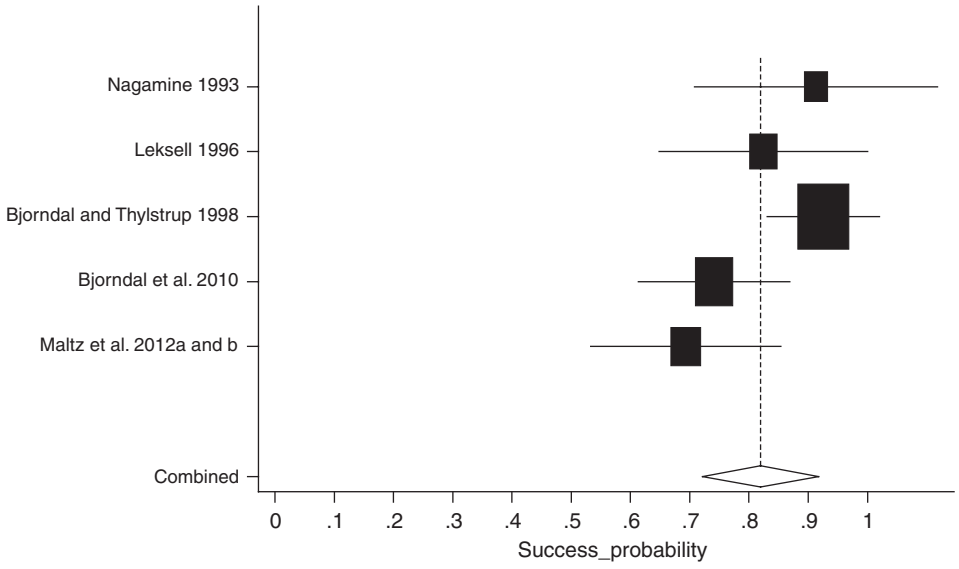


FIG. 11-13 Forest plot showing individual study and weighted pooled probabilities of success for stepwise indirect pulp capping.

TABLE 11-4

Studies Investigating the Clinical/Radiographic Outcome of Indirect Pulp Capping of Permanent Teeth

Study	Study Design	Type of Indirect Pulp Capping	No. of Teeth	Success (%)	Age	Pretreatment Status	Capping Material	Criteria for Success	Duration after Treatment	Notes
More, 1967	Case series	Stepwise	8	8 (100%)	11 to 18	Carious Vital Apical pathosis	1st visit: ZOE. 2nd visit: CH/Amal	Vital/ asymptomatic/ intact PDL	6 to 36 mo	
Jordan et al., 1971 (in Hayashi et al., 2011) and 1978	Case series	1-step	243	236 (97.1%)	8 to 37	Deep caries without pulpitis	CH, CH + cresatin, or ZOE/ZOE + amalgam	Vital/ asymptomatic/ intact PDL	Not given	24 teeth were followed up for 11 yr, 11 remained vital with no evidence of pathosis
Sawusch, 1982 (in Hayashi et al., 2011)	CCT	Stepwise	48	48 (100%)	14 or younger	Deep caries without pulpitis	CH (Dycal versus improved Dycal)/ ZOE or ZnPO4	Clinical	6 mo	
Fitzgerald & Heys, 1991	Case series	1-step	46	39 (84.8%)	20 to 60	Deep caries Vital	CH (Dycal versus Life)	Asymptomatic	1 yr	The brand of CH did not have significant effect
Nagamine, 1993 (in Hayashi et al., 2011)	CCT	Stepwise	23	21 (91.3%)	17 to 46	Deep caries without pulpitis	Polycarboxylate cement with tannin-fluoride versus hydraulic temporary sealing material/GIC	Vital	3 mo	
Leksell, 1996	RCT	Stepwise	57	47 (82.5%)	6 to 16	Not given	CH/ZOE then CH/GIC	Asymptomatic/ intact PDL	1 to 11 yr (mean = 43 mo)	47/57 without exposure; duration of CH dressing had no significant influence on exposure
		1-step	70	42 (60.0%)						42/70 without exposure

Bjorndal & Thylstrup, 1998	Case series	Stepwise	94	87 (92.6%)	Not given	Deep caries	CH/temporary filling	Absence of perforation, asymptomatic	1 yr	88/94 without exposure
Bjorndal et al., 2010	RCT	Stepwise 1-step	143 149	106 (74.1%) 93 (62.4%)	29 (25-38)	Deep caries Only provoked pain Vital	CH/GIC	Vital/intact PDL	1 yr	Stepwise better than complete; preoperative pain, older patients reduce success
Gruythuysen et al., 2010	Case series	1-step	34	33 (97.1%)	Up to 18	Carious (> 2/3) No spontaneous, persistent pain No apical pathosis	GIC	Survive/ asymptomatic/ intact PDL/no resorption	3 yr	
Maltz et al., 2011	Case series	1-step	26	16 (61.5%)		Vital Deep caries	CH/ZOE then composite	Vital	10 yr	
Maltz et al., 2012 (a and b)	RCT (multicenter)	1-step Stepwise	112 101	111 (18 mo) 102 (91.7%) (3 yr) 87 (18 mo) 70 (69.3%) (3 yr)	≥ 6	Vital Carious (> 1/2) No spontaneous, persistent pain No apical pathosis	GIC/amalgam or composite CH/ZOE then GIC/ amalgam or composite	Vital/intact PDL Vital/intact PDL	18 mo, 3 yr (clinical and radiographic outcome)	Indirect pulp capping was associated with significantly higher success rate than stepwise, regardless of duration after treatment; the low success rate of stepwise was attributed to patients not returning for completion of treatment

TABLE 11-5

Studies Investigating the Clinical/Radiographic Outcome of Direct Pulp Capping of Permanent Teeth

Study	Study Design	No. of Teeth	Success (%)	Age (year)	Pretreatment Status	Hemostasis	Capping Material	Base/ Restorative Material	Criteria for Success	Duration after Treatment	Notes
Weiss, 1966	Case series	160	141 (88.0%)	16 to 67	Not given	Not given	CH + cresatin	ZOE	Vital/ asymptomatic/ intact PDL	3 yr	
Shovelton et al., 1971	RCT	154 (1-step) 53 (2-step)	115 (74.7%) 33 (64.7%)	15 to 44	Carious or traumatic exposure Vital Asymptomatic Molar/premolar	Saline	Corticosteroid + antibiotic, glycerrhetinic acid + antibiotics, ZOE, or CH	ZOE/amalgam	Vital/ asymptomatic/ intact PDL	24 mo	There was no significant difference in success rates among the various capping materials
Haskell et al., 1978	Case series	133	117 (88.0%)	8 to 74	Carious exposure Asymptomatic	Not given	CH or penicillin	ZOE	Vital/ asymptomatic/ intact PDL	> 5 yr	5- to 22-year survival Success rate was not affected by age and tooth type
Gillien & Schuman, 1985	Case series	17	13 (76.4%)	6 to 9	Carious exposure	Not given	CH	Base material not given/ amalgam or crown	Asymptomatic/ intact PDL	6 to 12 mo	
Horsted et al., 1985	Case series	510	485 (95.1%)	Not given	Exposure during cavity preparation or caries removal No periapical pathosis No pain	2% chloramine or 0.2% CHX	CH	ZOE	Vital/ asymptomatic/ intact PDL	5 yr	Type of exposure and tooth type had no significant effects Older age had lower survival
Fitzgerald & Heys, 1991	Case series	8	6 (75.0%)	20 to 60	Vital Exposure during caries removal	Sterile cotton pellets	CH	$ZnPO_4$/ amalgam or composite	Asymptomatic	12 mo	The brand of setting CH paste did not have significant effect

Matsuo et al., 1996	Case series	44	36 (81.2%)	20 to 69	Carious exposure No intense pain	10% NaOCl and 3% H_2O_2	CH	ZOE/GIC	Vital/ asymptomatic/ intact PDL	3 yr	
Santucci, 1999	Case series	29	15 (51.7%)	Not given	Exposure due to caries, or its removal Sensitive to cold or sweat with no other pain No periapical pathosis	Not given	CH	Composite or cast gold restoration	Asymptomatic	4.5 yr	
Barthel et al., 2000	Case series	123	29 (23.6%)	10 to 70	Carious exposure	3% H_2O_2	CH/$ZnPO_4$ or other materials	Not given	Vital/ asymptomatic/ intact PDL	5 to 10 yr	Age, tooth type, site of exposure had no significant effects Immediate placement of permanent restoration had significantly higher success rate
Farsi et al., 2006	Case series	30	28 (93.3%)	9 to 12	Deep caries Potential exposure Reversible pulpitis Intact PDL	Saline	MTA	ZOE/composite	Vital/ asymptomatic/ intact PDL/ continuous root development	2 yr	
Bogen et al., 2008	Case series	49	48 (98.0%)	7 to 45	Carious exposure (.25 to 2.5 mm) Reversible pulpitis	5.25/6% NaOCl	MTA	Composite	Dentine Vital/ bridge/ asymptomatic/ root development/ intact PDL	1 to 9 yr	
Bjorndal et al., 2010	RCT	22	7 (31.8%)	25 to 38	Exposure during caries removal Only provoked pain Vital	Saline	CH	GIC	Vital/intact PDL	1 yr	

Continued

TABLE11-5

Studies Investigating the Clinical/Radiographic Outcome of Direct Pulp Capping of Permanent Teeth—cont'd

Study	Study Design	No. of Teeth	Success (%)	Age (year)	Pretreatment Status	Hemostasis	Capping Material	Base/ Restorative Material	Criteria for Success	Duration after Treatment	Notes
Mente et al., 2010	Case series	122	86 (70.5%)	8 to 78	Carious or mechanical exposure	0.12% CHX	MTA or CH	GIC/composite or crown	Vital/no clinical or radiographic evidence of apical pathosis	12 to 18 mo	Use of MTA for capping and immediate placement of permanent restoration had significantly higher success rate Age; sex; tooth location and type; exposure site and type; restoration type, size, and quality did not have significant effects
Miles et al., 2010	Case series	51	23 (45.1%)	21 to 85	Carious exposure	2.5% NaOCl	MTA	GIC/composite or amalgam	Vital/ asymptomatic/ intact PDL	12 to 27 mo	
Hilton et al., 2013	RCT	126 144	81 (64.3%) 116 (80.6%)	9 to 90 8 to 89	Carious, traumatic, mechanical exposure	5.25% NaOCl	CH MTA	GIC	Vital/intact PDL/ no resorption/ not requiring extraction or root canal treatment	2 yr	MTA was associated with significantly higher success rate than CH Patient, dentist, tooth, pulp exposure, and pulp capping characteristics did not have significant influence on the results

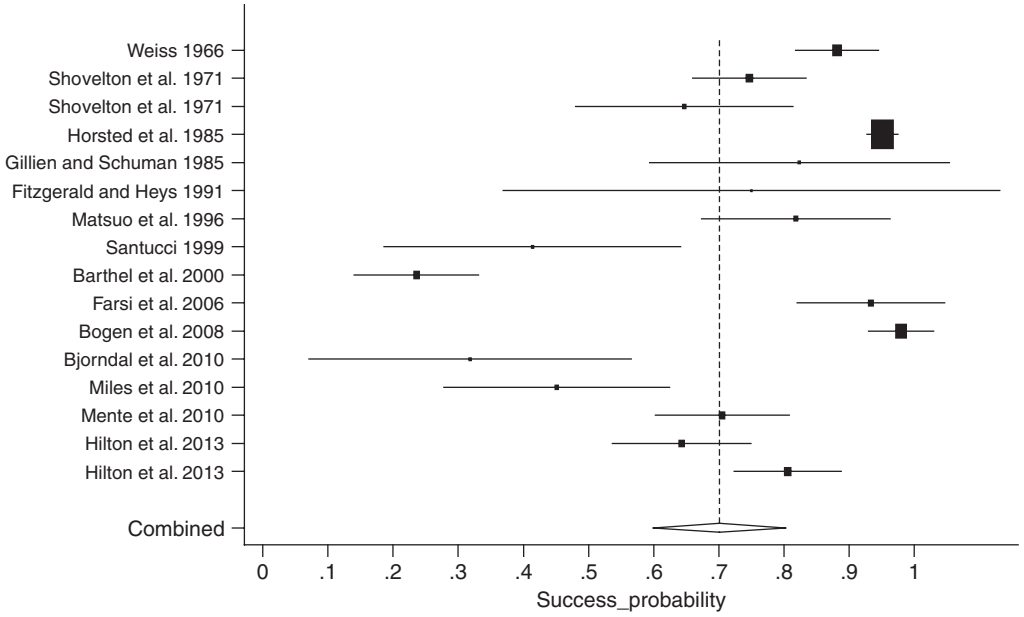


FIG. 11-14 Forest plot showing individual study and weighted pooled probabilities of success for direct pulp capping.

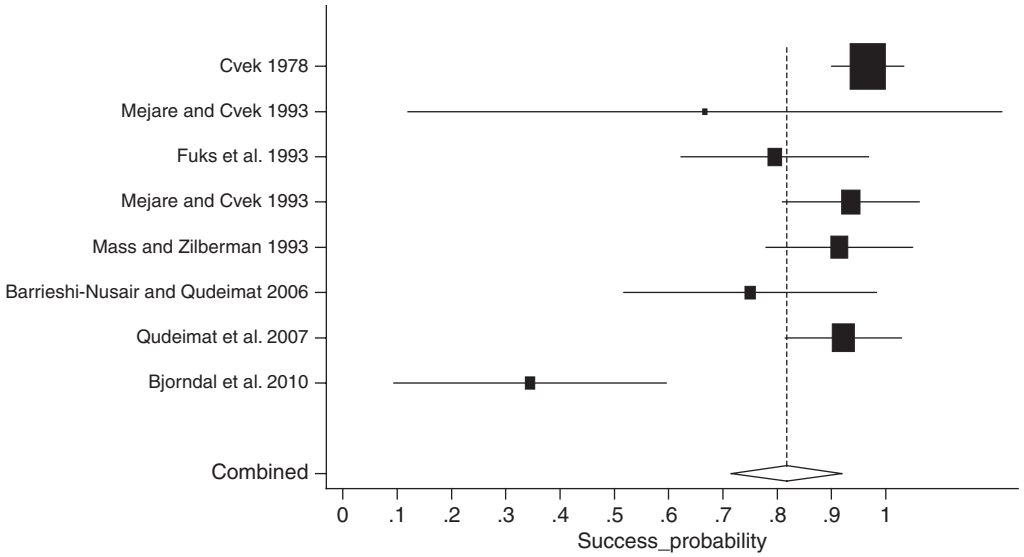


FIG. 11-15 Forest plot showing individual study and weighted pooled probabilities of success for partial pulpotomy.

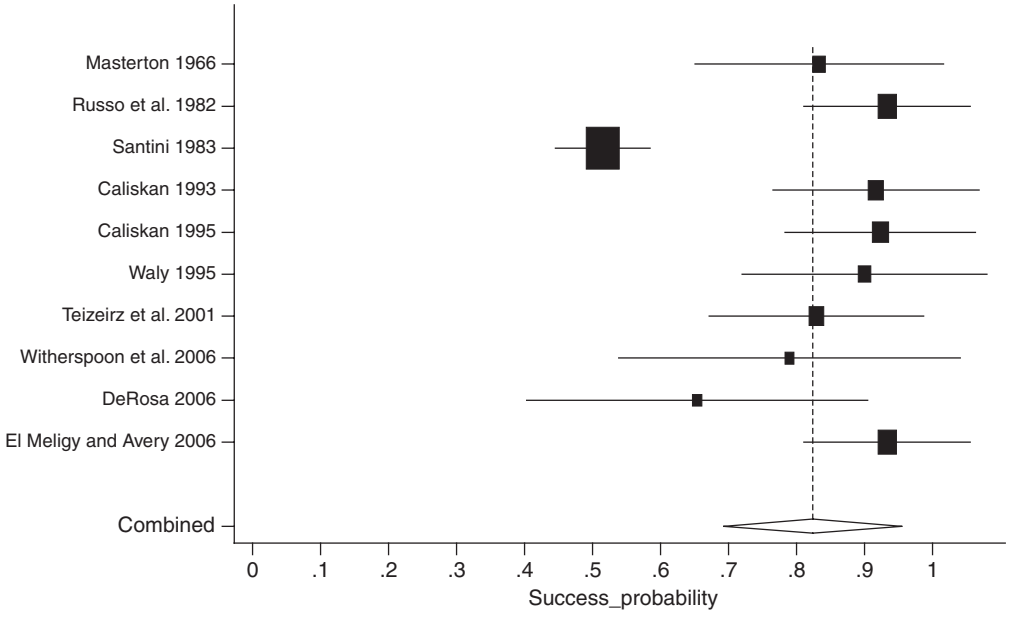


FIG. 11-16 Forest plot showing individual study and weighted pooled probabilities of success for full pulpotomy.

TABLE 11-6

Studies Investigating the Clinical/Radiographic Outcome of Partial Pulpotomy of Permanent Teeth

Author	No. of Teeth	Success (%)	Age	Pretreatment Status	Exposure Size	Hemostasis	Pulp Dressing Material	Base/ Restorative Material	Criteria for Success	Duration after Treatment	Notes
Cvek, 1978	60	58 (96.7%)	Not given	Traumatic exposure Vital Bleeding wound	0.5 to 4.0 mm	Saline	1st visit: nonsetting CH 2nd visit: setting CH	1st visit: ZOE 2nd visit: Composite	EPT/asymptomatic/ intact PDL/ continued root development/ hard tissue barrier	14 to 60 mo	Size and duration of exposure, root maturity did not affect outcome
Baratieri et al., 1989	26	26 (100%)	12 to 44	Exposure due to caries or caries removal Pulp tissue bleeding without signs of degeneration	Not given	Calcium hydroxide solution	CH powder then hard set CH cement	Zinc oxide cement	Asymptomatic/vital	1 to 2 yr	
Fuks et al., 1993	44	35 (79.5%)	Not given	Traumatic exposure Vital Bleeding wound	Not given	Saline	CH	ZOE	Asymptomatic/ bridge/root development/ vital	0.5 to 4 yr	
Mass & Zilberman, 1993	35	32 (91.4%)	7.5 to 25	Molar Deep caries Asymptomatic No apical pathosis	Less than 1 to 2 mm in diameter; 2 to 3 mm deep	Saline	CH	ZOE/ amalgam or crown	Asymptomatic/ intact PDL/root development	1 to 2 yr	

Mejare & Cvek, 1993	31 (2-step) 6 (1-step)	29 (93.5%) 4 (66.7%)	Not given	Carious exposure Asymptomatic No apical pathosis	N/A	Saline	CH	ZOE	Asymptomatic/ intact PDL/root development	24 to 140 mo	
Barrieshi-Nusair & Qudeimat, 2006	28	21 (75.0%)	7.2 to 13.1	Carious exposure Molar Reversible pulpitis No apical pathosis	2 to 4 mm deep	Saline	MTA	GIC/amalgam or crown	Vital/asymptomatic/ intact PCL/root development	1 to 2 yr	
Qudeimat et al., 2007*	23 28	21 (91.3%) 26 (92.9%)	6.8 to 13.3	Carious exposure Molar	2 to 4 mm deep		CH MTA	GIC/amalgam or crown	Absence of signs and symptoms/ intact PDL/root development	24.5 to 45.6 mo	There was no significant difference in outcome between CH and MTA
Bjorndal et al., 2010*	29	10 (34.5%)	25 to 38	Deep caries Only provoked pain Vital Expose during removal	Not given	Saline	CH	GIC	Vital/intact PDL	1 yr	

NB: All the studies were case series except those labeled with *.
Qudeimat et al. (2007) is a randomized controlled trial comparing calcium hydroxide versus MTA as pulp dressing material.
Bjørndal et al. (2010) is a randomized controlled trial comparing the outcomes of 1-step versus stepwise indirect pulp capping for teeth without exposure, and direct pulp capping versus partial pulpotomy for teeth with exposure during caries removal.

TABLE 11-7

Studies Investigating the Clinical/Radiographic Outcome of Full Pulpotomy of Permanent Teeth

Author	No. of Teeth	Success (%)	Age	Pretreatment Status	Hemostasis	Capping Material	Base/Restorative Material	Criteria for Success	Duration after Treatment	Notes
Masterson, 1966	30	25 (83.3%)	6 to 39	Not given	Not given	CH	Not given	Vital/asymptomatic	1 to 70 mo	
Russo et al., 1982	30	28 (93.3%)	9 to 28	Carious exposure No apical pathosis	Not given	CH	Not given	Intact PDL	8 w	
Santini, 1983	373	192 (51.4%)	Not given	Carious exposure or near exposure Symptomatic	Cotton wool pellet	CH or CH + Ledermix	ZOE	Vital/bridge/ asymptomatic	6 mo	Sex and medicament had no significant effect Poor healing was associated with age < 7.5
Caliskan, 1993	24	22 (91.7%)	10 to 22	Hyperplastic pulpitis	Saline	CH	ZOE/Amalgam or composite	Vital/asymptomatic/ dentine bridge/ intact PDL	1 to 4 yr	
Caliskan, 1995	26	24 (92.3%)	10 to 24	Carious exposure Asymptomatic Apical pathosis	Saline	CH	ZOE	Asymptomatic/bridge/ root development/ vital	16 to 72 mo	
Waly, 1995	20	18 (90.0%)		Carious exposure Molar		CH-glutaraldehyde CH	Not given	Not given	5 yr	
Teizeira et al., 2001	41	34 (82.9%)	6 to 16	Deep caries or exposed pulp With or without apical pathosis	Not given	CH	GIC	Vital/asymptomatic/ dentine bridge/ intact PDL	24 to 32 w	
DeRosa, 2006	26	17 (65.4%)		Not given		CH	Amalgam	Asymptomatic	14 to 88 mo	
El Meligy & Avery, 2006	15 15	13 (86.7%) 15 (100%)	6 to 12	Carious or traumatized teeth Immature apex No apical pathosis Saline		CH MTA	ZOE/Amalgam or composite	Asymptomatic/intact PDL/no resorption/ root development	1 yr	
Witherspoon et al., 2006	19	15 (79.0%)	7 to 16	Carious or traumatic exposure Irreversible pulpitis	6% NaOCl	MTA	Not given	Vital/asymptomatic/ intact PDL/root development	1 yr	
Asgary & Ehsani, 2009	12	12 (100%)	14 to 62	Carious Irreversible pulpitis	Saline	NEC	Permanent rest	Asymptomatic/intact PDL	13 to 20 mo	

NB: All the studies were case series except El Meligy & Avery (2006), which was a randomized controlled trial comparing calcium hydroxide and MTA as pulp dressing material.

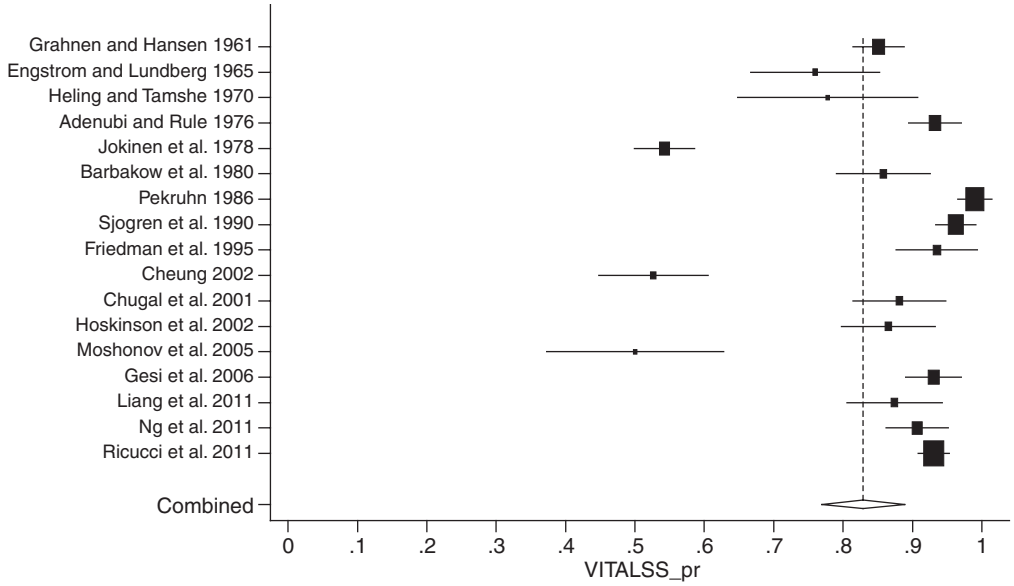


FIG. 11-17 Forest plot showing results of pooled and individual study's probability of maintained periapical health for preoperatively vital teeth undergoing root canal treatment (pooled probability = 0.83; 95% confidence interval: 0.77, 0.89).

Summary of Prognostic Factors for Vital Pulp Therapy

In summary, vital pulp therapy performed under guideline standards with optimal coronal seal achieved promising long-term success in teeth with carious, mechanical, or traumatic exposures of healthy pulps.

The most important factors affecting the outcome of vital pulp therapy are preexisting health of the pulp, adequate removal of infected hard or soft tissues, careful operative technique to avoid damage to residual tissues, and elimination of microbial leakage around the final restoration. It can be difficult to gauge the health of the residual pulp as it is a matter of subjective assessment and relies on experience in pulp diagnosis. The degree of pulp bleeding upon exposure is a more reliable tool to judge the status of the pulp than the preoperative clinical signs and symptoms. Continued bleeding after 10 minutes, even after rinsing with sodium hypochlorite solution, may suggest that the residual pulp was still heavily inflamed and a complete pulpectomy may be a more effective treatment modality. Removal of infected tissue is a matter of subjective experience but may be aided by various dyes. The final factor relies on the correct choice of restorative material and its adequate manipulation to prevent leakage.

Factors such as age and health of the patient, size and nature (carious or traumatic) of pulp exposure, and its duration of exposure to the oral environment (up to 48 hours) do not in themselves compromise outcomes of vital pulp therapy.

OUTCOMES OF NONSURGICAL ROOT CANAL TREATMENT

In contrast to other areas of endodontics, the number of studies and extent of investigation of nonsurgical root canal treatment is more comprehensive, yielding a much greater insight even though the quality and scope of the research does not always reach the highest levels.

A systematic review and meta-analysis of the factors affecting primary root canal treatment outcome conducted by the authors revealed the following: the mean success rate was 83% when vital pulpectomy was performed (Fig. 11-17), which reduced to 72% when the root canal treatment procedure was used to eradicate the established infection associated with a periapical lesion (Fig. 11-18).

Factors Affecting Periapical Health or Healing Following Root Canal Treatment

The factors influencing the maintenance of periapical health or periapical healing of preexisting lesions following root canal treatment may be broadly classified into patient factors (age, sex, general health, tooth anatomy, preoperative pulpal and periapical status), treatment factors (operator variables, canal enlargement, irrigation, medication, culture test, and obturation), and restorative factors. Some factors had a profound impact on success rates, whereas others showed a negligible effect. Patient factors characterizing the nature of the disease showed the most significant effect (periapical status), whereas most of the treatment factors were found to exert a less significant effect; the exceptions were the apical extent of root canal treatment relative to the root canal terminus. In addition, the quality of the postoperative restorative care also exerted a profound influence on outcome of treatment.

Patient Factors

Patient's age and sex consistently had no significant effect on outcome, whereas some specific health conditions (diabetes,[54,67] compromised immune response[123]) apparently had a significant influence. The evidence for the effect of host immune response characterized by the general health of the patient is, however, weak. Emerging evidence indicates that the host response measured by polymorphisms of various genes involved in periapical healing may have an effect on outcomes[142,183,212,213] (Table 11-8). The importance of the host

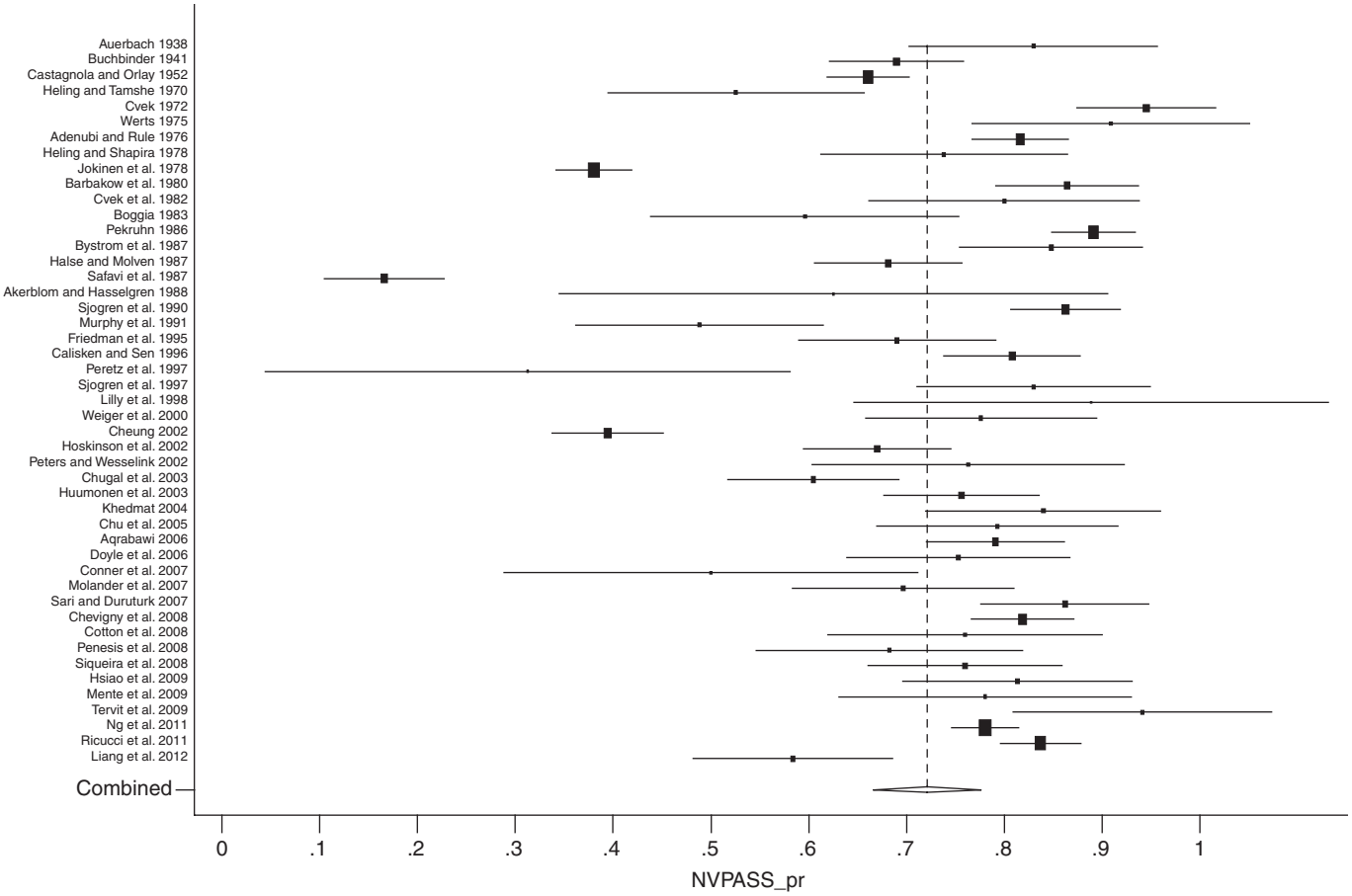


FIG. 11-18 Forest plot showing results of pooled and individual study's probability of periapical health for teeth associated with nonvital pulps and periapical radiolucencies undergoing root canal treatment (pooled probability = 0.72; 95% confidence interval: 0.67, 0.78).

TABLE 11-8

Type of Genes Investigated in Studies on SNPs and Periapical Healing

Study	Gene Function	Genes	Findings
Siqueira et al., 2009	Interleukin 1 Fcγ receptor	IL-1α, IL-1β FcγRIIa, FcγRIIIb	No significant association Significant association
Morsani et al., 2011	Interleukin	IL-1β	Significant association
Siqueira et al., 2011	Fcγ receptor	FcγRIIIa	No significant association
Rôças et al., 2014	Pattern recognition receptors	CD14, TLR4	No significant association

response to maintenance of periapical health or periapical healing was also supported by the statistically significant clustering effect of multiple teeth within the same patient in a prospective study.[150]

The widespread perception that single-rooted teeth with less complicated anatomy should benefit with more predictable and favorable outcomes proves to be untrue. Having accounted for potential confounding factors such as the presence of periapical disease, tooth type does not seem to exert a strong influence on success rates. This would appear to be counterintuitive but may be explained by the logical inference that canal complexities in the apical anatomy probably play a more dominant role than other complexities such as the number and curvature of canals.

The presence and size of a periapical lesion seem to have the most negative effect on periapical health/healing; it therefore follows that these factors must be accounted for when analyzing the influence of any other factor. The profound change in success rates once a periapical lesion becomes established is interesting, as it is correlated with the establishment of infection in the apical canal anatomy. This seems to suggest that once the apical canal complexities become infected, it may be much more difficult to eradicate the infection. The negative influence of large periapical lesions has a reasonable biologic

explanation: the diversity of bacteria (by number of species and their relative abundance) is greater in teeth with larger periapical lesions.[230] An endodontic infection is more likely to persist in canals with a higher number of bacteria present preoperatively.[27] In addition, larger lesions may represent longer-standing root canal infections that may have penetrated deeper into dentinal tubules and accessory anatomy in the complex canal system[206] where mechanical and chemical decontamination procedures may not be so effective. Larger lesions may also represent cystic transformation.[145] Finally, the host response may also play a part, as patients with larger lesions may innately respond less favorably to residual bacteria.[147] This speculation may crystallize into distinct questions for further biologic research into the nature of interactions among host, bacterial infection, and treatment intervention.

Most of the other investigated preoperative factors (pain, tooth tenderness to percussion, soft tissue tenderness to palpation, soft tissue swelling and sinus tract, periodontal probing defect of endodontic origin, root resorption) are in fact different clinical manifestations of periapical disease.[254] They may therefore act as surrogate measures or complement "presence and size of periapical lesion" in measuring the effect of severity of periapical disease within a broad continuous spectrum. Of these, only presence of preoperative pain,[68] sinus tract,[150] swelling,[150] and apical resorption[229] have been found to be significant prognostic factors that have been associated with significantly reduced success rates in root canal treatment.

The biologic explanation for the negative impact of sinus tract and swelling, either in the acute or chronic form, on periapical healing is interesting, as both represent suppuration and the proliferation of microbiota into the periapical tissues, with the inference being that the host tissues must have become locally overwhelmed. The precise reasons for reduced success rates under these conditions remain unclear but must somehow be related to the nature of the host-microbial interaction.

Treatment Factors

Operator

Although the impact of operator qualification and skill has not been specifically investigated, systematic review has shown that the involved clinicians may be grouped into undergraduate students, general practitioners, postgraduate students, and specialists. Studies show a clear trend in superior outcomes by greater experience and training. Clearly technical skills play an important role, but this is difficult and often impossible to quantify. In addition, the technical abilities must be augmented by the overall understanding of the biologic issues and the quest for superior treatment by the operator. If clinicians do not feel they can do optimal treatment, it is incumbent upon them to refer the patient to a more qualified practitioner.

Isolation

The use of rubber dams in modern root canal treatment is widely accepted, and the justification seems almost empirical. One study on retreatment[244] analyzed the influence of rubber dam use compared to cotton roll isolation and found significantly higher success rates with the former approach. Another study reported a significantly higher success rate of root canal–treated teeth[76] when a rubber dam was employed during post placement compared to when it was not. Perhaps as a consequence, the principal justification for rubber dam use is based on medicolegal implications of root canal instrument ingestion or aspiration by the patient.[61]

Magnification and Illumination

Endodontists have repeatedly reinforced the value of magnification and illumination during root canal treatment,[166] but a systematic review failed to draw any objective conclusions on their influence as no article was identified in the current literature that satisfied their inclusion criteria.[46] A prospective study investigated this factor,[150] but researchers found only an insignificant influence on the final outcome. Use of a microscope may sometimes assist location of the second mesiobuccal canal in maxillary molars, but this only made a small difference to the success rates associated with mesiobuccal roots, when a periapical lesion was present.[150] The true benefit of a microscope can only be verified through a randomized controlled trial. However, canal negotiation with less tooth structure removal and fewer procedural accidents is favorable and seems intuitively more consistent with the use of superior magnification and illumination.

Mechanical Preparation: Size, Taper, Extent, and Procedural Errors

The root canal system may be mechanically prepared to a requisite size and taper[199] using a variety of instruments of different cutting designs, tips, tapers, and materials of construction. Their efficacy is often tested in laboratory studies, and the instruments and their utility may have well-characterized properties.[95] Investigation of the influence of type of instrument used for canal enlargement has been undertaken in one nonrandomized prospective study, but the outcome is likely subjective because of many factors, including the protocol adopted for teaching technical skills.[150] In this study, the better success rates for hand or rotary NiTi instruments compared with stainless steel instruments[150] were attributable to the fact that tactile skills training was achieved through a preliminary focus on the use of stainless steel files to develop tactile sensitivity and consistency. Only on demonstration of this competency did the trainees progress to NiTi instruments. More important, such senior students may also have had a better understanding of the biologic rationale for root canal treatment. The ability to gain and maintain apical patency as well as to avoid procedural errors was better instilled in the senior students, whereas in selected cases, NiTi instruments appear capable of achieving the same in primary root canal treatment undertaken by undergraduates.[175]

A key tenet of the European Society of Endodontology (ESE)[61] guidelines is that root canal debridement must be extended to the terminus of the canal system, which is expressed variously as extension to the "apical constriction," or to "0.5 to 2 mm from the radiographic apex," or to the "cementodentinal junction." This guideline is broadly supported by the fact that outcome of treatment is compromised by canal obstruction or failure to achieve patency to the canal terminus.[150,216,229] Ng and colleagues[150] reported a twofold reduction in the success of treatment when the patency to the canal terminus was not achieved. It could be speculated that the lack of mechanical negotiability of canals may be due to the presence of obstructions caused by "denticles," tertiary dentine, acute branching or a fine plexus of apical canals, or dentine/organic debris.

The continued debate on the optimal size of apical preparation remains topical in the absence of definitive evidence; the

findings from relevant in vitro and clinical studies have been previously reviewed.[15] So far, four clinical outcome studies have considered this issue or have systematically investigated the effect of apical size of canal preparation on treatment outcome.[93,102,150,188,223,229] One randomized controlled trial revealed that enlargement of the canal to three sizes larger than the first apical binding file was adequate[188] (the mean final size was ISO #30). The observational studies[95,150,229] had not designed their investigation with apical canal size as their principal focus and neither had they found a statistically significant influence from this factor; nevertheless, they all reported the same inverse trend of decreasing success rates with an increase in size of apical preparation. It was speculated that canal preparation to larger apical sizes may compromise treatment success by generation of more apical dentine debris, which in the absence of an adequate irrigation regimen serves to block apical canal exits that may still be contaminated with bacteria. Continued generation of dentine debris, in the absence of sufficient irrigation, may lead to what is termed *dentine mud,* which ultimately creates a blockage. The impatient or neophyte clinician fails to resist the temptation to force the instrument back to length, resulting in the classically described procedural errors of apical transportation, canal straightening, and perforation. An alternative mechanism is required to explain the higher failures in initially large canals; it is likely that immature roots present a different debridement challenge, where the canal shape is not amenable to planing of the main portions of the canal by conventional instruments. Perhaps an intracanal brush may be a more suitable cleaning device in such teeth. The findings from these studies therefore do not concur with views that more effective bacterial debridement may be achieved with larger apical preparations.[33,164,184]

The issue of apical preparation size should be considered together with that of the size and taper of the rest of the canal preparation. Again, there is a paucity of sufficient direct evidence for the influence of degree of canal taper on root canal treatment outcome. The ESE guidelines[63] recommend only that canal preparation should be tapered from crown to apex without stipulating any particular degree of taper. Three studies have analyzed the influence of canal preparation taper on primary treatment and retreatment outcome, although again, none had focused their investigation primarily on this factor.[93,150,220] Smith and coworkers,[220] using loose criteria for determination of success, found that a "flared" preparation (wide taper) resulted in a significantly higher success rate compared with a "conical" preparation (narrow taper); the exact degree of taper was not reported and the effects of other treatment and nontreatment parameters were not controlled. In contrast, Hoskinson and associates[93] and Ng and colleagues,[150] using strict criteria, did not find any significant difference in treatment outcome between narrow (.05) and wide (.10) canal tapers. The controlled use of stainless steel instruments in a step-back technique may create .05 (1 mm step-back) or .10 (0.5 mm step-back) tapers, although, of course, uncontrolled use of such instruments may generate a variety of shapes. Ng and colleagues[150] also compared these (.05 and .10) preparation tapers with .02, .04, .06, and .08 tapers (generally achieved by using greater taper nickel-titanium instruments) and found no significant effect on treatment outcome. They cautioned that their investigation of the influence of canal preparation taper without randomization could be influenced by the initial size of the canal, the type of instrument used, and operator experience.

Triangulation of the data on the effects of canal preparation size and taper on treatment outcome may intuitively lead to the conclusion that as far as current best evidence indicates, it is not necessary to over-enlarge the canal to achieve periapical healing. An apical preparation size of ISO 30 with a .05 taper for stainless steel instrumentation or .06 taper for NiTi instrumentation is sufficient. Precisely what biologic and hydrodynamic mechanisms underpin such sufficiency is more difficult to define based on available evidence. Although a number of laboratory studies[4,84,112] have investigated the interaction between canal dimensions and irrigation or obturation dynamics, the precise physical, chemical, or biologic mechanisms that ultimately enable periapical healing remain unknown, although collaborations with fluid dynamics specialists[84] and (micro) biologists[82] may ultimately yield a clearer picture.

Procedural errors during root canal preparation include canal blockage, ledge formation, apical zipping and transportation, straightening of canal curvature, tooth or root perforation at the pulp chamber or radicular level, and separation of instruments. Instrument separation during treatment has been found to reduce the success rate significantly[150,229]; however, the reported prevalence of instrument separation was low (0.5% to 0.9%) in these studies, precluding an analysis of causative factors. A case-control study[225] revealed no significant difference in success rates between periapically involved teeth with or without retained separated instruments. The stage of canal debridement at which instrument separation occurred and the justification for their retention may have implications on the outcome. The coronoapical location of a separated instrument and whether the instrument was successfully bypassed were found to have no effect on treatment outcome.

Irrigant

Different chemical agents have been used as irrigants for root canal treatment, singly or in various combinations, both in clinical practice and in the studies reviewed. They have included water, saline and solutions of local anesthetic, sodium hypochlorite, iodine, chloramine, sulfuric acid, EDTA, hydrogen peroxide, organic acid, Savlon, urea peroxide, and Biosept (a quaternary ammonium compound).[153] Most of the studies had used sodium hypochlorite as an irrigant[153] regardless of whether it was primary treatment or retreatment. This is consistent with the ESE guidelines[61] for irrigation, which recommend a solution possessing disinfectant and tissue-dissolving properties.

One prospective study[150] systematically investigated the effect of the irrigant on the success rates of root canal retreatment, which, although not a randomized controlled trial, revealed interesting new findings on the effects of irrigants. Even though a higher concentration of sodium hypochlorite made negligible difference to treatment outcome, the additional use of other specific irrigants had a significant influence on success rates.[150] The finding of a lack of improvement in periapical healing with the use of a higher concentration NaOCl solution is consistent with previous clinical/microbiologic findings.[29,41] Comparing 0.5% to 5.0% NaOCl solution for irrigation, it was found that the concentration of solution, per se, did not appear to increase the proportion of teeth rendered culture-negative[29] or associated with greater periapical healing.[41] As iodine and sodium hypochlorite are both halogen-releasing

agents and attack common key protein groups,[129] the finding that the additional use of 10% povidone-iodine for irrigation had no additional influence on treatment success was as expected. Surprisingly, however, the additional use of 0.2% chlorhexidine solution for irrigation was found to reduce the success of treatment significantly.[150] This finding was in complete contrast to previous reports[211,252] on its equivalent or superior in vivo antibacterial efficacy when compared with sodium hypochlorite solution. The use of chlorhexidine as a final irrigant following sodium hypochlorite irrigation had been recommended[107] and was justified on several grounds, including its substantivity in root dentin (i.e., prolonged antibacterial effect),[185] relative lack of toxicity,[117] and broad-spectrum efficacy.[130] Not until recently has alternate irrigation with sodium hypochlorite and chlorhexidine solution raised serious concerns because of their interaction product. The interaction product is thought to be an insoluble precipitate containing para-chloroaniline, which is cytotoxic and carcinogenic.[14,24] Apart from mutually depleting the active moiety in the two solutions for bacterial inactivation, the precipitate may cause persistent irritation to the periapical tissue and block dentinal tubules and accessory anatomy, possibly explaining the observed lower success rate when chlorhexidine was used as an additional irrigant.

Ng and associates[150] also found that the additional use of EDTA had a profound effect on improving radiographically observed periapical healing associated with root canal treatment (OR = 1.5 [1.1, 2.0]). In contrast, the observed synergistic effect of sodium hypochlorite and EDTA had been previously demonstrated in terms of bacterial load reduction[26] but not periapical healing. The long-term (≥ 2 years) outcome of their cases stratified by canal disinfection protocols[25] did not support their microbiologic findings. Their reported success rate for alternate irrigation with sodium hypochlorite and EDTA solutions (67%) was low when compared to the success rate for irrigation using saline (91%), 0.5% sodium hypochlorite (92%), or 5% sodium hypochlorite (86%) solutions.[162] The reported outcome data were unexpected, as preobturation negative bacterial culture was achieved in all cases. Given the complexity of their study design (clinical and microbiologic), their sample size was restricted to 11 to 15 teeth per group, limiting their outcome data. The synergistic effect of the two disinfectants has been attributed to the chelating properties of the sodium salts of EDTA, and their roles have been reviewed by Zehnder.[261] EDTA solution assists negotiation of narrow or sclerosed canals by demineralization of root dentine and helps in the removal of compacted debris from noninstrumented canal anatomy. It may also facilitate deeper penetration of sodium hypochlorite solution into dentine by opening dentinal tubules and removing the smear layer from the instrumented surface. Lastly, it may help detach or break up biofilms adhering to root canal walls.[85]

Medicament

Most previous treatment outcome studies have not standardized the type of root canal medicament used in the interappointment period, but the use of several different medicaments has been reported. The list was consistent with that recommended in the ESE guidelines for a medicament with disinfectant properties and included calcium hydroxide, creosote, and iodine solutions.[153] However, there is an absence of studies investigating the influence of this factor on treatment outcome.

The use of a mixture of calcium hydroxide and chlorhexidine has been tested based on the speculation that the mixture would be more effective against *E. faecalis*.[13,78,198]

Root Canal Bacterial Culture Results Prior to Obturation

In the past, in various centers of endodontic excellence, completion of root canal treatment by obturation would only be acceptable after a negative culture test was obtained from the canal, confirming the absence of bacteria in the part of the root canal system that could be sampled.[23,70,143] This practice has fallen out of clinical favor because of the perceived predictability and good prognosis of root canal treatment without microbiologic sampling. Sampling procedures are considered lengthy, difficult, often inaccurate, requiring laboratory support and having low benefit-to-cost ratio.[138,139] A preobturation negative culture result may increase treatment success twofold (Fig. 11-19). One large study[201] helped contribute to the demise of the canal-culture test; however, even this study showed a 10% difference in success in favor of the negative culture test when periapical disease was present. The outcome is even worse when a positive culture test result combines with the presence of a periapical lesion.

Numerous studies* have evaluated the effect of different stages of root canal treatment on the intraradicular microbiota, both qualitatively and quantitatively (Table 11-9). Some studies merely report positive culture tests, whereas others have identified and quantified intraradicular microbiota before and after various stages of treatment.

The effect of the "mechanical preparation" of the canal(s) on the microbiota has been tested using only water or saline as the irrigant. Taken collectively, the studies show that negative cultures were achieved on a weighted pooled average in 31% of the cases (range 0 to 79%). When sodium hypochlorite (concentration range 0.5% to 5%) irrigation supplemented the "mechanical preparation," the frequency of negative cultures immediately increased to a weighted pooled average of 52% (range 13% to 95%) (see Table 11-9).

Most studies report culture reversals during the interappointment period when active antibacterial dressing is not used in the root canal system between appointments. The reversals are due to regrowth of residual bacteria or recontamination by bacteria from coronal restoration leakage. When active interappointment antibacterial dressing is used, negative cultures in the subsequent visit were achieved on average in 71% of cases (range 25% to 100%) (see Table 11-9).

Effect of Persistent Bacteria on Root Canal Treatment Outcome

The bacteria present in preobturation cultures have included *Enterococcus*, *Streptococcus*, *Staphylococcus*, *Lactobacillus*, *Veillonella*, *Pseudomonas*, *Fusobacterium* species, and yeasts. Studies have been variable as to the relationship between individual species and treatment failure. Although the overall failure rate for cases with positive cultures was 31%, teeth testing positive for *Enterococcus* species had a failure rate of 55%, and teeth with positive cultures for *Streptococcus* species had 90%

Text continued on p. 504

*Please see the following references: 2, 7, 10, 16, 26-29, 33, 37, 41, 42, 60, 77, 79, 99, 104, 108, 110, 124, 137, 154, 161, 163, 168, 169, 173, 179, 181, 208, 209, 210, 214, 215, 217, 227, 248, 252, 257, and 258.

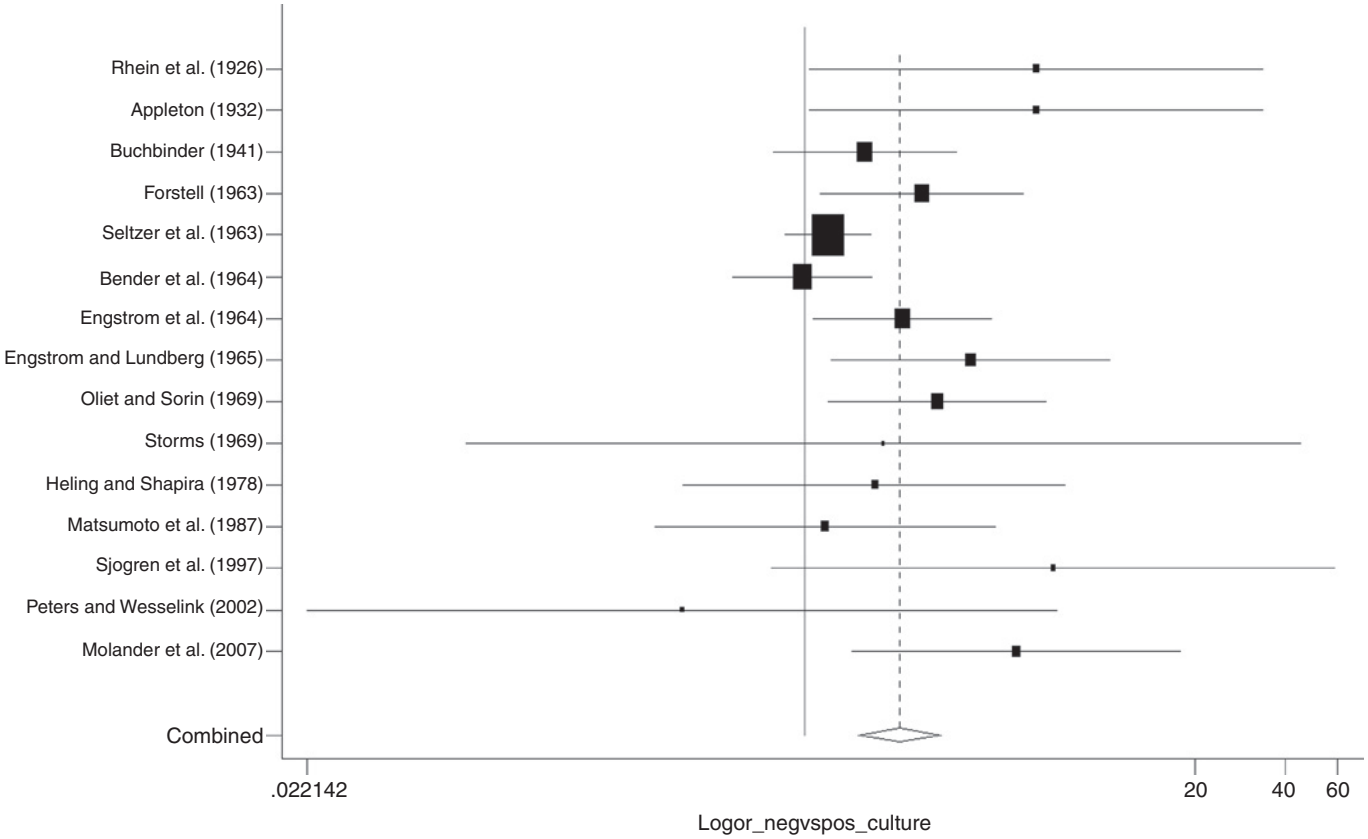


FIG. 11-19 Forest plot showing pooled and individual study's odds ratio (OR) for periapical health of teeth undergoing root canal treatment with preobturation negative versus positive culture test results (pooled OR = 2.1; 95% CI: 1.5, 2.9).

TABLE 11-9

Summary of Studies Evaluating the Effect of Root Canal Treatment Procedures on Bacterial Presence by Culture

			Percentage of Samples with Bacterial Presence		
Study	**Year**	**Sample Size**	**At Baseline**	**After Preparation ± Irrigation**	**Next Visit (after Dressing+/–)**
Auerbach	1953	60 teeth	93% (56/60)	**Chlorinated soda (double strength):** 22% (12/56)	—
Ingle & Zeldow	1958	89 teeth	73% (65/89)	**H_2O:** 70% (62/89) Some initially –ve became +ve after treatment	—
Stewart et al.	1961	77 teeth	100% (77/77)	**0.5% NaOCl + Gly-oxide:** 2% (1/44) **0.5% NaOCl + 3% H_2O_2:** 9% (3/33)	**No dressing:** 0.5% NaOCl + Gly-oxide: 34% (15/44) 0.5% NaOCl + 3% H_2O_2: 39% (17/33)
Nicholls	1962	155 teeth	100% (155/155)	**Alkaline chloramine:** 53% (39/74) **H_2O_2 and 2% NaOCl:** 50% (30/60) **H_2O and 2% NaOCl:** 71% (15/21)	—
Grahnén & Krasse	1963	97 teeth	77% (75/97)	**NaCl:** 72% (23/32) **Biocept:** 66% (21/32) **Nebacin:** 36% (12/33) Some initially –ve became +ve after Tx	**No dressing:** NaCl: 47% (15/32) Biocept: 47% (15/32) Nebacin: 18% (6/33)

TABLE 11-9

Summary of Studies Evaluating the Effect of Root Canal Treatment Procedures on Bacterial Presence by Culture—cont'd

			Percentage of Samples with Bacterial Presence		
Study	Year	Sample Size	At Baseline	After Preparation ± Irrigation	Next Visit (after Dressing+/–)
Engström	1964	223 teeth (untreated or retreated)	60% (134/223)	**Biosept or Iodophor, plus alcohol, chloroform, and 0.5% NaOCl:** No data	**5% I_2 in 10% IKI:** 2nd visit: 43% (58/134); 3rd visit: 22% (29/134); 4th visit: 8% (9/134); 5th visit: 3% (4/134); 6th visit: 2% (3/134); 7th visit: 16% (22/134)
Olgart	1969	207 teeth	72% (149/207)	**H_2O_2 and 0.5% NaOCl or H_2O_2 and 1% NaOCl:** 43% (88/207)	**No dressing:** 34% (70/207)
Bence et al.	1973	33 teeth	100% (33/33)	**Pre-irritation:** 1st file: 93%, enlargement with #3: 14%, #4: 11%, #5: 21% (32% of instruments showed +ve culture, regardless of size) **5.25% NaOCl:** 48-hr culture: 4% dentine, 10% pp 5-day culture: 8% dentine, 26% pp	**No dressing:** 8% dentine, 12% pp samples of teeth with negative culture after irrigation
Akpata	1976	20 extracted teeth	100% (20/20)	**NaCl:** 65% (13/20)	**38% CMCP:** 20% (2/10) When PP sample –ve, crushed tooth yielded –ve culture; When PP +ve, crushed teeth yielded +ve or –ve cultures
Cvek et al.	1976	108 teeth	NaCl group: 53% (18/34) 0.5% NaOCl group: 63% (29/46) 5% NaOCl group: 79% (22/28)	**NaCl:** 83% (15/18) **0.5% NaOCl:** 59% (17/29) **5% NaOCl:** 68% (15/22)	—
Byström & Sundqvist	1981	15 teeth	100% (15/15)	**Saline:** 100% (15/15)	**No dressing:** 47% (7/15) (5th visit) Where initial bacteria load high, difficult to eliminate
Byström & Sundqvist	1983	15 teeth	100% (15/15)	**0.5% NaOCl:** 87% (13/15)	**No dressing:** 20% (3/15) (5th visit)
Byström & Sundqvist	1985	60 teeth	100% (60/60)	**0.5% NaOCl:** No data **5% NaOCl:** No data **5% NaOCl + 15% EDTA:** No data	**No dressing:** 0.5% NaOCl: 12/20 (2nd visit); 8/20 (3rd visit) 5% NaOCl: 10/20 (2nd visit); 6/20 (3rd visit) 5% NaOCl + 15% EDTA: 11/20 (2nd visit); 3/20 (3rd visit)
Byström et al.	1985	65 teeth	100% (65/65)	**0.5% NaOCl:** No data **5.0% NaOCl:** No data	**CH:** 0/35 (1 month), 1/35 (2-4 days) **CP/CMCP** (2 wks): 10/30
Sjögren & Sundqvist	1987	31 teeth	100% (31/31)	**0.5% NaOCl plus ultrasonic debridement:** No data	**No dressing:** 29% (9/31) at 2nd visit; 23% (7/31) at 3rd visit

Continued

TABLE 11-9

Summary of Studies Evaluating the Effect of Root Canal Treatment Procedures on Bacterial Presence by Culture—cont'd

			Percentage of Samples with Bacterial Presence		
Study	Year	Sample Size	At Baseline	After Preparation ± Irrigation	Next Visit (after Dressing+/–)
Koontongkaew et al.	1988	15 teeth	100% (15/15)	**3% H_2O_2/5.25% NaOCl:** No data	**CMCP**: 1-day dressing: 40% (2/5); 3-day dressing: 20% (1/5); 7-day dressing 10% (1/10) **No dressing**: 60% (3/5) after 1 day, 20% (1/5) after 3 or 7 days
Reit & Dahlén	1988	35 teeth	91% (32/35)	**0.5% NaOCl:** No data	**CH**: After 14 days: 23% (8/35); after 21 days: 26% (9/35)
Molander et al.	1990	25 teeth	96% (24/25)	**0.04% iodine:** No data	**Clindamycin**: After 14 days: 16% (4/25); after 21 days: 24% (6/25)
Sjögren et al.	1991	30 teeth	100% (30/30)	**0.5% NaOCl:** 50% (15/30)	**CH:** 10 min: 50% (6/12) at 1 wk later 7 day: 0% (0/18) (none after 1-5 wks later without dressing)
Ørstavik et al.	1991	23 teeth	96% (22/23)	**NaCl** irrigation and enlarged to: #20-25: 87% (20/23) further to #35-80: No data	**CH**: 34% (8/23); #35/40: 40% (6/15) #>40: 25% (2/8)
Yared & Bou Dagher	1994	60 teeth	100% (60/60)	**1% NaOCl:** Enlarged to #25: 73% (22/30) Enlarged to #40: 23% (7/30)	**CH:** 0% (0/60)
Gomes et al..	1996	42 root canals: Untreated (n = 15) Retreated (n = 27)	95% (40/42)	**2.5% NaOCl:** No data	**Empty canal** (7-10 days): 73% (29/40)
Sjögren et al.	1997	55 teeth (single canal)	100% (55/55)	**0.5% NaOCl:** 40% (22/55)	—
Dalton et al.	1998	46 teeth	100% (46/46)	NaCl + NiTi files: 68% (15/22); NaCl + K-files: 75% (18/24)	—
Reit et al.	1999	50 teeth	84% (42/50)	Enlarged to #35 (curved) or #50 (straight) with **0.5% NaOCl**: No data	**5% IKI** (5-7 days): 44% (22/50) **Empty** (7 days): 44% (22/50)
Peciuliene et al.	2000	25 teeth	80% (20/25);	**2.5% NaOCl and 17% EDTA:** No data	**Medication unknown:** 28% (7/25)
Shuping et al.	2000	42 teeth	98% (41/42)	**1.25% NaOCl:** 38% (16/42)	**CH:** 8% (3/40)
Lana et al.	2001	31 teeth	87% (27/31)	**2.5% NaOCl:** No data	**CH:** 13% (4/31) **Empty for 7 days:** 23% (7/31)
Peciuliene et al.	2001	40 teeth	83% (33/40)	**2.5% NaOCl and 17% EDTA:** 30% (10/33)	**CH (10-14 days)**: 25% (5/20) **IKI: 2% I_2 in 4% KI (10 min):** 5% (1/20)
Peters et al.	2002	42 teeth	Instrumentation to #20: 100% (42/42)	**Enlarged to #35 with 2% NaOCl:** 23% (10/42)	**CH** (4 weeks): 71% (15/21); further irrigation: 43% (9/21)

TABLE 11-9

Summary of Studies Evaluating the Effect of Root Canal Treatment Procedures on Bacterial Presence by Culture—cont'd

			Percentage of Samples with Bacterial Presence		
Study	Year	Sample Size	At Baseline	After Preparation ± Irrigation	Next Visit (after Dressing+/–)
Card et al.	2002	40 mandibular teeth/canals	95% (38/40)	**1% NaOCl** Profile instrumentation (.04 taper): 0/13 of cuspids and bicuspids, 5/27 of mesiobuccal canals Further LightSpeed instrumentation to size 57.5-65: 3/27 mesiobuccal canals of molars Only 1/16 of those mesiobuccal canals with detectable communication with the mesiolingual canals had +ve culture after the first preparation using ProFile instruments	No data
Kvist et al.	2004	96 teeth	98% (94/96)	**0.5% NaOCl:** 63% (60/96)	**CH** (7 days): 36% (16/44) **IPI** (10 min): 29% (15/52)
Chu et al.	2006	88 canals	99% (87/88)	**0.5% NaOCl:** No data	**CH, Septomixine forte, or Ledermix**: 36% (32/88) Exposure of pulp, tooth type, acute versus chronic condition, size of lesion, and type of medication had no significant effect
Paquette et al.	2007	22 teeth (single canal)	100% (22/22)	**2.5% NaOCl:** 68% (15/22)	**2% CHX**: 45% (10/22)
Siqueira et al.	2007a	11 teeth (single root)	100% (11/11)	**2.5% NaOCl:** 55% (6/11)	**CH/CPMC:** 9% (1/11)
Siqueira et al.	2007b	11 teeth (single root)	100% (11/11)	**2.5% NaOCl:** 45% (5/11)	**CH:** 18% (2/11)
Vianna et al.	2007	24 teeth (single root)	100% (24/24)	**Saline + 2% CHX gel:** 33% (8/24)	**2% CHX, CH or mixture:** 54% (13/24) Type of medication had no significant effect
Wang et al.	2007	43 canals	91% (39/43)	**Saline + 2% CHX gel:** 8% (4/39)	**2% CHX + CH:** 8% (3/36) Size of apical preparation (40 versus 60) had no significant effect.
Markvart et al.	2012	24 teeth	88% (21/24)	**2.5% NaOCl:** 63% (15/24)	**17% EDTA irrigation and 10 min 5% IKI medication:** 50% (12/24) Box preparation (#60): 67% (8/12) Cone preparation (#25-30): 33% (4/12)
Xavier et al.	2013	48 teeth (single canal)	100% (40/40)	**1% NaOCl:** 75% (9/12) **2% CHX:** 75% (9/12)	**CH:** 75% (18/24)

NaOCl, sodium hypochlorite; CH, calcium hydroxide; CP, camphorated phenol; CMCP, camphorated monochlorophenol; PP, paper point sample; r culture, culture test before obturation.

failures.[70] In another study, good-quality root canal treatment on 54 teeth with asymptomatic periapical disease gave an overall success rate of 74%, but teeth with positive cultures for *Enterococcus faecalis* achieved a success rate of only 66%.[231] The success rate for teeth with no bacteria was 80%, whereas that for teeth with bacteria in the canal before obturation was 33%. These associations cannot be regarded as direct cause-and-effect associations, but they further emphasize the need to determine a relationship between microbial diversity and treatment outcome.

A monkey-model study[63] used a four- or five-strain infection to test the effect of debridement and obturation procedures on outcome. When bacteria remained after chemomechanical debridement, 79% of the root canals were associated with nonhealed periapical lesions, compared with 28% when no bacteria were found to remain. Combinations of several residual bacterial species were more frequently related to nonhealed lesions than were single strains. When no bacteria remained at the end of chemomechanical debridement, healing occurred independently of the quality of the root filling. In contrast, when bacteria remained in the canal system, there was a greater correlation with nonhealing associated with poor-quality root fillings than in technically well-performed fillings. In root canals where bacteria were found after removal of the root filling, 97% had not healed, compared with 18% for those root canal systems with no bacteria detected upon removal of root filling. The study emphasizes the importance of reducing bacteria below detection limits before permanent root filling in order to achieve optimal healing conditions for the periapical tissues. It also reinforces the view that obturation does indeed play a role when residual infection is present.

Regardless of the technique used for obtaining a sample for a canal culture, the presence of a negative culture seems to have a positive impact on treatment outcome. The association of specific species with treatment failure is not well established but the identity of the small group of species isolated from positive cultures is relatively constant and may hold answers to treatment resistance and failure. However, it is important to understand that there are many other factors that can influence root canal treatment outcome.

Root Filling Material and Technique

The interrelationship among the core root filling material, sealer (for filling the gaps between the core material and canal surface), and technique for their placement complicates the investigation of the effect of obturation and technique on treatment outcome. In previous studies on treatment outcome, the most commonly used core root filling material was gutta-percha with various types of sealer or gutta-percha softened in chloroform (chloropercha).[153] The sealers used may be classified into zinc oxide–eugenol–based, glass ionomer–based, and resin-based types.[153] Materials such as Resilon, SmartSeal, and MTA have been adopted but have not significantly penetrated the market, except for the use of MTA in surgical repairs or repairs for immature apices. In any case, there is no evidence to show that the nature of root filling material and the technique used for placement has any significant influence on treatment outcome.

Apical Extent of Root Filling

Of the many intraoperative factors associated with success and failure of root canal treatment, the apical extent of the root canal filling material has been the most frequently and thoroughly investigated. In these previous studies, the apical extent of root fillings has been classified into three categories for statistical analyses: more than 2 mm short of radiographic apex (short), 0 to 2 mm within the radiographic apex (flush), and extended beyond the radiographic apex (long).[153] The apical extent of root filling was found to have a significant influence on the success rates of treatment, regardless of the periapical status.[150,153] Flush root fillings were associated with the highest success rates, whereas long root fillings were associated with the lowest success rates.

Most previous retrospective studies could not distinguish between the effects of apical extent of instrumentation versus the apical extent of obturation; however, the London Eastman study[150] was able to separate the effect of these two factors and found them both to independently and significantly affect periapical healing. The factors correlated with each other, consistent with the fact that canals are normally filled to the same extent as canal preparation.

Extrusion of cleaning, medication, or filling materials beyond the apical terminus into the surrounding tissues may result in delayed healing or even treatment failure due to a foreign body reaction.[105,146,218,260] Magnesium and silicon from the talc-contaminated extruded gutta-percha were found to induce a foreign body reaction, resulting in treatment failure.[146] An animal study has shown that large pieces of subcutaneously implanted gutta-percha in guinea pigs were well encapsulated in collagenous capsules, but fine particles of gutta-percha induced an intense, localized tissue response.[218] The inference that perhaps extrusion of large pieces of gutta-percha may not impact on periapical healing was not supported by data from previous studies.[150,153] The discrepancy may possibly be accounted for by bacterial contamination of the extruded gutta-percha in the clinical data.

The radiographic evidence of "sealer puffs" extruding through the main apical foramina and lateral/accessory canals has been pursued with vigor by some endodontists based on the undaunted belief of its value as "good practice." Their perception is that this represents a measure of root canal system cleanliness, and they ardently argue that healing would follow, albeit with some delay. The published evidence on the effects of sealer extrusion into the periapical tissues has been contradictory. Friedman and colleagues[68] found that extrusion of a glass ionomer-based sealer significantly reduced success rates. In contrast, Ng and associates[150] reported that extrusion of a zinc oxide–eugenol based–sealer had no significant effect on periapical healing. The discrepancy may be attributed to the difference in sealer type and the duration of treatment follow-up. The radiographic assessment of the presence or resorption of sealer may be complicated by the radiolucent property of its basic components and the insufficient sensitivity of the radiographic method used to detect small traces of it.[150] It is possible that, in some cases, the radiographic disappearance of extruded sealer may simply be due to resorption of the radio opaque additive, barium sulfate, or its uptake by macrophages, still resident in the vicinity.[146]

Extruded glass ionomer–based[68] zinc oxide–eugenol–based,[98] silicone-based[98] sealers or endomethasone[22] were not found to be resorbed/absorbed by periapical tissues after 1 year. Traces of calcium hydroxide–based sealer (Sealapex) could still be detected after 3 years.[194] In the latter study, treatments were carried out on primary molar teeth and the canals were

obturated with Sealapex without gutta-percha. With longer duration of follow-up, complete resorption of extruded zinc oxide–eugenol–based sealers (Procosol, Roth Elite)[8] and a resin-based sealer (AH Plus, Dentsply/DeTrey, Konstanz, Germany)[194] was demonstrated in 69% and 45% of the cases after 4 and 5 years, respectively. Ng and associates[150] advanced two explanations for the difference between the effect of extruded core gutta-percha and the zinc oxide–eugenol sealer: the latter is antibacterial and may kill residual microorganisms, whereas it is also more soluble and readily removed by host cells compared to gutta-percha.

Quality of Root Filling

Another much investigated parameter of obturation in retrospective studies has been the radiographic measure of the "quality of root filling." The rationale for complete obturation of the root canal system is to prevent recontamination by colonization from the residual infection or newly invading bacteria. Both are supposedly prevented by a "tight" seal with the canal wall and an absence of voids within the body of the material. Quality of root filling may therefore be regarded either as poor root-filling technique or as a surrogate measure of the quality of the entire root canal treatment, because good obturation relies on properly executed preliminary steps in canal preparation. A systematic review[153] reported that the criteria for judging the quality of root fillings have not been well defined in previous studies.[43,93,216] An unsatisfactory root filling has been defined as "inadequate seal," "poor apical seal," or "radiographic presence of voids." Nevertheless, satisfactory root fillings were found to be associated with significantly higher success rates than unsatisfactory root fillings.[153]

Acute Exacerbation During Treatment

The causes for interappointment "flare-up" or pain have not been precisely determined, and several hypothetical mechanisms involving chemical, mechanical, or microbial injury to the periradicular tissues, as well as psychological influences, have been suggested as contributory to postpreparation pain or swelling.[202,203] Although these factors have not been specifically studied in the context of periapical healing, acute "flare-ups" during treatment were not found to be significantly associated with periapical healing in two studies.[102,216] In contrast, the London Eastman study[150] found that pain or swelling occurred in 15% of cases after chemomechanical debridement and was found to significantly reduce success as measured by periapical healing. This interesting finding may be explained by the hypothesis that "flare-ups" were caused by extrusion of contaminated material during canal preparation. Such material may elicit a foreign body reaction or (transient) extraradicular infection, resulting in treatment failure in a proportion of such cases. Alternatively, acute symptoms may be the result of incomplete chemomechanical debridement at the first appointment, leading to a shift in canal microbial ecology favoring the growth of more virulent microorganisms, leading to further postpreparation pain and treatment failure. The exact biologic mechanisms of failure in these cases remain obscure and warrant further investigation.

Number of Treatment Visits

The number of treatment visits for completing root canal treatment and its effect on periapical healing remains an ongoing controversy. Generally, the argument for single-visit treatment centers around better patient acceptability and cost-effectiveness versus the preference of multiple-visit treatments based on biologic rationale.[224] The premise for multiple-visit treatments has been that primary debridement is not completely effective in eliminating all the adherent bacterial biofilm[147] and the residual bacteria may multiply and recolonize the canal system.[25,29] Therefore, the proponents consider it desirable to use the interappointment period to dress the canal with a long-lasting or slow-release antibacterial agent capable of destroying or incapacitating residual bacteria, as well as to take the opportunity to gauge the initial periapical response before root filling. Calcium hydroxide has served in this capacity for many years because of its ability to dissolve organic tissue, kill bacteria, detoxify antigenic material, and act as a slow-release agent because of its low solubility-product in an aqueous environment. However, its antibacterial ability has come under close scrutiny, with advocates suggesting that the material is not suitable for this purpose.[195] A final resolution to this debate is awaited based on robust clinical evidence. Most of the published randomized controlled trials found no significant influence of healing attributable to number of treatment visits, but they all lacked robust statistical power.

The debate about the merits of single- or multiple-visit treatments will continue unabated given the respective strengths of the motivational drivers among the opposing groups. The issue may only be resolved by properly documented, large randomized controlled trials (which are currently unavailable) because undocumented variables (i.e., operator skill, biologic or technical case complexity, and patient compliance) would continue to bias the outcome.

Post Root Canal Treatment Restorative Factors

Effect of Quality and Type of Restoration

The placement of a coronal restoration after root canal obturation is the final step in the management of teeth undergoing root canal treatment. It has been shown to have a major influence on endodontic outcomes. Teeth with satisfactory coronal restorations were found to have significantly better periapical healing compared with those with unsatisfactory restorations (OR = 3.31; 95% CI: 1.07, 10.30).[153] The term *satisfactory restorations* has been defined as a restoration with no evidence of marginal discrepancy, discoloration, or recurrent caries with absence of a history of decementation.[93,182]

Given that one of the roles of coronal restorations is to prevent postoperative root canal reinfection via coronal leakage, the criteria for unsatisfactory restoration given by Hoskinson and colleagues[93] could not infer coronal leakage when the inner core was still intact. Consequently, the London Eastman study[150] adopted a different classification and definition for unsatisfactory restorations in order to illustrate obvious and potential coronal leakage more accurately. The two groups of unsatisfactory restorations were defined as those with (1) obvious signs of exposed root filling and (2) potential leakage indicated by marginal defects and history of de-cementation. It is perhaps this strategy that contributed to the finding of a profound effect (OR = 10.7; 95% CI: 3.7, 31.5) of coronal leakage on the endodontic outcome.

A number of investigations have been performed based on comparisons between the types of post root canal treatment restorations, including permanent versus temporary restorations,[43,68,150] crown versus acrylic restorations,[43,68,150,216] presence versus absence of posts,[68,150] and nonabutment versus

abutment.[150,216] Teeth that had been permanently restored were associated with significantly higher success rates than their temporarily restored counterparts in some studies[43,68] but not in others.[38,150] The type of permanent restoration[43,68,150,216] was found to have no significant influence on the outcome of treatment.

It has often been recommended that it would be wise to provide a subseal over the root filling in case of loss of a permanent or temporary restoration; the subseal would be glass ionomer (GIC) or zinc oxide–eugenol cement.[196] The placement of a GIC or zinc oxide–eugenol (IRM) cement lining coronal to the gutta-percha filling and underneath the permanent core in order to provide an additional antibacterial coronal seal was found in a prospective study to have no beneficial effect on treatment success.[150]

In summary, the preceding findings overall support the ESE guidelines[61] that an adequate restoration should be placed after root canal treatment to prevent subsequent bacterial recontamination. Therefore, the provision of a good-quality coronal restoration, regardless of type, should be considered the final part of the root canal treatment procedure following obturation.

Use of Root Treated Teeth as Abutments for Prostheses and Occlusal Contacts

Mechanical stress on restorations is a function of the role of individual teeth in the occlusal scheme. The pattern of occlusal loading both in static and dynamic occlusion is dictated by whether the teeth are involved as single units or abutments (bridge/denture) and whether they have holding or guiding contacts. It is reasonable to expect that bridge and denture abutments may be placed under unfavorable loads, as may last-standing teeth in the dental arch.[127] These teeth may therefore be expected to have lower success rates because of a potential increase in the development of cracks and fractures due to fatigue. This observation has been confirmed for teeth functioning as bridge abutments compared with those restored as individual units following root canal treatment.[216]

Summary of Factors Influencing Periapical Healing Following Nonsurgical Root Canal Treatment

The following factors are considered as having a major impact on periapical health subsequent to root canal treatment:

- Presence (Fig. 11-20) and size (Fig. 11-21) of periapical lesion
- Patency at the canal terminus (achieving patency significantly increased the chance of success twofold)[150]
- Apical extent of chemomechanical preparation in relation to the radiographic apex (Fig. 11-22)
- Outcome of intraoperative culture test (see Fig. 11-19)
- Iatrogenic perforation (if present, reduces the odds of success by 30%)[150]
- Quality of root canal treatment judged by the radiographic appearance of the root filling (Fig. 11-23)
- Quality of the final coronal restoration (Fig. 11-24)

The following factors are considered as having minimal impact on root canal treatment outcome:

- Age of patient
- Gender of patient
- Tooth morphologic type
- Specific root canal treatment protocol and technique (preparation, irrigation, and obturation material and technique)

Contemporary improvements in mechanical and chemical canal preparation have not resulted in an increase in the success rate for root canal treatment over the past century (Fig. 11-25). This observation may be explained by the currently available techniques not being effective in eliminating the infection in the apical canal anatomy.

It is notable that all factors that have a strong influence on periapical health after root canal treatment are associated

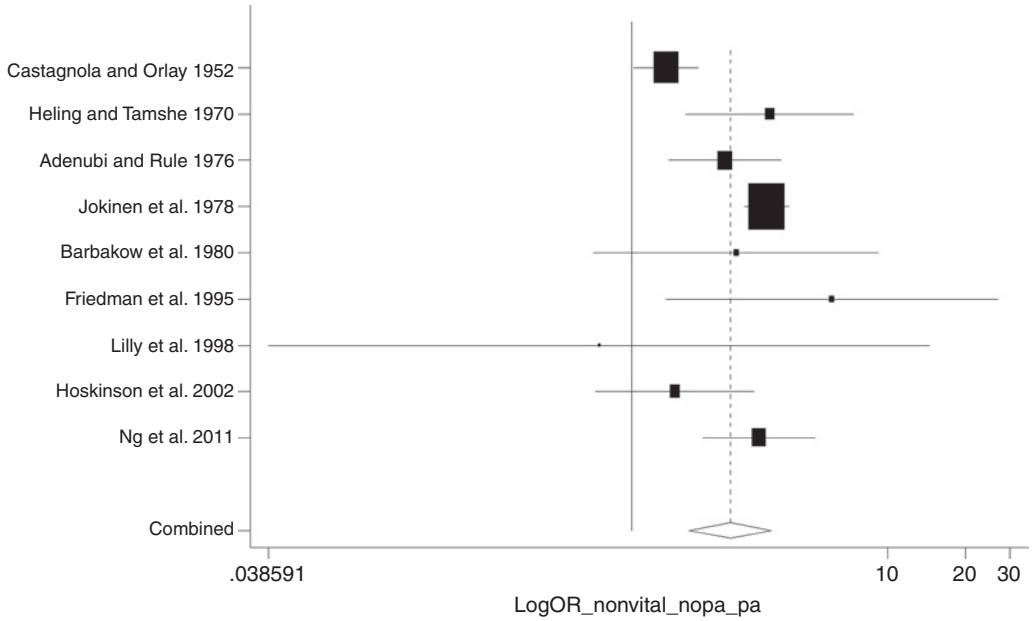


FIG. 11-20 Forest plot showing pooled and individual study's odds ratio (OR) for periapical health of teeth undergoing root canal treatment with preoperative nonvital pulps and absence of periapical radiolucencies versus teeth with nonvital pulps and presence of periapical radiolucencies (pooled OR = 2.4; 95% CI: 1.7, 3.5).

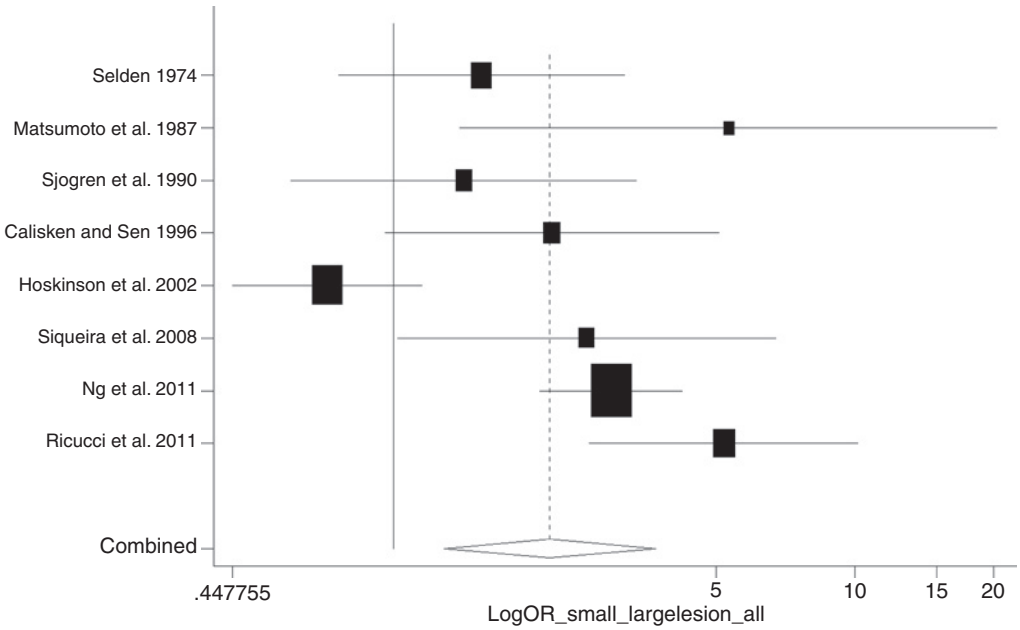
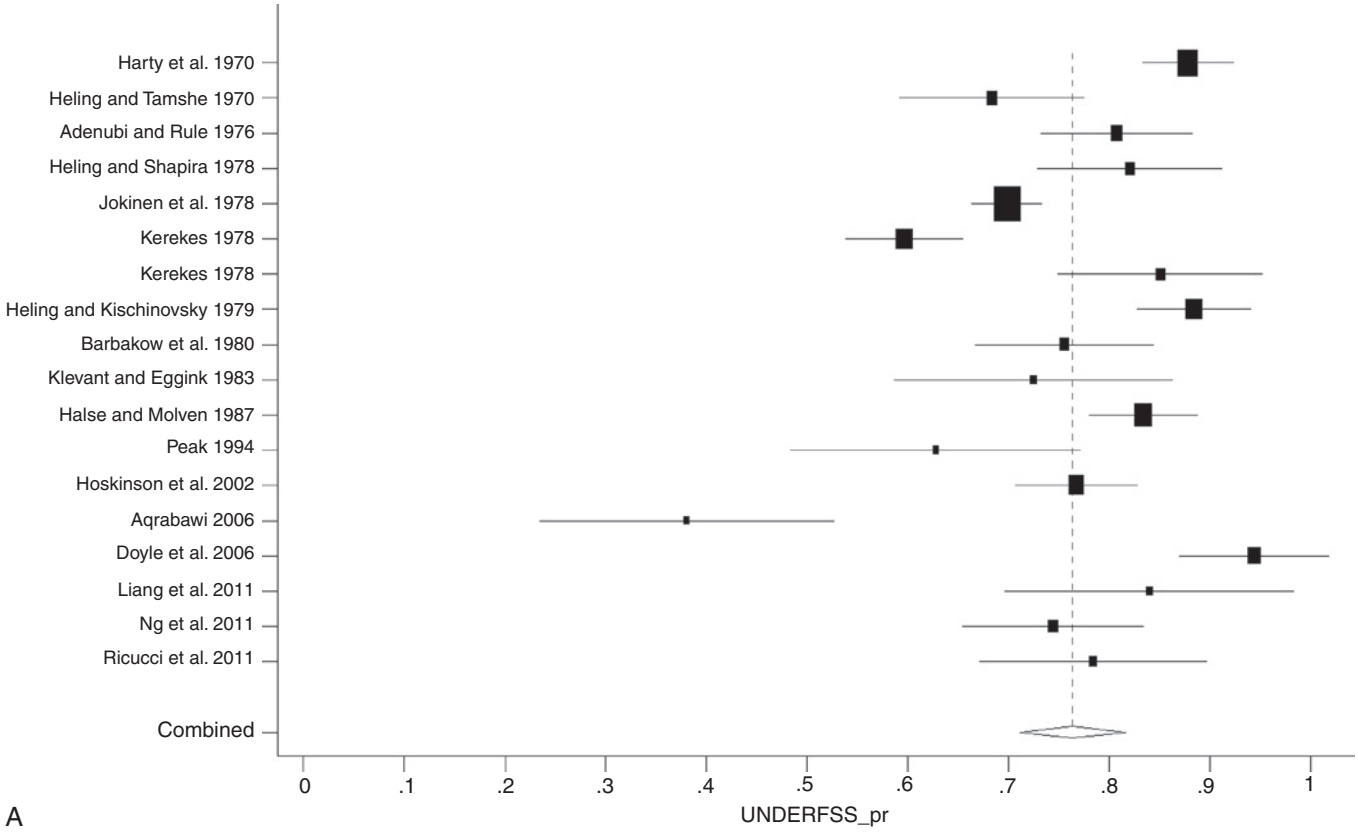


FIG. 11-21 Forest plot showing pooled and individual study's odds ratio (OR) for periapical health of teeth undergoing root canal treatment with preoperative large (> 5 mm) versus small (< 5 mm) periapical radiolucencies (pooled OR = 2.2; 95% CI: 1.3, 3.7).



A

FIG. 11-22 Forest plot showing pooled and individual study's probability of periapical health for teeth undergoing root canal treatment with underextended (0.76 [0.71, 0.82]) (**A**), flush (0.81 [0.76, 0.86]) (**B**), or overextended (0.66 [0.56, 0.75]) (**C**) root fillings.

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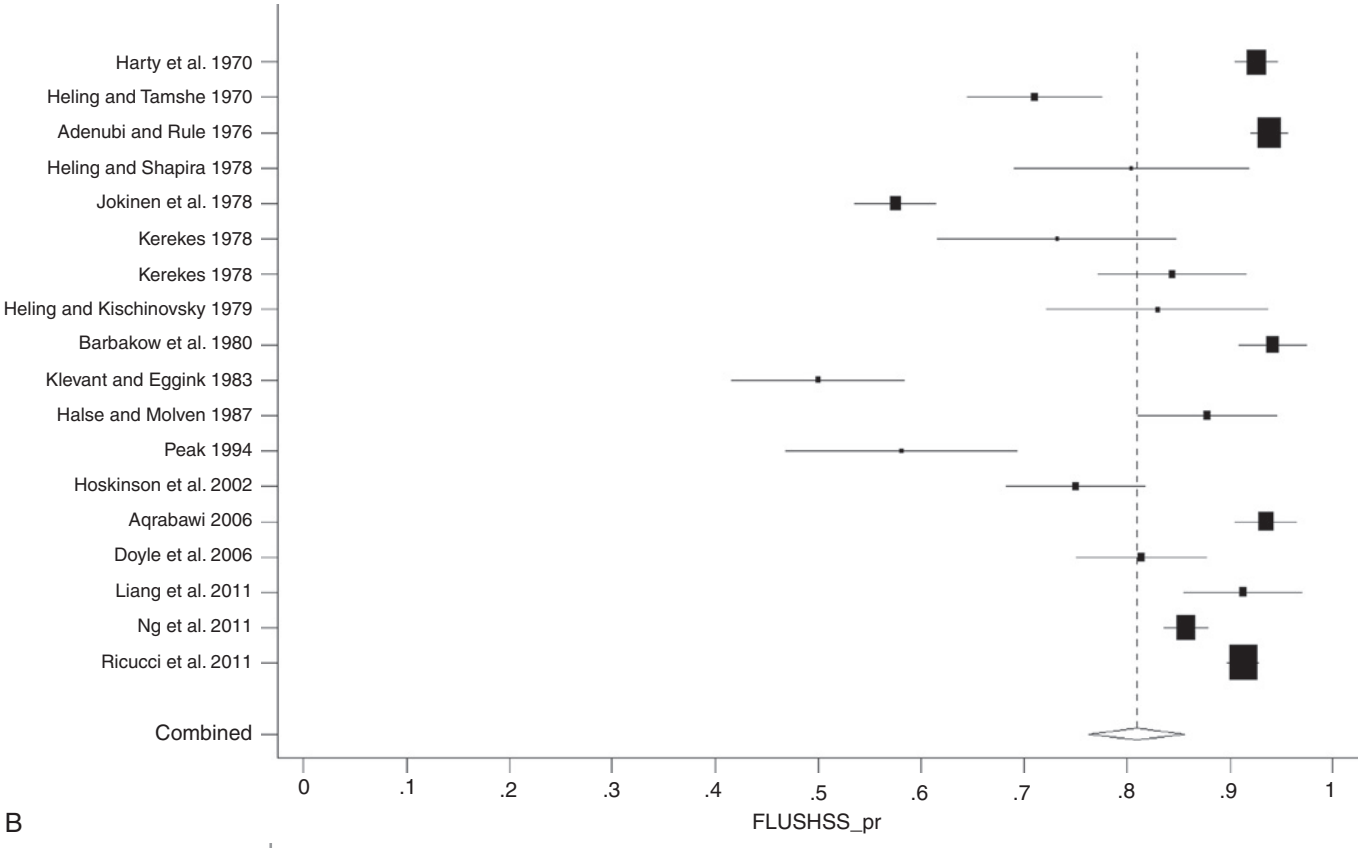
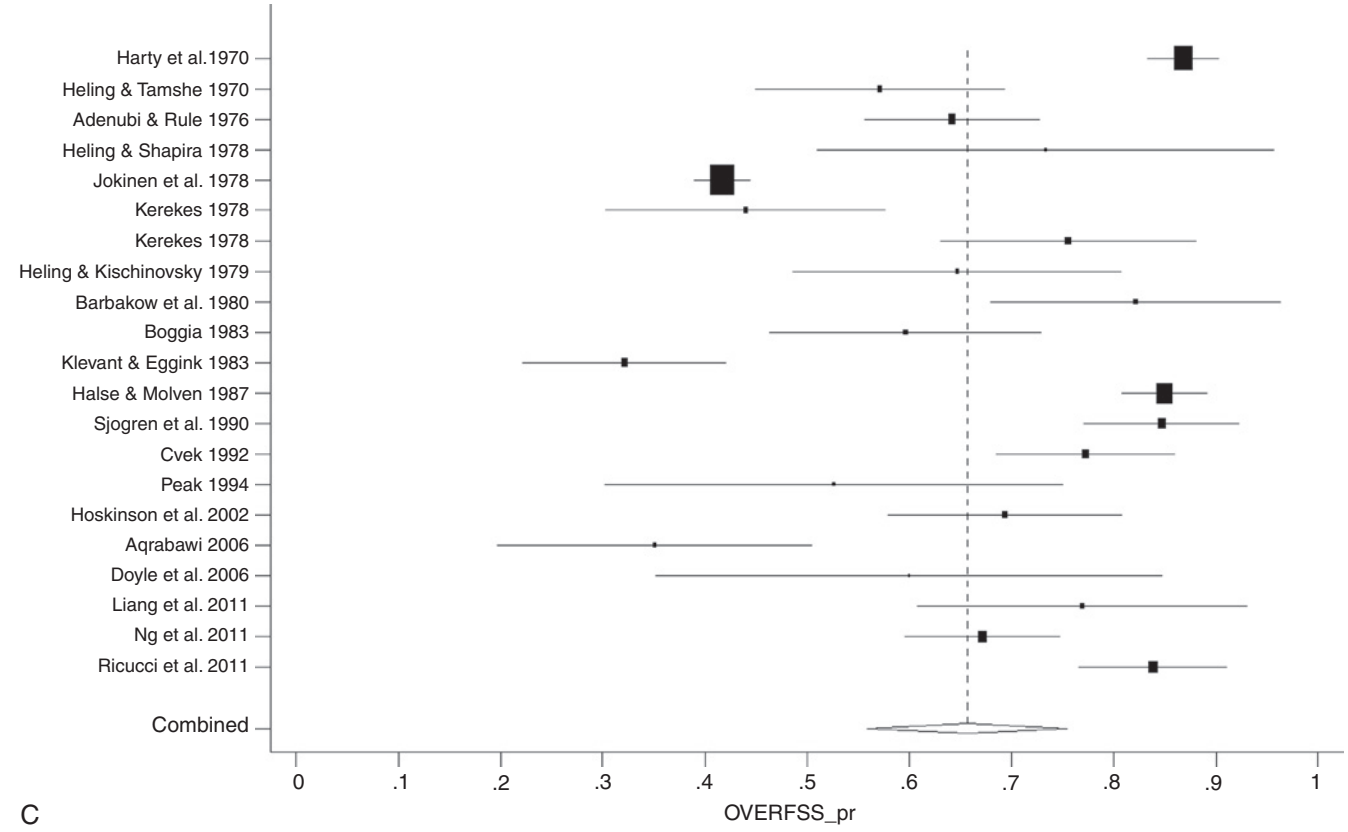


B



C

FIG. 11-22, cont'd

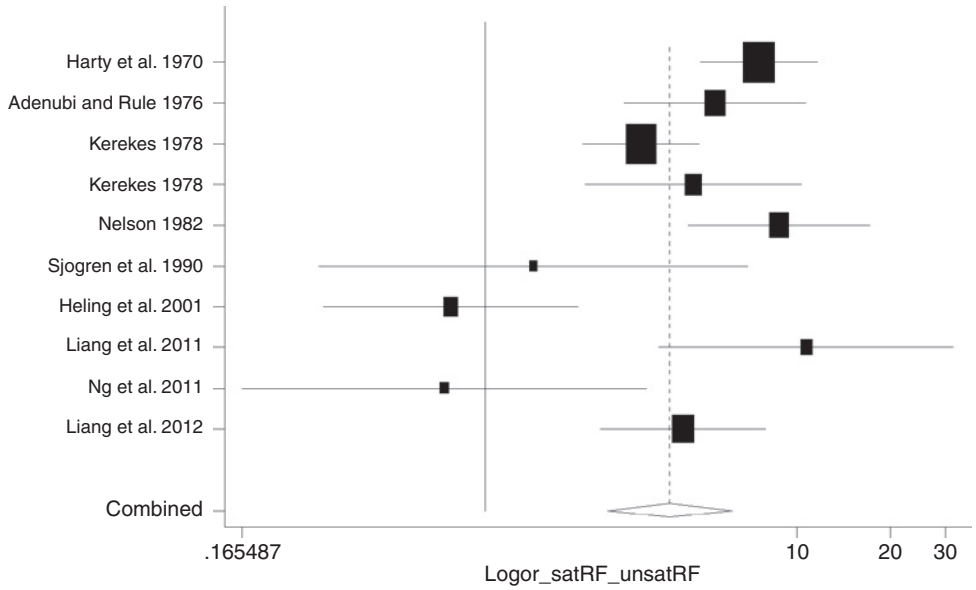


FIG. 11-23 Forest plot showing pooled and individual study's odds ratio (OR) of periapical health for teeth undergoing root canal treatment with good quality versus suboptimal quality root filling (pooled OR = 3.9; 95% CI: 2.5, 6.2).

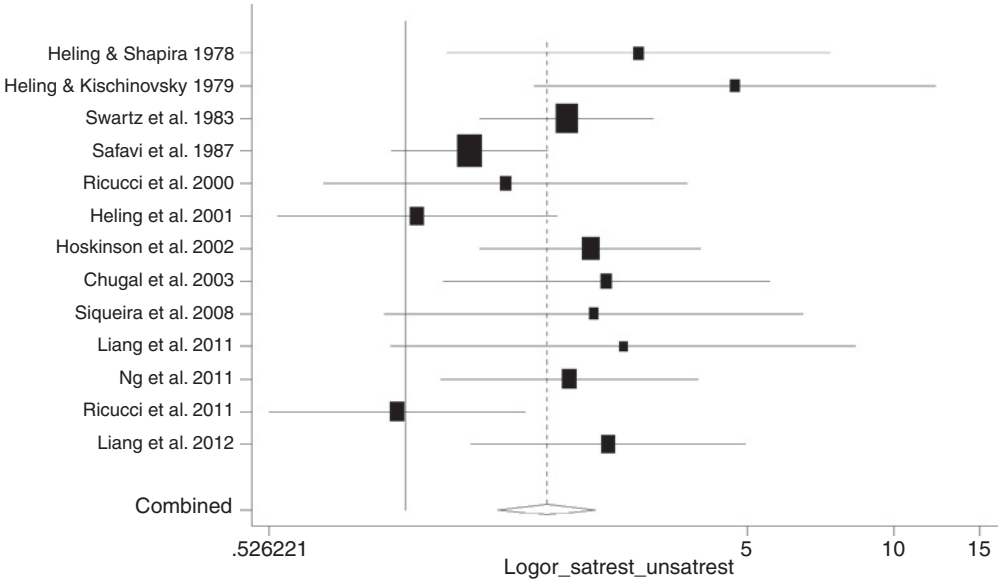


FIG. 11-24 Forest plot showing pooled and individual study's odds ratio (OR) of periapical health for teeth undergoing root canal treatment with satisfactory versus unsatisfactory coronal restoration at follow-up (pooled OR = 1.9; 95% CI: 1.5, 2.5).

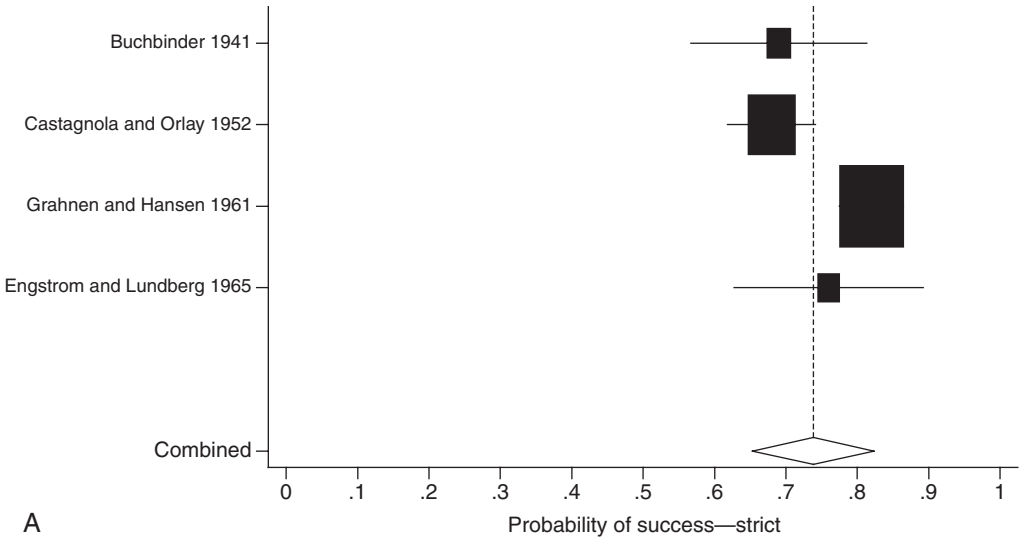
in some way with a persistent root canal infection. Further improvements in root canal treatment outcomes may therefore be achieved by understanding the nature of the root canal infection (especially apically) and the manner in which the microbiota is altered or eliminated during root canal treatment.

Factors Affecting Tooth Survival Following Root Canal Treatment

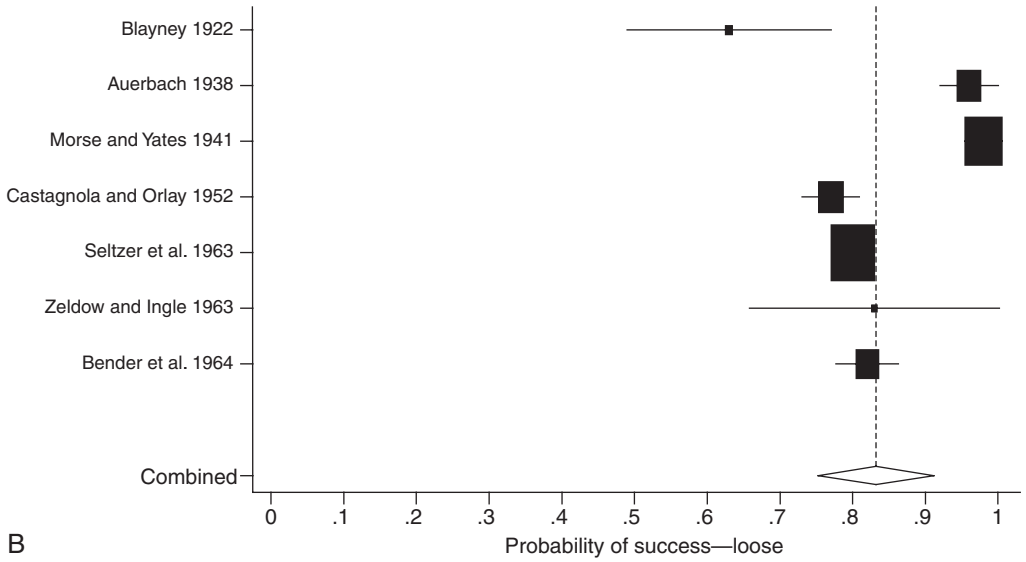
A systematic review and meta-analysis has shown that 93% of endodontically treated teeth survive at 2 years postoperatively; but this survival reduced to 88% at 10 years following treatment (Fig. 11-26). The most common reasons for such tooth loss were due to problems of endodontic origin, tooth/root fracture, or restoration failure.[149,151] Consistent with the studies on periapical healing, the considerations affecting the survival of endodontically treated teeth may be divided into patient, intraoperative, and restorative factors.

Patient Factors

Ng and colleagues[151] found that teeth in patients suffering from diabetes or receiving systemic steroid therapy had a higher chance of being extracted after root canal treatment. The negative influence of diabetes on tooth survival is consistent with



A



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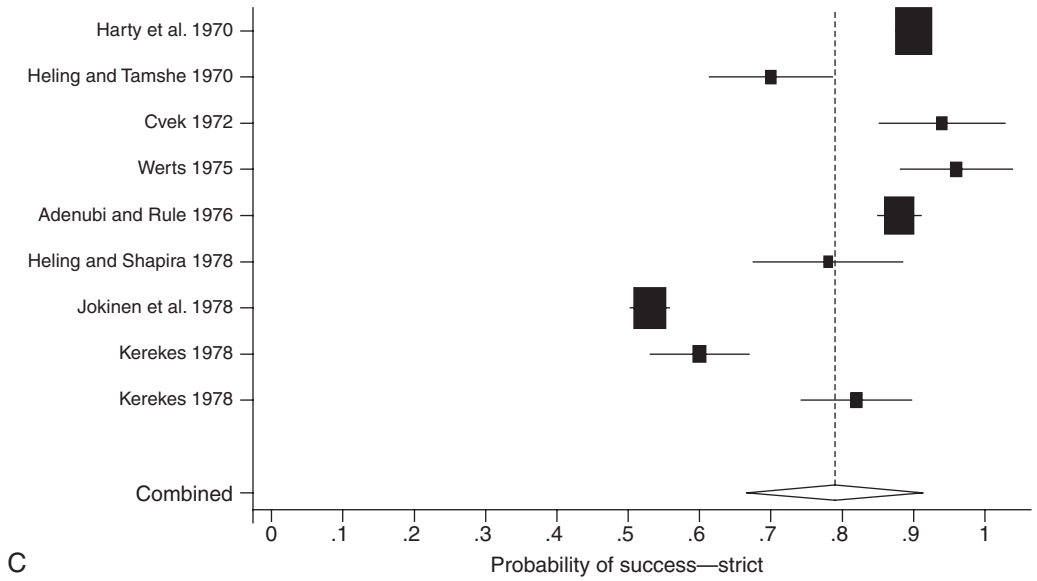
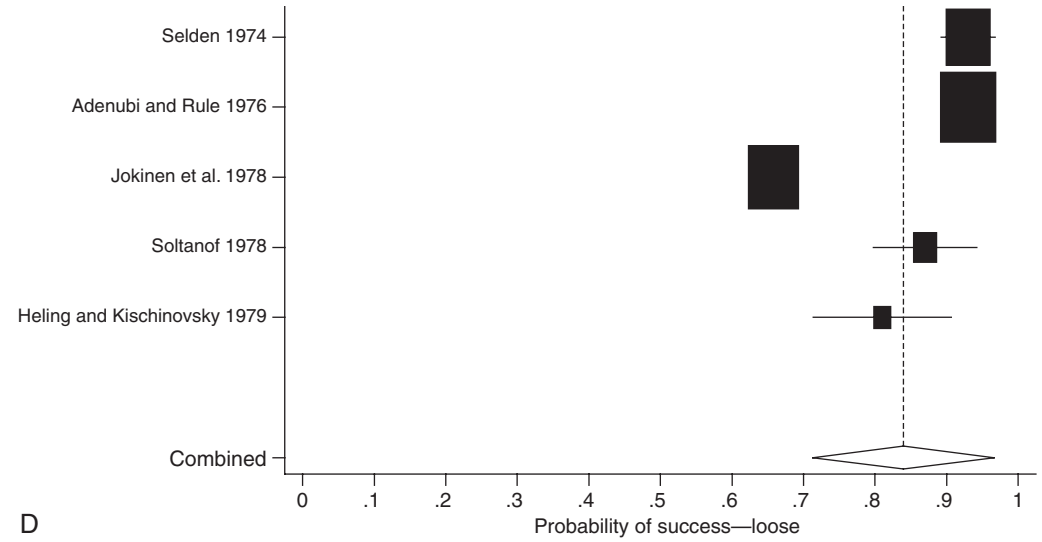
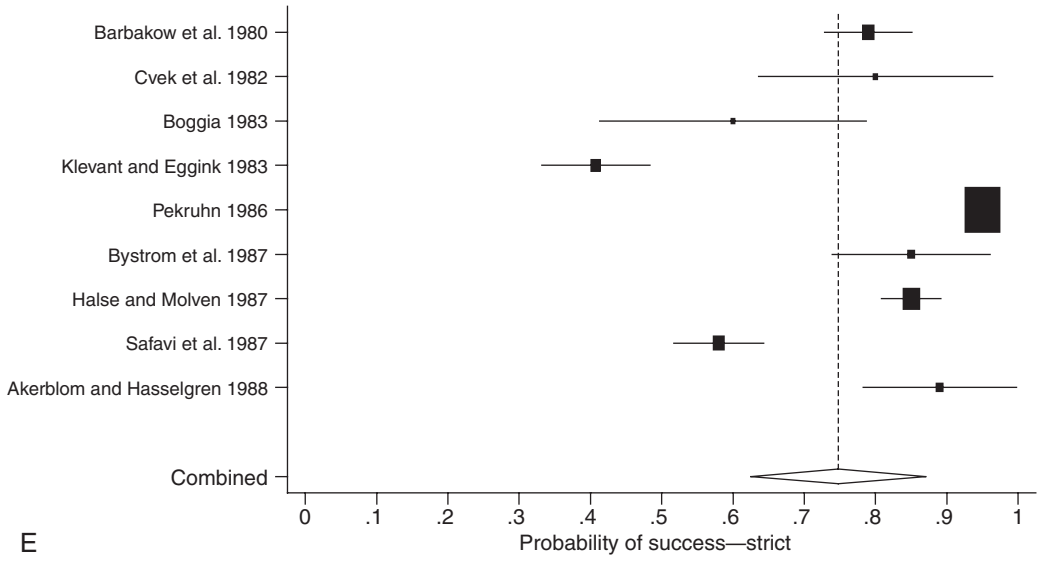


C

FIG. 11-25 Forest plot showing pooled and individual study's probability of periapical health for teeth undergoing root canal treatment by "decade of publication" and "criteria for success."



D



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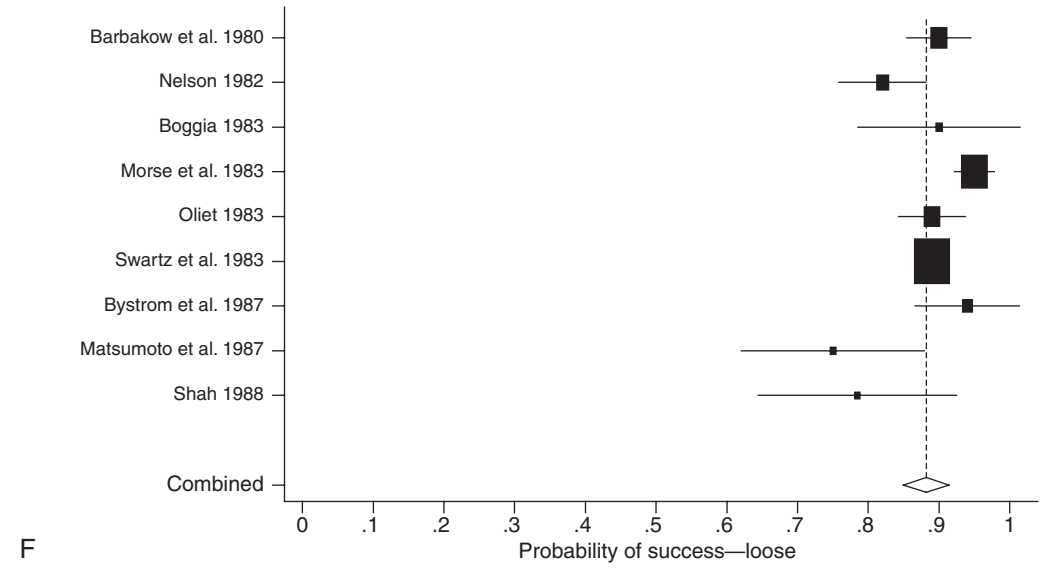

F

FIG. 11-25, cont'd

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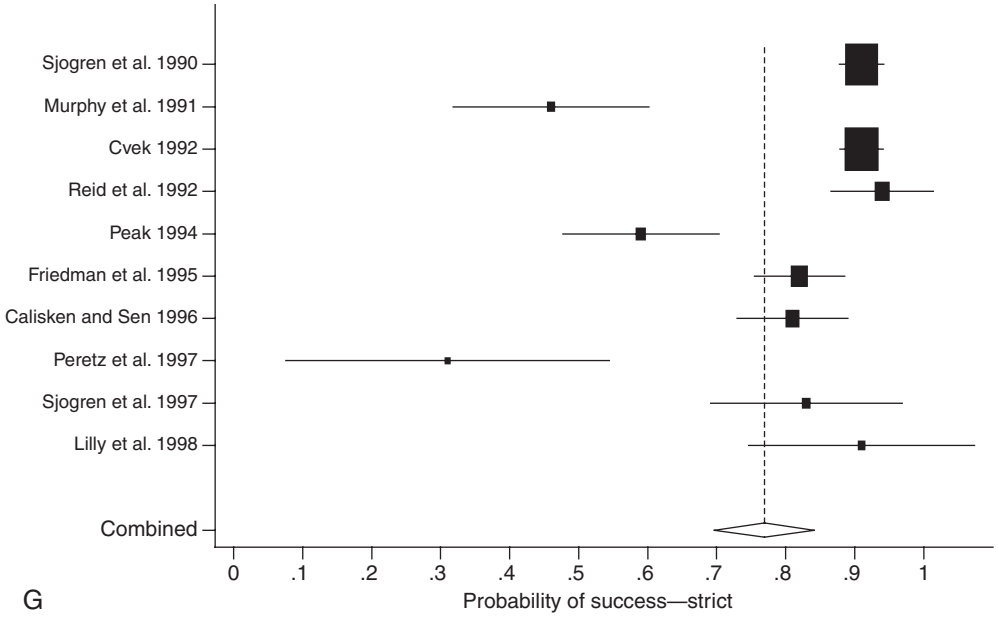

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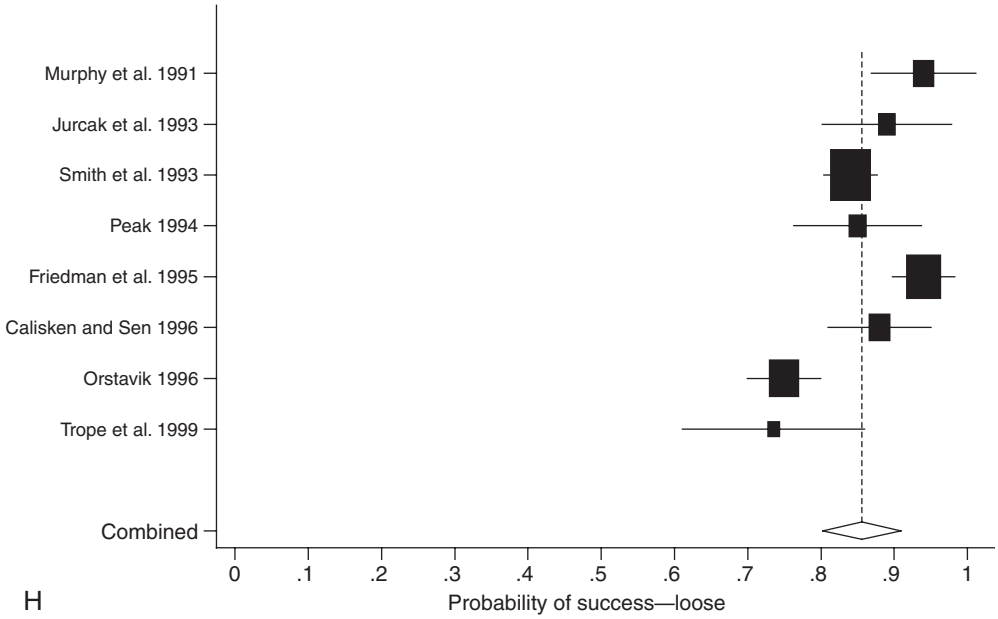


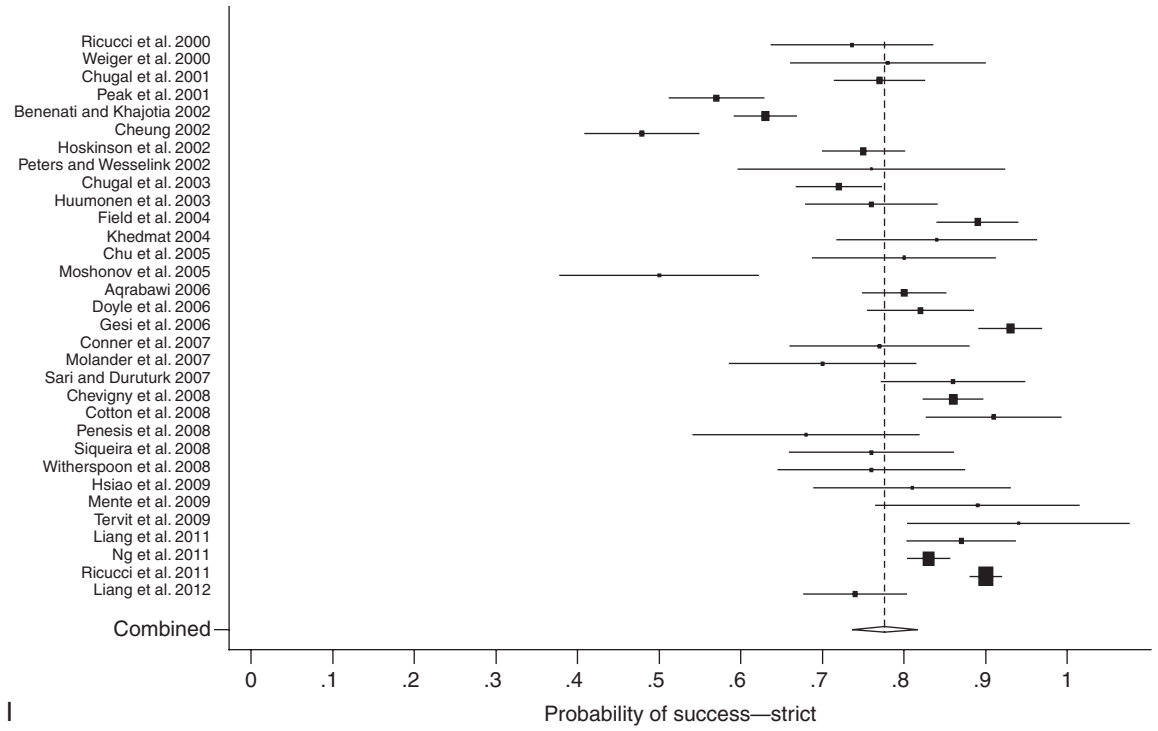
H

FIG. 11-25, cont'd

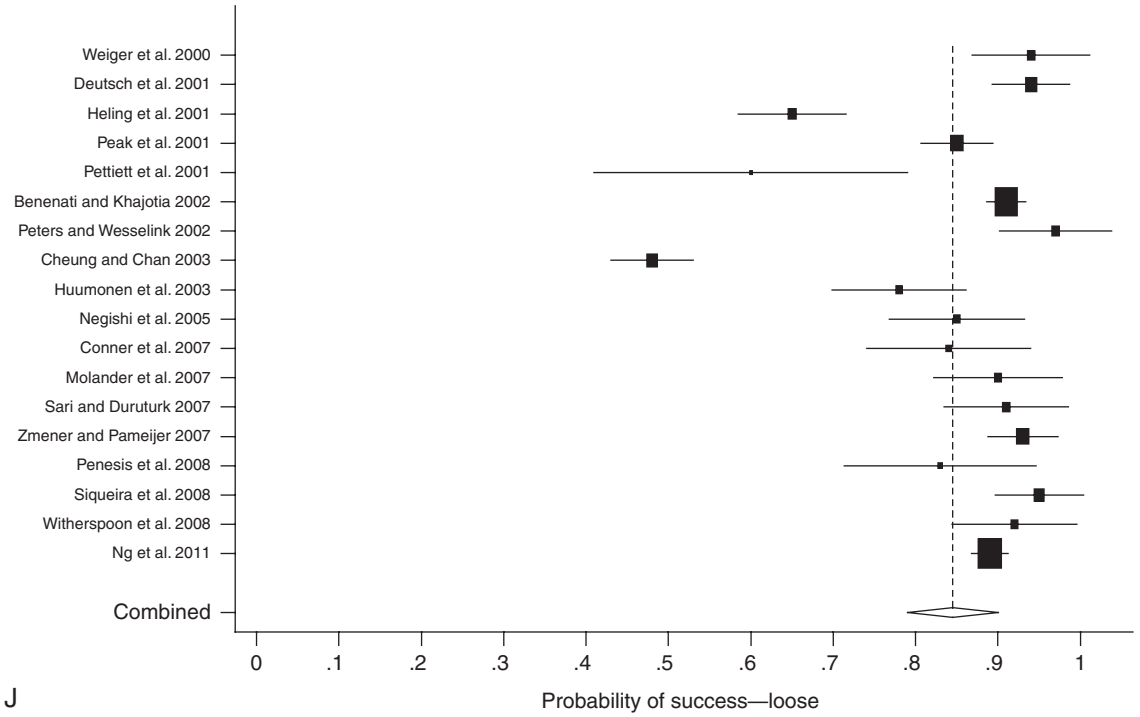
the report by Mindiola and associates,[135] whereas the influence of steroid therapy had never been reported previously. It may be argued that patients with diabetes are more susceptible to periodontal disease[74] or have a lower success rate of root canal treatment[67] because of being immunologically compromised. However, the researchers reported that over 50% of such teeth were extracted due to persistent pain. Some of these observations may be explained by the presence of neuropathy, a debilitating painful complication of diabetes.[57] It is interesting to note that systemic steroid therapy is often prescribed to control such chronic pain.[39,51,101]

Tooth Morphologic Type and Location

Tooth types (i.e., location within the arch) may vary with respect to their susceptibility to tooth fracture, a common reason for tooth loss after endodontic treatment. Ng and coworkers[149] found that tooth type had a significant influence on survival. Maxillary premolars and mandibular molars were found to have the highest frequency of extraction, with tooth fracture being the most common reason. The observation is consistent with previous reports on fracture incidence of maxillary premolars and mandibular molars.[56,109] The factors "proximal contacts" and "terminal (last standing) teeth" were found to affect tooth survival considerably,[149,151] but they were significantly correlated with molar teeth. Most of the extractions of terminal teeth or teeth with one or fewer proximal contacts were due to tooth fracture.[151] The observation may be explained by the unfavorable distribution of occlusal forces and higher nonaxial stress on terminal teeth and those with fewer than two proximal contacts. Other possible reasons for



I



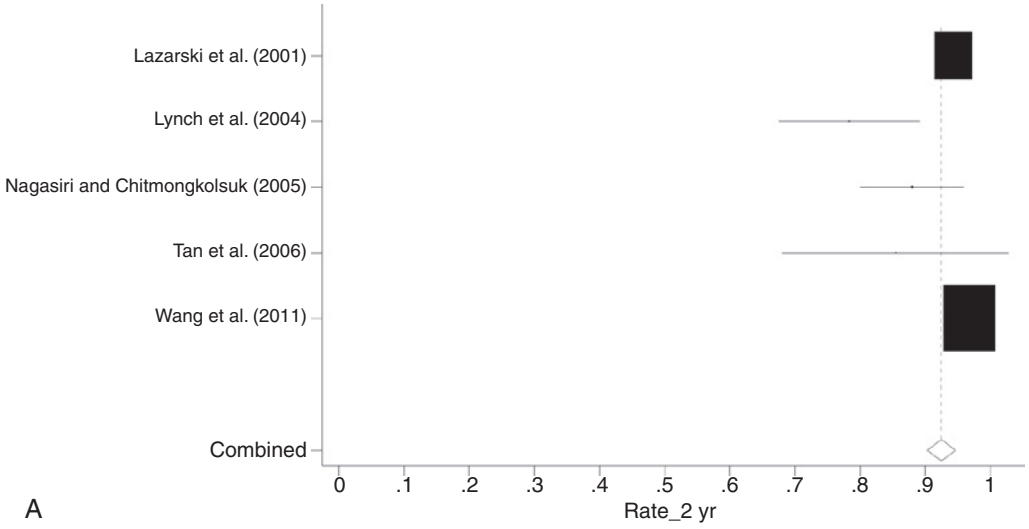
J

FIG. 11-25, cont'd

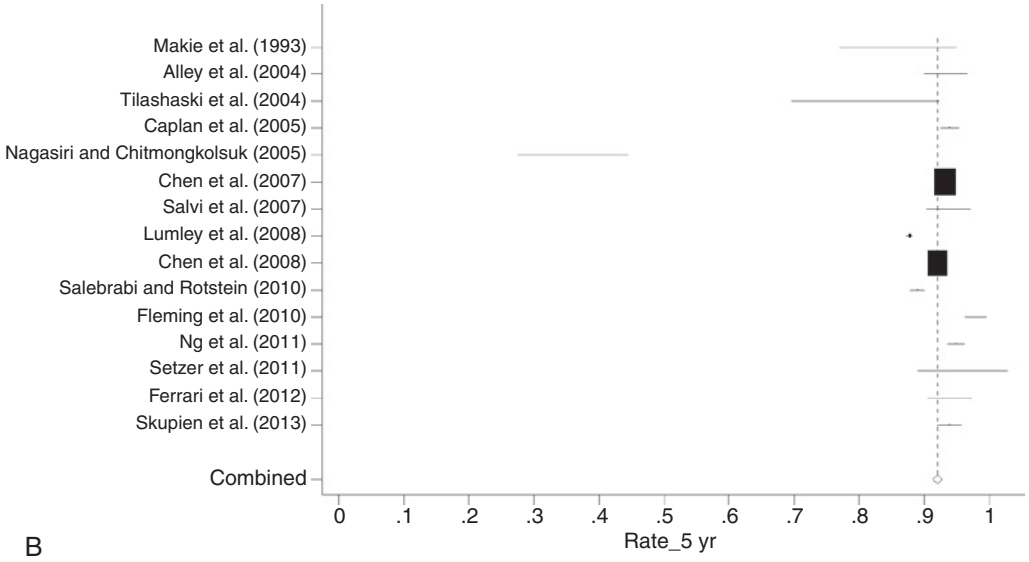
their higher rate of loss are (1) failure of root canal treatment in a terminal tooth may be accepted more willingly because of little perceived aesthetic value and (2) clinicians may be less likely to offer further treatment on terminal molar teeth due to difficult access. Therefore, when restoring molar teeth, the favorable distribution of occlusal forces must be considered, especially on teeth with one or fewer adjacent teeth or on terminal teeth.

Preoperative Conditions of Teeth

The presence of preoperative periapical lesions, which is the most significant prognostic factor for periapical healing, was found to have no significant influence on tooth survival.[151] On the other hand, the presence of preoperative periodontal probing defects of endodontic origin, preoperative pain, and preoperative sinus tracts were found to reduce tooth survival.[151] These observations are consistent with a previous



A



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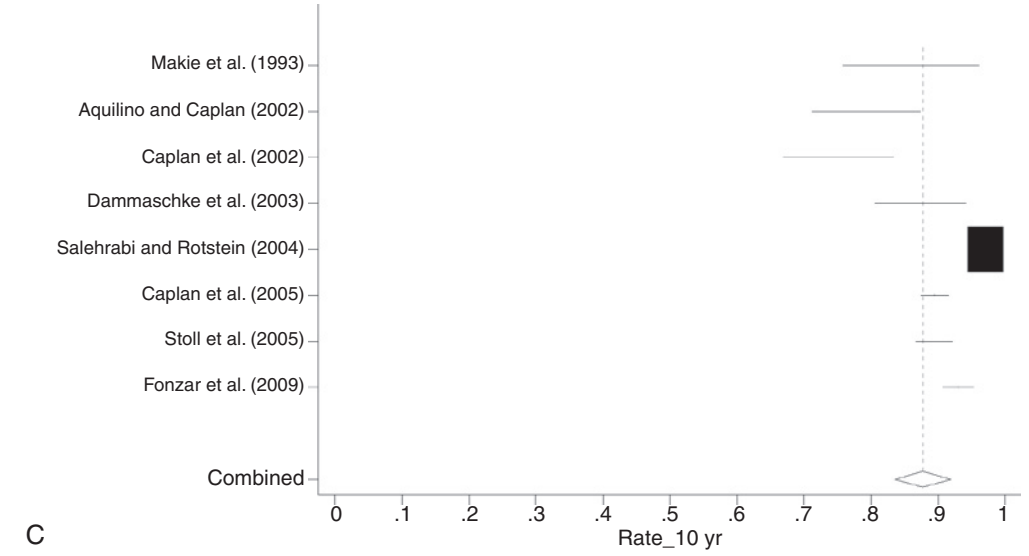

C

FIG. 11-26 Forest plot showing pooled and individual study's probability of 2-year (pooled probability = 0.93; 95% CI: 0.90, 0.95) **(A)**, 5-year (pooled probability = 0.92; 95% CI: 0.91, 0.93) **(B)**, and 10-year (pooled probability = 0.88; 95% CI: 0.84, 0.92) **(C)** survival of teeth undergoing root canal treatment.

report that the mere presence of a persistent periapical lesion was not a sufficient reason for dentists and patients to opt for further treatment, either retreatment or extraction.[180] The negative impact of preoperative pain on survival outcome highlights the importance of accurate pain diagnosis. In some instances, the pain may have been of nonendodontic origin and therefore would persist after treatment.[176] In other instances, preoperative pain of endodontic origin may persist following treatment, as a result of peripheral or central sensitization. Therefore, effective pain diagnosis and management for patients presenting with preoperative pain are crucial.

The presence of preoperative cervical resorption and perforation was also found to significantly reduce tooth survival.[151] This was as expected because tooth fracture and reinfection due to leakage through the resorption and perforating defects are likely sequelae in such cases. In the presence of reinfection, clinicians are more inclined to suggest extraction due to the intuitive perception of poor long-term prognosis of such teeth.

Treatment Factors

Considering all of the intraoperative factors, the "lack of patency at apical foramen" and the "extrusion of gutta-percha root filling" were found to be the most significant intraoperative factors in reducing tooth survival.[151] In the presence of persistent problems and knowing that the treatment objective of cleaning to the canal terminus could not be achieved, patients and dentists may be more likely to opt for extraction sooner than later.

Restorative Factors

Protection of teeth with crowns or cast restorations has not been shown to influence periapical healing; however, the placement of good cores had a positive effect on endodontic outcome. In contrast, placement of crowns or cast restorations was found to improve tooth survival.[149,151,189] This suggests that crowns and cast restorations help prevent tooth fracture, whereas the mere placement of a satisfactory core is sufficient to prevent reinfection after endodontic treatment. Unfortunately, the study was not able to investigate the interrelationship between tooth morphologic type, the extent of tooth tissue loss after treatment, or the type of final restoration. Although the clinical inference from these findings is that cast restorations should preferably be placed on all teeth after root canal treatment, this is probably a gross exaggeration of the true need. On the basis of laboratory[178] and clinical findings,[144] posterior teeth with compromised marginal ridges (mesial or distal), together with heavy occlusal loading evidenced by faceting, may benefit from full coverage restorations. The restoration design should attempt to preserve as much remaining tooth tissue as possible; the implication is that the so-called nonaesthetic but technically demanding partial veneer onlays and partial coverage crowns would be the restorations of choice for root-treated teeth. In anterior teeth, the missing tooth tissue may often be adequately replaced with composite restorative materials. A crown is only indicated when tooth structure or aesthetics become compromised.

The use of cast post and cores for retention of restorations have also been found to reduce tooth survival.[149,151] It may be speculated that the presence of a post has different effects on anterior versus posterior teeth as they are subjected to different directions and amount of occlusal force. It has been reported that only 12% of the extracted teeth with cast post and cores were incisors or canines. Therefore, the inference is that the use of such retention should be particularly avoided in premolar and molar teeth. Alternative treatment options should be considered for molar or premolar teeth lacking sufficient tooth structure.

Ng and colleagues[151] observed that teeth functioning as prosthetic abutments had poorer survival rates; however, the number of teeth (n = 94) in the study that functioned as abutments was too small a sample to be considered statistically significant. As previously described, the explanation may reside in the excessive and unfavorable distribution of occlusal stresses on abutment teeth. If possible, root-treated teeth should be avoided as abutments for prostheses or in provision of occlusal guidance in excursive movements.

Summary of Factors Influencing the Survival of Teeth Following Root Canal Treatment

The following conditions have been found to significantly improve tooth survival following root canal treatment:

- Nonmolar teeth (Fig. 11-27)
- Teeth with both mesial and distal adjacent teeth (Fig. 11-28)
- Teeth not located as the distal-most tooth in the arch[151]
- Teeth (molar) with cast restorations after treatment (Fig. 11-29)
- Teeth not requiring cast post and core for support and retention of restoration[111,151]
- Teeth not functioning as abutments for fixed prosthesis[3,111,191,151]
- Absence of preoperative deep periodontal probing defects, pain, sinus tract, or perforation[151]
- Achievement of patency at canal terminus and absence of root-filling extrusion during treatment[151]

In addition, it is important to ensure favorable distribution of occlusal forces when designing restorations for endodontically treated teeth.

Impact of Root Canal Treatment on Quality of Life

The impact of root canal treatment on the oral health–related quality of life of patients has been evaluated using the short form (OHIP-14) or modified version (OHIP-17) of the Oral Health Impact Profile (OHIP-14)[219] (Table 11-10). The distinctly positive impact of root canal treatment was apparent, regardless of cultural background of the patient group or the measure used.[55,73,87,115] As expected, physical pain, psychological discomfort (feeling tense), and disability (difficulty in relaxing) were the most improved domains following treatment.

OUTCOME OF NONSURGICAL RETREATMENT

When root canal treatment fails to resolve periapical disease, it is often considered appropriate to retreat the tooth using conventional approaches first, especially when the previous treatment is technically deficient (Fig. 11-30). This requires removal of the previous root-filling material and any other material placed for restorative reasons. Correction of any procedural errors may also be required, if possible. All materials must be removed in their entirety to ensure delivery of

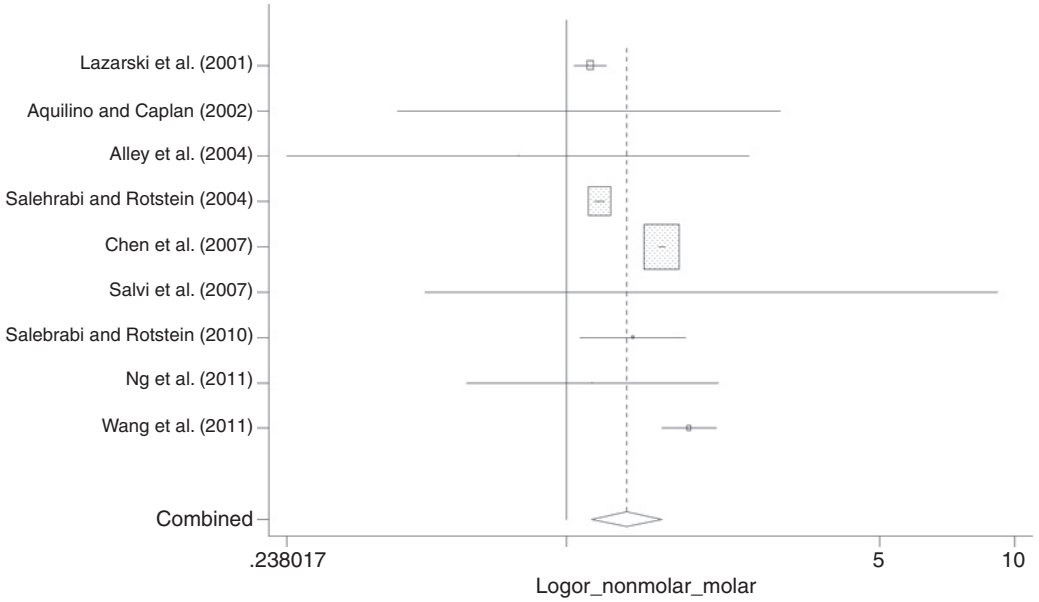

FIG. 11-27 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for nonmolar versus molar teeth (pooled OR = 1.4; 95% CI: 1.1, 1.6).

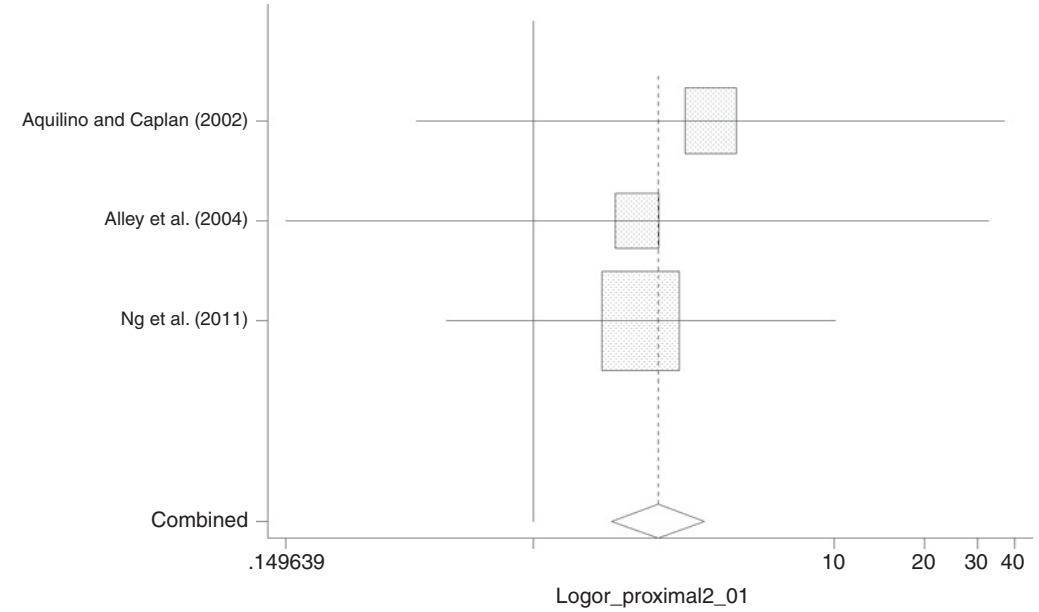

FIG. 11-28 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for teeth with both mesial and distal contacts present versus absent (pooled OR = 2.6; 95% CI: 1.8, 3.7).

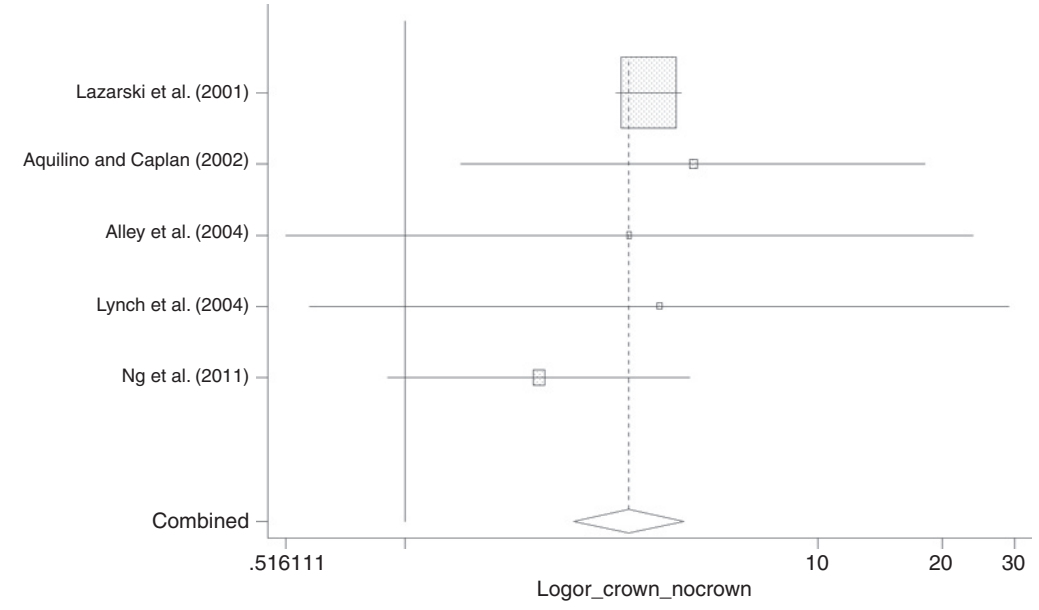

FIG. 11-29 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for teeth restored with cast versus plastic restoration (pooled OR = 3.5; 95% CI: 2.6, 4.7).

TABLE 11-10

The OHIP-14 and Modified Oral Health Impact Profile Instrument (OHIP-17) (Dugas et al., 2002)

OHIP #	Item Question
OHIP1	Have you had trouble pronouncing words because of your teeth and mouth?
OHIP2	Have you felt that your sense of taste has worsened because of your teeth or mouth?
OHIP3	Have you had painful aching in your mouth?
OHIP4	Have you found it uncomfortable to eat any foods because of your teeth or mouth?
OHIP5*	Have you had to alter the temperature of the foods that you eat because of your teeth or mouth?
OHIP6	Have you been self-conscious because of your teeth or mouth?
OHIP7	Have you felt tense because of your teeth or mouth?
OHIP8	Has your diet been unsatisfactory because of your teeth or mouth?
OHIP9	Have you had to interrupt meals because of your teeth or mouth?
OHIP10	Have you found it difficult to relax because of your teeth or mouth?
OHIP11*	Have you found it difficult to fall asleep because of your teeth or mouth?
OHIP12*	Have you ever been awakened by problems with your teeth or mouth?
OHIP13	Have you been embarrassed because of your teeth or mouth?
OHIP14	Have you been irritable with other people because of your teeth or mouth?
OHIP15	Have you had difficulty doing your usual jobs because of problems with your teeth or mouth?
OHIP16	Have you felt that life in general was less satisfying because of your teeth or mouth?
OHIP17	Have you been totally unable to function because of your teeth or mouth?

OHIP-14 contains all the items except those with an asterisk (*).

antibacterial agents to all surfaces of the root canal dentin (see Fig. 11-30). The periapical healing rates of root canal retreatment are generally perceived to be lower compared to primary treatment for the following reasons:

- Obstructed access to the apical infection
- A potentially more resistant microbiota

The outcomes from a range of studies show that the mean weighted success rate of nonsurgical root canal retreatment is 66% (Figs. 11-31 and 11-32), which is about 6% lower than in the case of primary treatment on teeth with apical periodontitis.[148,150] However, it has also been shown that the survival rate of teeth having undergone nonsurgical root canal retreatment may be similar to that for primary root canal treatment.[151]

The factors affecting outcomes of periapical health and tooth survival following root canal retreatment are otherwise identical to those affecting primary root canal treatment. Of the potential prognostic factors unique to retreatment cases, the most significant factor influencing the outcome of treatment is the ability to remove or bypass preexisting root-filling material or separated instruments during retreatment. This is understandable because it would have a direct impact on the ability to achieve canal patency and canal disinfection at the apical terminus.[150]

OUTCOME OF SURGICAL RETREATMENT

Factors Affecting Periapical Health or Healing Following Periapical Surgery and Root-End Filling

There are a number of published systematic reviews on the prognostic factors for periapical surgery with apical filling.[45,47,48,155,204,205,239-241,250] The major limitations of this pooled data include the variable length of time for evaluating success following treatment as well as the radiographic criteria used to assess healing. An unpublished meta-analysis[130] of data from the studies listed in Table 11-11 reveals that the weighted pooled probability of success from periapical surgery with retrograde restorations, based on complete radiographic healing, is 67.5% (95% CI: 62.9%, 72.0%) (Fig. 11-33). The trend shows higher success rates reported in more recent studies. This observation is consistent with the much higher pooled success rate of 92% (95% CI: 86%, 95%) revealed in the meta-analysis of prospective outcome data of surgical endodontic treatments performed by a "modern" technique (using magnification, root-end resection with minimal or no bevel, retrograde cavity preparation with ultrasonic tips, and modern retrograde root canal filling materials).[240,241] In congruence with these findings, Setzer and colleagues reported that the pooled success rate of treatment using a microsurgical approach (94%; 95% CI: 89%, 98%) was more favorable than traditional root-end surgery (59%; 95% CI: 55%, 63%).[205] However, the studies included in the latter meta-analysis differed in design, case selection, duration after treatment, and the provision of preoperative nonsurgical treatment, which may bias the higher success rate of the contemporary treatment.

The unpublished meta-analysis[130] stratified by duration following treatment reveals that the pooled probability of success based on complete healing plateaued after 2 years postoperatively, being 51% (95% CI: 42%, 60%) after 6 months, 68% (95% CI: 63%, 73%) after 12 months, 76% (95% CI: 67%, 84%) after 24 months, 74% (95% CI: 52%, 95%) after 48 months, and 74% (95% CI: 66%, 82%) after more than 48 months. It is therefore advisable to follow up on cases that have undergone periapical surgery for a minimum period of 2 years and up to 4 years as suggested in quality guidelines.[61]

The factors having a major impact on outcome of periapical surgery with retrograde cavities and fillings in teeth with preoperative periapical radiolucencies revealed in the (as yet) unpublished meta-analyses[130] of data from studies listed in Table 11-11 are as follows:

- Small (≤ 5 mm) versus large (> 5 mm) periapical lesion (risk ratio = 1.2; 95% CI: 1.1, 1.3)
- Periapical lesion involving one versus both cortical plates (risk ratio = 1.2; 95% CI: 1.0, 1.5)
- Absence versus presence of previous surgery (risk ratio = 1.2; 95% CI: 1.1, 1.3)

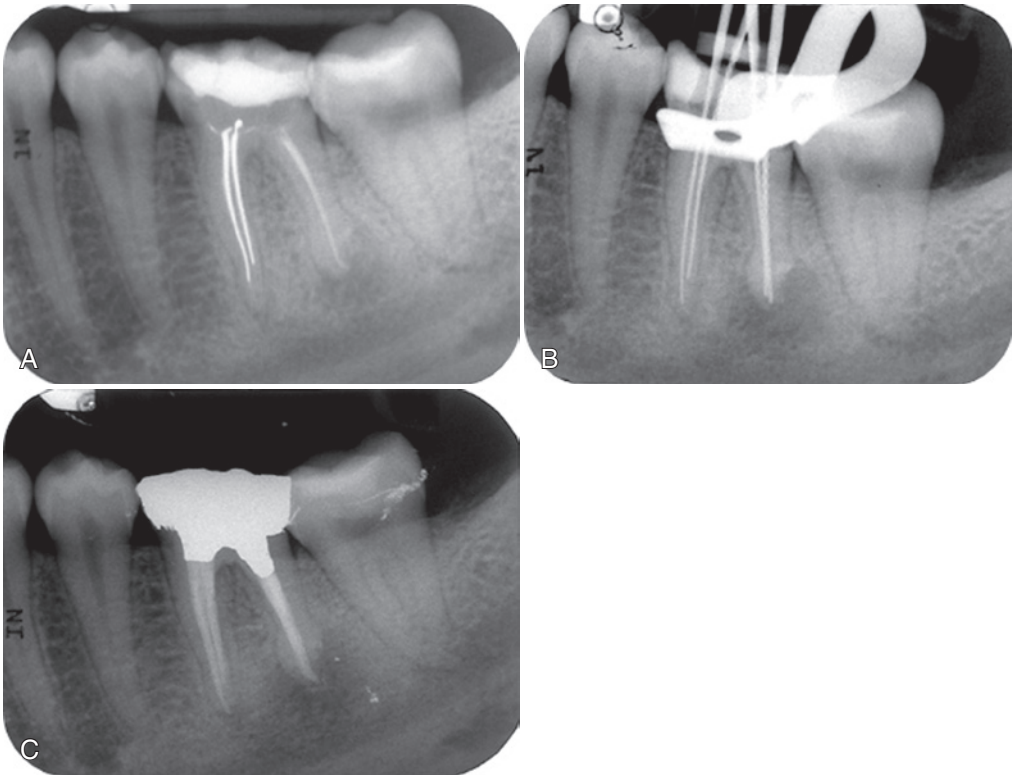

FIG. 11-30 Tooth with technically deficient root canal treatment (A); having undergone (B and C) root canal retreatment.

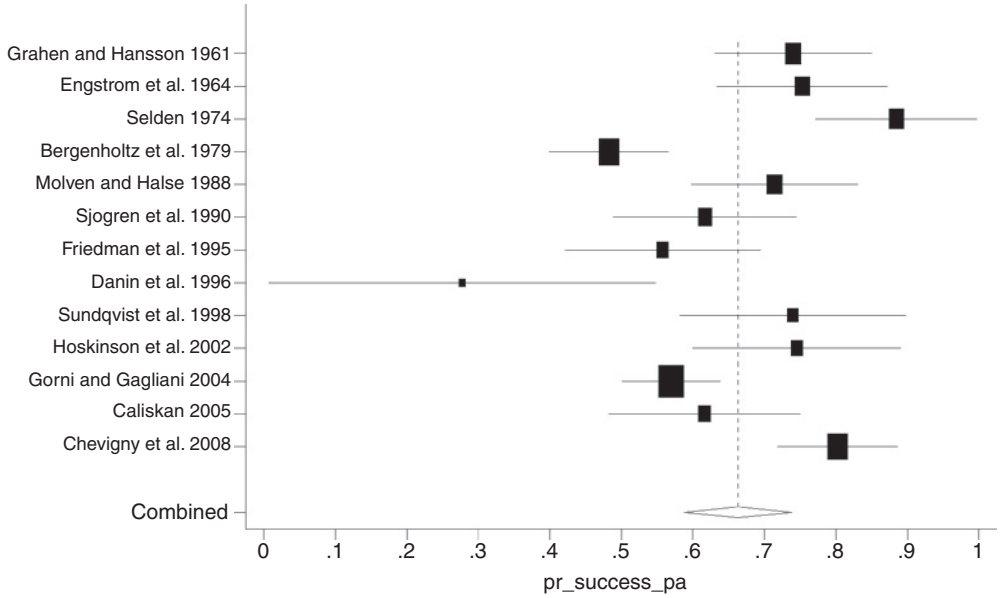

FIG. 11-31 Forest plot showing pooled and individual study's probability of complete periapical healing following root canal retreatment.

- Using magnification versus without the use of magnification during surgery (risk ratio = 1.5; 95% CI: 1.3, 1.8)
- Root-end resection with minimum versus obvious bevel (risk ratio = 1.3; 95% CI: 1.2, 1.4)
- Use of ultrasonic tip versus bur for retro-cavity preparation (risk ratio = 1.3; 95% CI: 1.2, 1.4)
- Use of retro-filling material with mineral trioxide aggregate (MTA) cement, super ethoxybenzoic acid (EBA) cement, or intermediate restorative material (IRM) versus amalgam; the use of MTA resulted in a similar outcome to SuperEBA (risk ratio = 1.0; 95% CI: 0.99, 1.1) or IRM (risk ratio = 1.1; 95% CI: 0.98, 1.1); SuperEBA and IRM were associated with significantly higher chances of success than amalgam (risk ratio = 1.2; 95% CI: 1.1, 1.3) as the retro-filling material

The preoperative presence of signs or symptoms (risk ratio = 1.2; 95% CI: 1.1, 1.3),[249] periodontal status (risk ratio = 2.1;

TABLE 11-11

Studies Investigating Periapical Healing Following Apical Surgery

Author	Design	Examination	Type of Radiograph	Radiographic Criteria for Success	Sample Size			Duration after Treatment (months)
					Patients	Teeth	Roots	
Harty et al., 1970	Retrospective	C & R	Pa	Other		169		6-60
Nordendram, 1970	Prospective	C & R	Pa	Rud et al., 1972	66	66		6-24
Rud et al., 1972	Prospective	R	Pa	Rud et al., 1972		237		12-180
Finne et al., 1977	Prospective	C & R	Pa	Persson, 1973	156	218		36
Hirsch et al., 1979	Prospective	C & R	Pa	Rud et al., 1972	467	467		6-36
Ioannides & Borstlap, 1983	Retrospective	C & R	Pa	Other	50	50	45	6-60
Allen et al., 1989	Retrospective	C & R	Pa	Rud et al., 1972		175		12-60
Amagasa et al., 1989	Prospective	C & R	Pa	Other	42	64		12-90
Dorn & Gartner, 1990	Retrospective	R	Pa	Other		488		6-120
Grung et al., 1990	Prospective	C & R	Pa	Rud et al., 1972		161		12-96
Lustmann et al., 1991	Retrospective	C & R	Pa	Rud et al., 1972			123	6-96
Rapp et al., 1991	Retrospective	R	Pa	Rud et al., 1972	331	226		6-24
Rud et al., 1991	Retrospective	C & R	Pa	Rud et al., 1972	388	388		12
Waikakul et al., 1991	Prospective	C & R	Pa	Other	34	62		6-24
Pantschev et al., 1994	Prospective	C & R	Pa	Persson, 1973	79	103		36
Jesselen et al., 1995	Prospective	C & R	Pa	Other	67	82		12-60
Rud et al., 1996	Prospective	R	Pa	Rud et al., 1972			347	12-48
Sumi et al., 1996	Retrospective	C & R	Pa	Other	86	157		6-36
Jansson et al., 1997	Retrospective	C & R	Pa	Other	59	59		11-16
Testori et al., 1999	Retrospective	R	Pa	Rud et al., 1972	130	181	302	12-72
Von Arx & Kurt, 1999	Prospective	C & R	Pa	Von Arx & Kurt, 1999	38	43		12
Zuolo et al., 2000	Prospective	C & R	Pa	Molven et al., 1987	106	102		12-48
Pecora et al., 2001	RCT	R	Pa	Rud et al., 1972	20	20		6
Penarrocha et al., 2001	Retrospective	R	Pa	Von Arx & Kurt, 1999	30	31	71	12
Rahbaran et al., 2001	Retrospective	C & R	Pa	Other	154	154		48-108
Rud et al., 2001	Prospective	C & R	Pa	Rud et al., 1972		520	834	6-150
Von Arx et al., 2001	Prospective	C & R	Pa	Other	24	25	39	12
Jensen et al., 2002	RCT	C & R	Pa	Rud et al., 1972		122		
Rubinstein & Kim, 2002	Prospective	C & R	Pa	Rud et al., 1972	52	59	59	68
Tobon et al., 2002	RCT	R	Pa	Rud et al., 1972	25	26		12
Vallecillo et al., 2002	Prospective	R	Pa	Other	29	29		12
Chong et al., 2003	RCT	C & R	Pa	Molven et al., 1987	86	86		12-24
Maddalone & Gagliani, 2003	Prospective	C & R	Pa	Molven et al., 1987	79	120		3-36
Schwartz-Arad et al., 2003	Prospective	R	Pa	Other	101	122		6-45
Platt et al., 2004	RCT	C & R	Pa	Molven et al., 1987	28	34		12
Gagliani et al., 2005	Prospective	C & R	Pa	Rud et al., 1972	164	168	231	60
Lindeboom et al., 2005	RCT	C & R	Pa	Rud et al., 1972	100	100		12
Marti-Bowen et al., 2005	Retrospective	C & R	DPT	Von Arx & Kurt, 1999	52	71	95	6-12
Taschieri et al., 2005	Prospective	C & R	Pa	Rud et al., 1972	32	46		12
Taschieri et al., 2007	Prospective	C & R	Pa	Molven et al., 1987	28	28		12
Tsesis et al., 2006	Retrospective	C & R	Pa	Rud et al., 1972	71	88		6-48
Marin-Botero et al., 2006	RCT	C & R	Pa	Rud et al., 1972	30	30		12

Continued

TABLE 11-11

Studies Investigating Periapical Healing Following Apical Surgery—cont'd

Author	Design	Examination	Type of Radiograph	Radiographic Criteria for Success	Sample Size			Duration after Treatment (months)
					Patients	Teeth	Roots	
Taschieri et al., 2006	RCT	C & R	Pa	Molven et al., 1987	53	71		12
De Lange et al., 2007	RCT	C & R	Pa	Rud et al., 1972	290	290		12
Leco-Berrocal et al., 2007	Prospective	R	Pa and DPT	Other	45	45		6-24
Penarrocha et al., 2007	Prospective	C & R	Pa	Von Arx & Kurt, 1999	235	333	384	6-144
Taschieri et al., 2007a	Prospective	C & R	Pa	Molven et al., 1987	17	27		12
Taschieri et al., 2007b	Prospective	C & R	Pa	Molven et al., 1987	41	59		12
Von Arx et al., 2007	Prospective	C & R	Pa	Rud et al., 1972	200	177		12
Walivaara et al., 2007	Prospective	C & R	Pa	Rud et al., 1972	54	55		12
Garcia et al., 2008	Prospective	C & R	DPT	Von Arx & Kurt, 1999	92	106	129	6-12
Kim et al., 2008	Prospective	C & R	Pa	Molven et al., 1987		148		24
Penarrocha et al., 2008	Prospective	C & R	DPT	Von Arx & Kurt, 1999	278	278		12
Taschieri et al., 2008b	Prospective	C & R	Pa	Molven et al., 1987	27	31		12
Taschieri et al., 2008a	RCT	C & R	Pa	Molven et al., 1987	61	100		25
Christiansen et al., 2009	RCT	C & R	Pa	Molven et al., 1987		25		12
Dominiak et al., 2009	Retrospective	C & R	Pa	Other	106	106		12
Ortega-Sanchez et al., 2009	Retrospective	C & R	DPT	Von Arx & Kurt, 1999	30	30	37	
Pantschev et al., 2009	Retrospective	C & R	Pa	Other		147		12
Waalivaara et al., 2009	RCT	C & R	Pa	Molven et al., 1987	131	147		12
Barone et al., 2010	Prospective	C & R	Pa	PAI		129		48-120
Garcia-Mira et al., 2010	Retrospective	C & R	DPT	Von Arx & Kurt, 1999	75	87		12
Taschieri et al., 2010	Retrospective	C & R	Pa	Molven et al., 1987	76	112		48
Von Arx et al., 2010	Prospective	C & R	Pa	Rud et al., 1972		339		12
Goyal et al., 2011	RCT	C & R	Pa	Rud et al., 1972	25	25		12
Penarrocha et al., 2011	Retrospective	C & R	DPT	Von Arx & Kurt, 1999	150	178	178	12
Song et al., 2011a	Retrospective	C & R	Pa	Molven et al., 1987		441		3-12
Song et al., 2011b	Prospective	C & R	Pa	Molven et al., 1987	42	42		12
Taschieri et al., 2011	Retrospective	C & R	Pa	Molven et al., 1987	33	43		12-48
Waalivaara et al., 2011	RCT	C & R	Pa	Molven et al., 1987	153	194		12-21
Penarrocha et al., 2012	Prospective	C & R	DPT	Von Arx & Kurt, 1999	23	31		12-19
Song & Kim, 2012	RCT	C & R	Pa	Molven et al., 1987	192	192		12
Von Arx et al., 2012	Prospective	C & R	Pa	Rud et al., 1972		170		12-60
Kreisler et al., 2013	Prospective	C & R	Pa	Rud et al., 1972	255	281		6-12
Penarrocha et al., 2013	Retrospective	C & R	Pa and DPT	Von Arx & Kurt	96	139		6-12
Song et al., 2013a	Prospective	C & R	Pa	Molven et al., 1987		344		12-120
Song et al., 2013b	Prospective	C & R	Pa	Molven et al., 1987		135		12
Song et al., 2014	Retrospective	C & R	Pa	Rud et al., 1972		115		12-96
Taschieri et al., 2013	Retrospective	C & R	Pa	Molven et al., 1987		86		6-12
Villa-Machado et al., 2013	Retrospective	C & R	Pa	PAI	154	171		12-192
Li et al., 2014	Retrospective	C & R	Pa	Molven et al., 1987	82	101		48

RCT, randomized controlled trial; C, clinical; R, radiographic; Pa, periapical; DPT, dental pantomogram; PAI, periapical index.

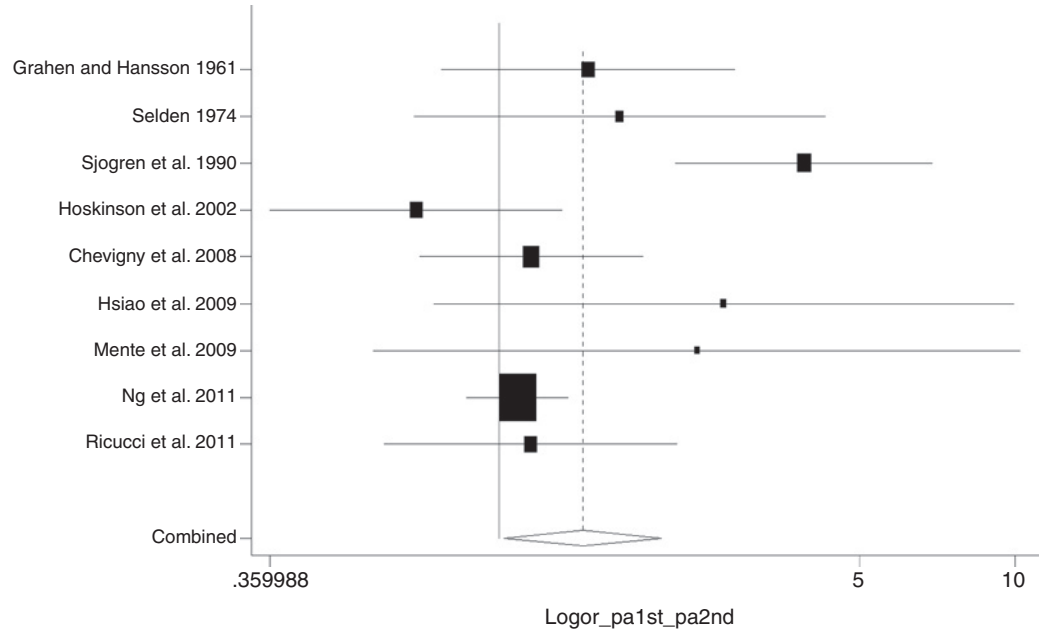

FIG. 11-32 Forest plot showing pooled and individual study's odds ratio of periapical health for teeth with preoperative periapical radiolucency undergoing primary root canal treatment versus root canal retreatment (pooled OR = 1.5; 95% CI: 1.0, 2.1).

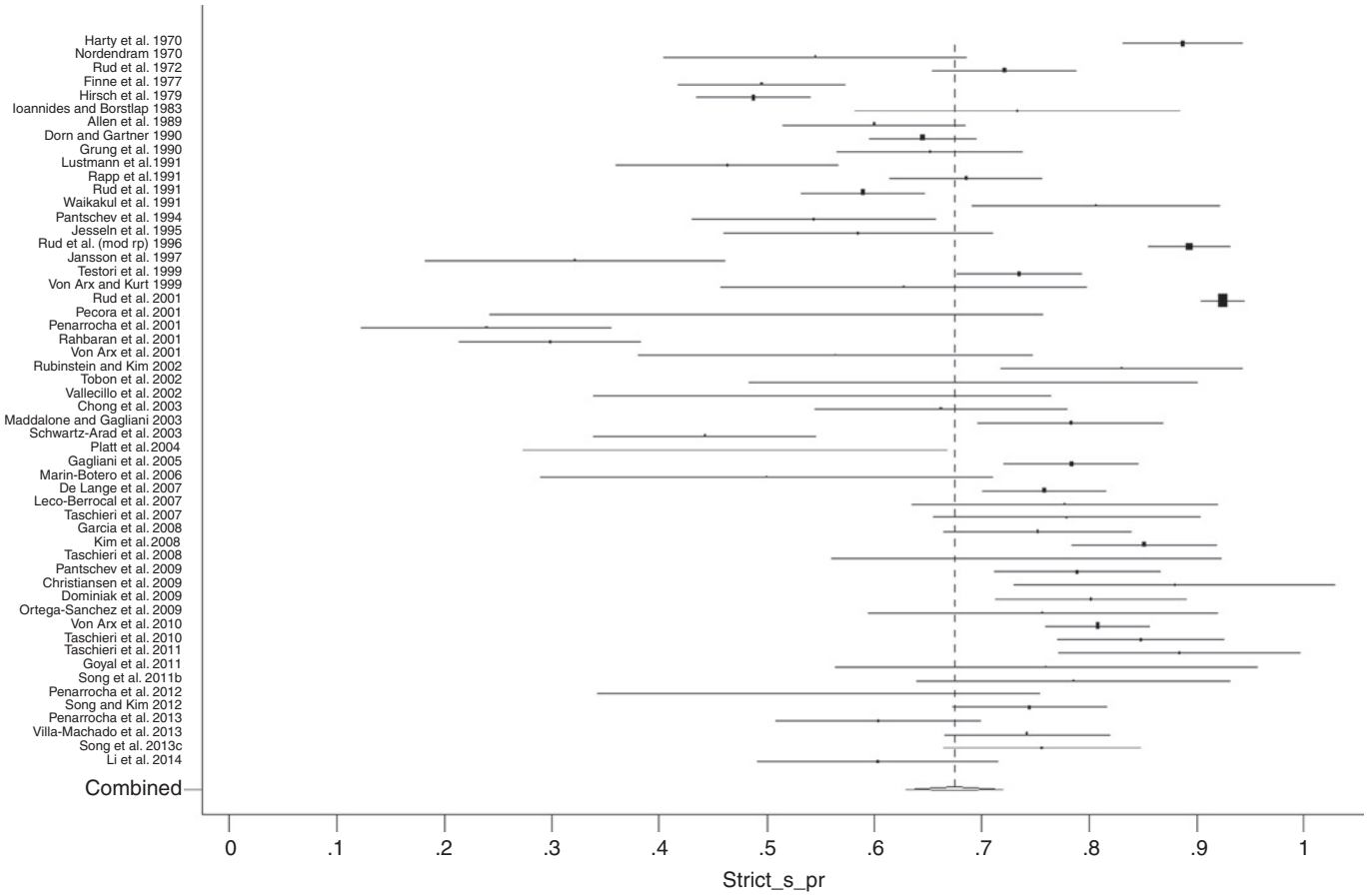

FIG. 11-33 Forest plot showing pooled and individual study's probability of complete periapical healing following apical surgery.

95% CI: 1.1, 3.8),[222] and quality of the coronal restoration (risk ratio = 1.6; 95% CI: 1.2, 2.1)[249] have also been revealed as significant prognostic factors by individual studies.

The following factors have minimal effect on surgical retreatment[43]:

- Age of patient
- Gender of patient
- General health of patient
- Tooth type
- Quality of the preexisting root canal filling, as judged radiographically
- Histologic diagnosis of the biopsied periapical lesion (cyst or granuloma)

The use of guided periodontal tissue regenerative membrane or grafting material has been advocated for cases with through-and-through defects (missing buccal and palatal cortical plates), but there are conflicting reports as to their benefits.[53,162,170,232-234,237,249] The unpublished meta-analyses[130] of data from these studies did not reveal a significant influence of such approaches on the periapical healing outcome.

Factors Affecting Periodontal Incisional Wound Healing

Periodontal attachment level after periapical surgery is an additional physical outcome measure compared to nonsurgical root canal treatment. Studies have compared the effect of different soft tissue incision techniques (intrasulcular with or without involving interproximal papilla, submarginal, papilla-base, etc.).[106,245-247] As expected, all concluded that negligible marginal recession could be achieved by adopting a flap design that avoided reflection of the interproximal papilla.

The improvement in outcomes of periapical surgery has been attributed to modern surgical techniques plus greater biologic awareness of clinicians.[205,250] In addition, and perhaps even more important, case selection may be more critical in excluding potential failures, inferring that prognostication may have improved.

Factors Affecting Tooth Survival Following Periapical Surgery and Root-End Filling

Unlike nonsurgical root canal treatment, the outcome measure of tooth survival had only been adopted by one study[253] on surgical cases at the time of this writing. The reported median survival time for first-time surgery was 92.1 months (95% CI: 40.9, 143.4) and that for resurgery was 39.1 months (95% CI: 6.1, 72.1).[253] The failure events, however, included tooth extraction and clinical and radiographic signs of periapical disease after treatment.

Impact of Periapical Surgery on Quality of Life

The impact of periapical surgery on the patient's quality of life has only been evaluated using a questionnaire including three domains: physical function (chewing, talking, sleeping, daily routine, and work), physical pain, and other physical symptoms (swelling, bleeding, nausea, bad taste/breath).[49] It was concluded that the papilla-base incision flap design resulted in a lower impact on physical pain and other symptoms within the first week postoperation. The impact on physical pain has also been explored by two other studies[35,36]; both reported the postoperative pain was of relatively short duration, with the intensity peaking at 3 to 5 hours postoperatively and progressively decreasing with time with no significant influencing factors identified.

CONCLUDING REMARKS

The procedures used to maintain pulp vitality and for prevention and treatment of periapical disease are able to achieve excellent outcomes. The outcome data and potential prognostic factors should be considered during treatment options appraisal and planning. Despite the fact that most important prognostic factors are beyond the control of clinicians, optimal outcomes for individual cases may still be achieved by performing the procedure to guideline standards. From a health and economic perspective, conventional root canal treatment is a highly cost-effective treatment as a first-line intervention to extend the life of the tooth with a preoperative periapical lesion.[103,171] If a root canal treatment subsequently fails, nonsurgical and surgical retreatments are also more cost-effective than replacement with a prosthesis.[103,171] Ultimately, all sources of evidence must be assessed for biasing influences based on the local cultures, expertise, treatment predilection, and funding sources.

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CHAPTER 12

Structure and Functions of the Dentin-Pulp Complex

INGE FRISTAD | ELLEN BERGGREEN[1]

CHAPTER OUTLINE

Morphologic Zones of the Pulp
The Pulp-Dentin Complex
Odontoblast Layer
Cell-Poor Zone
Cell-Rich Zone
Pulp Proper
Cells of the Pulp
Odontoblast
Odontoblast Process
Relationship of Odontoblast Structure to Secretory Function
Pulp Fibroblast
Macrophage
Dendritic Cell
Lymphocyte
Mast Cell
Metabolism
The Pulpal Interstitium And Ground Substance
Hyaluronan
Elastic Fibers
The Inflamed Interstitium
Connective Tissue Fibers of the Pulp
The Trigeminal System
Innervation
Steps and Mechanisms in Pain Perception
Detection: The First Step in Pain Perception
Neuropeptides
Pulp Testing
Sensitivity of Dentin
Peripheral Sensitization
Hyperalgesia and Allodynia
Inflammatory Mediators
Painful Pulpitis
Plasticity of Intradental Nerve Fibers
Tissue Injury and Deafferentation
Processing: The Second Step in Pain Perception
Perception: Thalamus to Cortex
Vascular Supply
Regulation of Pulpal Blood Flow
Fluid Drainage
Circulation in the Inflamed Pulp
Pulpal Repair
Pulpal Calcifications
Age Changes

MORPHOLOGIC ZONES OF THE PULP

The Pulp-Dentin Complex

The dental pulp and dentin function as a unit, and the odontoblasts represent a crucial element in this system. The odontoblasts are located in the periphery of the pulp tissue, with extensions into the inner part of dentin. Dentin would not exist unless produced by odontoblasts, and the dental pulp is dependent on the protection provided by the dentin and enamel. Likewise, integrated dynamics of the pulp-dentin complex imply that impacts on dentin may affect the pulpal components and that disturbances in the dental pulp will in turn affect the quantity and quality of the dentin produced.

[1]The authors acknowledge the outstanding work of Drs. Henry Trowbridge, Syngcuk Kim, Hideaki Suda, David H. Pashley, and Fredrick R. Liewehr in previous editions of this text. The present chapter is built on their foundational work.

Odontoblast Layer

The outermost stratum of cells of the healthy pulp is the odontoblast layer (Figs. 12-1 and 12-2). This layer is located immediately subjacent to the predentin. The odontoblast processes, however, pass on through the predentin into the inner part of dentin. Consequently, the odontoblast layer is actually composed of the cell bodies of odontoblasts. In addition, capillaries, nerve fibers, and dendritic cells may be found among the odontoblasts.

In the coronal portion of a young pulp that is actively secreting collagen, the odontoblasts assume a tall columnar form.[62] The odontoblasts vary in height; consequently, their nuclei are not all at the same level and are aligned in a staggered array, often described as a palisade appearance. This organization makes the layers appear to be three to five cells in thickness even though there is only one actual layer of odontoblasts. Between adjacent odontoblasts there are small intercellular

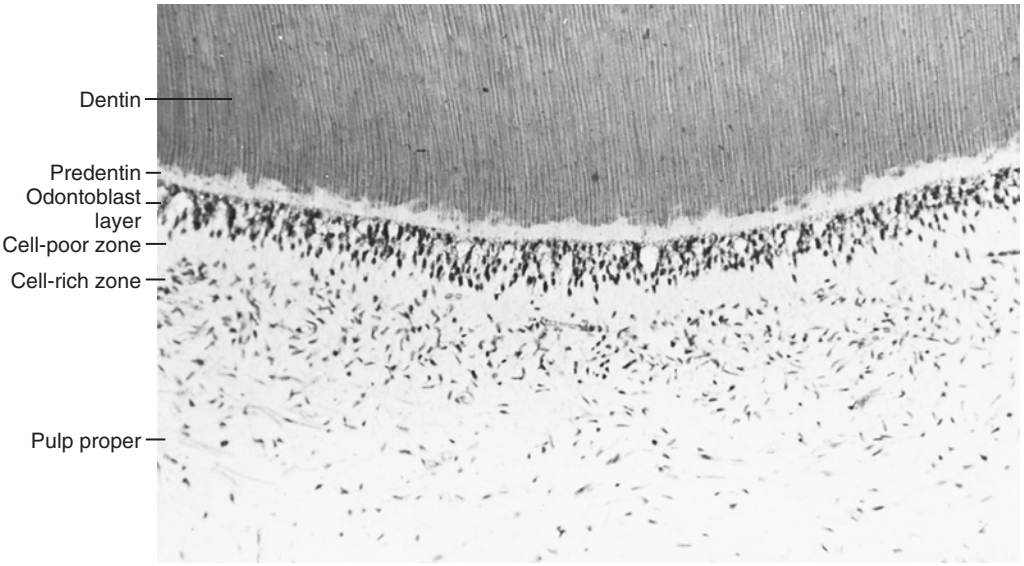

FIG. 12-1 Morphologic zones of the mature pulp.

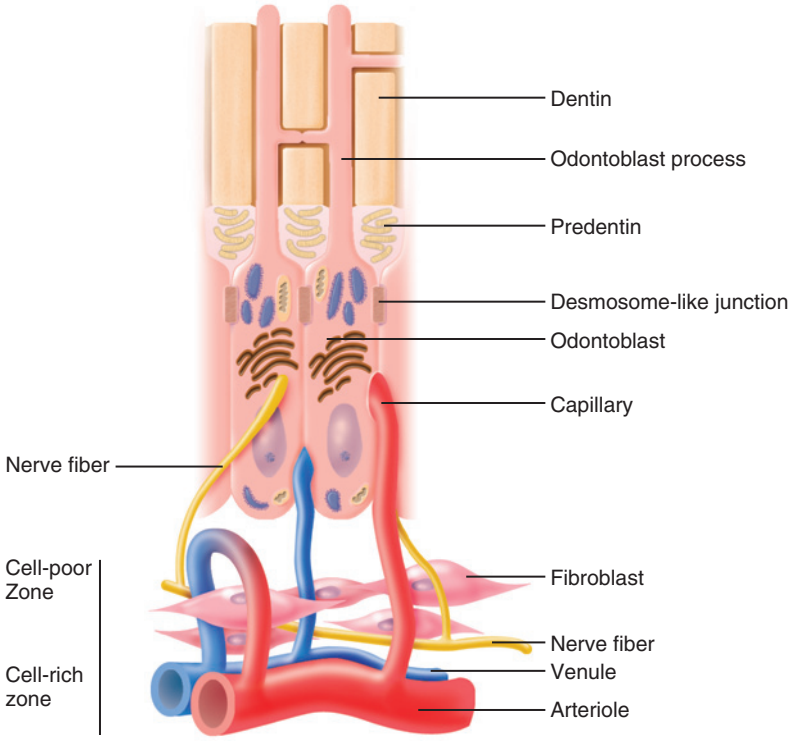

FIG. 12-2 Diagrammatic representation of the odontoblast layer and subodontoblastic region of the pulp.

spaces approximately 30 to 40 nm in width. Odontoblast cell bodies are connected by tight and gap junctional complexes.[29,62,160] Gap junctions are formed by connexin proteins[113] that permit cell-to-cell passage of signal molecules.

The odontoblast layer in the coronal pulp contains more cells per unit area than in the radicular pulp.[240] Whereas the odontoblasts of the mature coronal pulp are usually columnar, those in the midportion of the radicular pulp are more cuboidal (Fig. 12-3).[62] Near the apical foramen, the odontoblasts appear as a squamous layer of flattened cells. Because fewer dentinal tubules per unit area are present in the root than in the crown of the tooth, the odontoblast cell bodies are less crowded and are able to spread out laterally.[240] During maturation and aging, there is a continued ongoing crowding in the odontoblast layer, particularly in the coronal pulp, due to narrowing of the pulp space. Apoptosis of odontoblasts seems to adjust for this limited space during development.[252]

There is a series of specialized cell-to-cell junctions (i.e., junctional complexes), including desmosomes (i.e., zonula adherens), gap junctions (i.e., nexuses), and tight junctions (i.e., zonula occludens) that connect adjacent odontoblasts. Spot desmosomes located in the apical part of odontoblast cell bodies mechanically join odontoblasts together. The numerous gap junctions provide permeable pathways through which

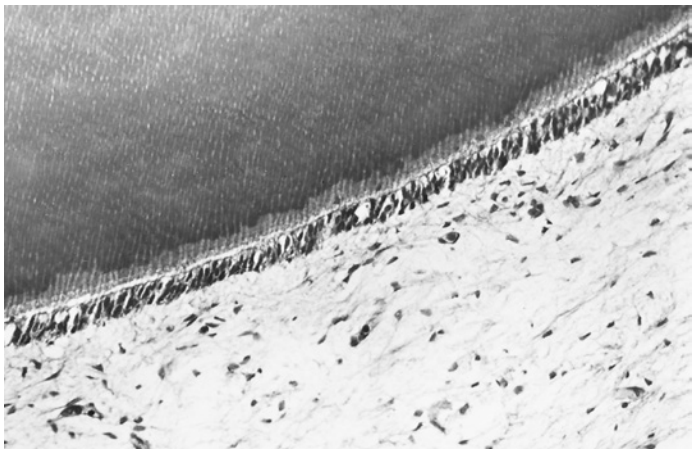

FIG. 12-3 Low columnar odontoblasts of the radicular pulp. The cell-rich zone is inconspicuous.

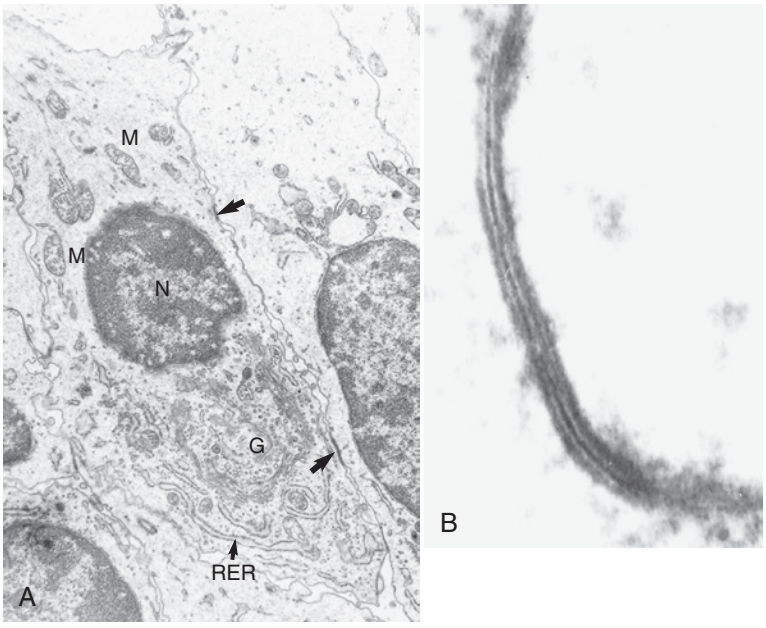

FIG. 12-4 A, Electron micrograph of a mouse molar odontoblast demonstrating gap junctions *(arrows),* nucleus (N), mitochondria (M), Golgi complex (G), and rough endoplasmic reticulum (RER). B, High magnification of a section fixed and stained with lanthanum nitrate to demonstrate a typical gap junction. (Courtesy Dr. Charles F. Cox, School of Dentistry, University of Alabama.)

signal molecules can pass between cells (Fig. 12-4) to synchronize secretory activity that produces relatively uniform predentin layers (see Fig. 12-2). These junctions are most numerous during the formation of primary dentin. Gap junctions and desmosomes have also been observed joining odontoblasts to the processes of fibroblasts in the subodontoblastic area. Tight junctions are found mainly in the apical part of odontoblasts in young teeth. These structures consist of linear ridges and grooves that close off the intercellular space. However, tracer studies suggest direct passage of small elements from subodontoblastic capillaries to predentin and dentin between the odontoblasts.[352] It appears that tight junctions determine the permeability of the odontoblast layer when dentin is covered by enamel or cementum by restricting the passage of molecules, ions, and fluid between the extracellular compartments of the pulp and predentin.[29] During cavity preparation, these junctions are disrupted, thereby increasing dentin permeability.[375,376]

Cell-Poor Zone

Immediately subjacent to the odontoblast layer in the coronal pulp, there is often a narrow zone approximately 40 µm in width that is relatively free of cells (see Fig. 12-1) and hence is called the *cell-free layer of Weil*. It is traversed by blood capillaries, unmyelinated nerve fibers and the slender cytoplasmic processes of fibroblasts (see Fig. 12-2). The presence or absence of the cell-poor zone depends on the functional status of the pulp.[62] It may not be apparent in young pulps, where dentin forms rapidly, or in older pulps, where reparative dentin is being produced.

Cell-Rich Zone

In the subodontoblastic area, there is a stratum containing a relatively high proportion of fibroblasts compared with the more central region of the pulp (see Fig. 12-1). It is much more prominent in the coronal pulp than in the radicular pulp. Besides fibroblasts, the cell-rich zone may include a variable number of immune cells like macrophages and dendritic cells, but also undifferentiated mesenchymal stem cells.

On the basis of evidence obtained in rat molar teeth, it has been suggested[126] that the cell-rich zone forms as a result of peripheral migration of cells populating the central regions of the pulp, commencing at about the time of tooth eruption. Migration of immunocompetent cells out of and into the

cell-rich zone has been demonstrated as a result of antigenic challenge.[421] Although cell division within the cell-rich zone is rare in normal pulps, the death of the odontoblasts triggers a great increase in the rate of mitosis. Because odontoblasts are postmitotic cells, irreversibly injured odontoblasts are replaced by cells that migrate from the cell-rich zone onto the inner surface of the dentin.[98] This mitotic activity is probably the first step in the formation of a new odontoblast layer.[73,256,258,339] Studies implicate stem cells as a source for these replacement odontoblasts.[346]

Pulp Proper

The pulp proper is the central mass of the pulp (see Fig. 12-1). It consists of loose connective tissue and contains the larger blood vessels and nerves. The most prominent cell in this zone is the fibroblast.

CELLS OF THE PULP

Odontoblast

Because odontoblasts are responsible for dentinogenesis, both during tooth development and aging, the odontoblast is the most characteristic and specialized cell of the dentin-pulp complex. During dentinogenesis, the odontoblasts form dentin and the dentinal tubules, and their presence within the tubules makes dentin a living responsive tissue.

Dentinogenesis, osteogenesis, and cementogenesis are in many respects quite similar, and odontoblasts, osteoblasts, and cementoblasts have many characteristics in common. Each of these cells produces a matrix composed of collagen fibrils, noncollagenous proteins, and proteoglycans that are capable of undergoing mineralization. The ultrastructural characteristics of odontoblasts, osteoblasts, and cementoblasts are likewise similar in that each exhibits a highly ordered rough endoplasmic reticulum (RER), a prominent Golgi complex, secretory granules, and numerous mitochondria. In addition, these cells are rich in RNA, and their nuclei contain one or more prominent nucleoli. These are the general characteristics of protein-secreting cells.

The most significant differences among odontoblasts, osteoblasts, and cementoblasts are their morphologic characteristics and the anatomic relationship between the cells and the mineralized structures they produce. Whereas osteoblasts and cementoblasts are polygonal to cuboidal in form, the fully developed odontoblast of the coronal pulp is a tall columnar cell.[62,240] In bone and cementum, some of the osteoblasts and cementoblasts become entrapped in the matrix as osteocytes or cementocytes, respectively. The odontoblast, on the other hand, leaves behind a cellular process to form the dentinal tubule, and the cell body resides outside the mineralized tissue. Lateral branches between the major odontoblast processes interconnect[178,253] through canaliculi, just as osteocytes and cementocytes are linked through the canaliculi in bone and cementum. This provides a pathway for intercellular communication and circulation of fluid and metabolites through the mineralized matrix.

The ultrastructural features of the odontoblast have been the subject of numerous investigations. The cell body of the active odontoblast has a large nucleus that may contain up to four nucleoli (Fig. 12-5). The nucleus is situated at the basal end of the cell and is contained within a nuclear envelope. A well-developed Golgi complex, centrally located in the supranuclear cytoplasm, consists of an assembly of smooth-walled vesicles and cisternae. Numerous mitochondria are evenly distributed throughout the cell body. RER is particularly prominent, consisting of closely stacked cisternae forming parallel arrays that are dispersed diffusely within the cytoplasm. Numerous ribosomes closely associated with the membranes of the cisternae mark the sites of protein synthesis. Within the lumen of the cisternae, filamentous material (probably representing newly synthesized protein) can be observed.

The odontoblast appears to synthesize mainly type I collagen,[208,401] although small amounts of type V collagen have been found in the extracellular matrix (ECM). In addition to proteoglycans[38,83,123] and collagen,[208,220] the odontoblast secretes dentin sialoprotein[40] and phosphophoryn,[40,69] a highly phosphorylated phosphoprotein involved in extracellular mineralization.[40,77] Phosphophoryn is unique to dentin and is not found in any other mesenchymal cell types.[77] The odontoblast also secretes both acid phosphatase and alkaline phosphatase. The latter enzyme is closely linked to mineralization, but the precise role of alkaline phosphatase in dentinogenesis is not completely understood. Acid phosphatase, a lysosomal enzyme, may be involved in digesting material that has been resorbed from predentin matrix.[87]

In contrast to the active odontoblast, the resting or inactive odontoblast has a decreased number of organelles and may become progressively shorter.[62,240] These changes can begin with the completion of root development and eruption when dentin production shifts from primary to secondary dentin.

Direct actions of odontoblasts on dental nerves and vice versa have been proposed based on the excitability of odontoblasts, the differential expression of receptors for neuropeptides on odontoblasts (Fig. 12-6), the demonstration of the thermosensitive transient receptor potential (TRP) ion channels, and the finding that all nine voltage-gated sodium channels are variably expressed on odontoblasts in developing, mature, and aging rat teeth.[52,71,107,221,231,232] In addition, a possible function of odontoblast in immune regulation has been proposed by the finding of innate immune components in the odontoblast layer.[382] Hence, the odontoblasts should be capable of recognizing and differentially responding to bacterial components, thereby serving immune and pulp-dentine barrier functions.

Odontoblast Process

A dentinal tubule forms around each of the major odontoblastic processes. The odontoblast process occupies most of the space within the tubule and coordinates the formation of peritubular dentin.

Microtubules and microfilaments are the principal ultrastructural components of the odontoblast process and its lateral branches.[110,158] Microtubules extend from the cell body out into the process.[110,151] These straight structures follow a course that is parallel with the long axis of the cell and impart the impression of rigidity. Although their precise role is unknown, theories as to their functional significance suggest that they may be involved in cytoplasmic extension, transport of materials, or the provision of a structural framework. Occasionally, mitochondria can be found in the process where it passes through the predentin.

The plasma membrane of the odontoblast process closely approximates the wall of the dentinal tubule. Localized

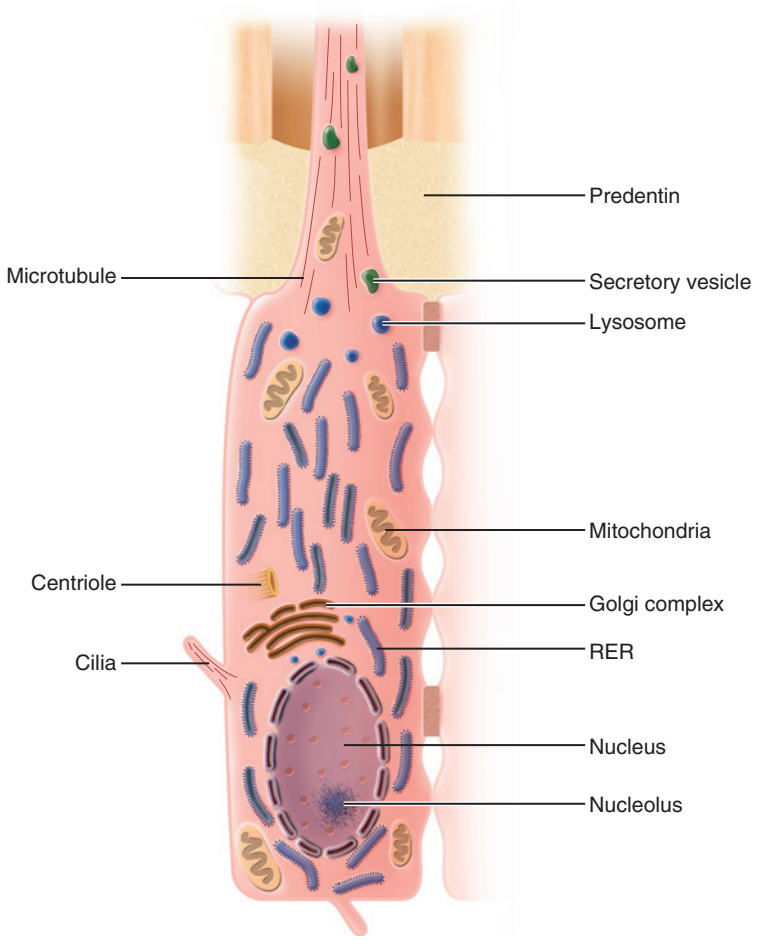

FIG. 12-5 Diagram of a fully differentiated odontoblast. *RER,* Rough endoplasmic reticulum.

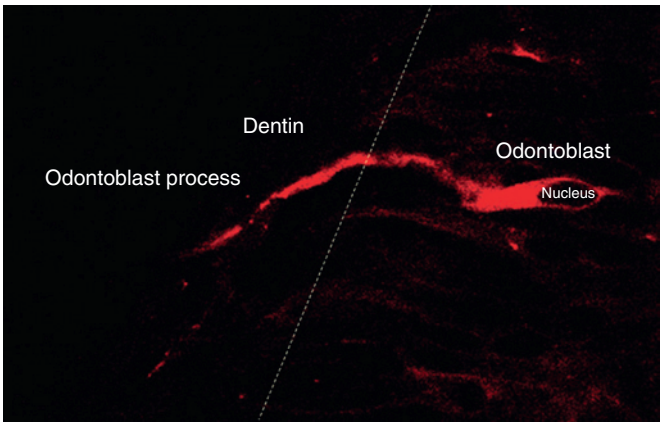

FIG. 12-6 Confocal microscopic image showing an odontoblast with its process expressing neurokinin 2 receptor. Neurokinin 2 has affinity to all the neuropeptides of the neurokinin family. The dotted line represents the border of the predentin. (From Fristad I, Vandevska-Radunovic V, Fjeld K, et al: NK1, NK2, NK3 and CGRP1 receptors identified in rat oral soft tissues, and in bone and dental hard tissue cells, *Cell Tissue Res* 311:383-391, 2003.)

constrictions in the process occasionally produce relatively large spaces between the tubule wall and the process. Such spaces may contain collagen fibrils and fine granular material that presumably represents ground substance (see also The Pulpal Interstitium and Ground Substance in this chapter). The peritubular dentin matrix within the tubule is lined by an electron-dense limiting membrane called the *lamina limitans*.[260,354,398] A narrow space separates the limiting membrane from the plasma membrane of the odontoblast process, except for the areas where the process is constricted.

In restoring a tooth, the removal of enamel and dentin often disrupts odontoblasts.[41,45,73,80,202,257] It would be of considerable clinical importance to establish the extent of the odontoblast processes in human teeth, because with this knowledge, the clinician would be in a better position to estimate the impact of the restorative procedure on the underlying odontoblasts. However, the extent to which the process penetrates into dentin has been a matter of considerable controversy. It has long been thought that the process is present throughout the full thickness of dentin. Although ultrastructural studies using transmission electron microscopy have described the process as being limited to the inner third of the dentin,[48,111,354,398] it should be noted that this could possibly be the result of shrinkage occurring during fixation and dehydration. Other studies employing scanning electron microscopy have described the process extending further into the tubule, often as far as the dentoenamel junction (DEJ),[130,179,337,412] but it has been suggested that what has been observed in scanning electron micrographs is actually the lamina limitans.[354,355,398]

In an attempt to resolve this issue, monoclonal antibodies directed against microtubules were used to demonstrate tubulin in the microtubules of the process. Immunoreactivity was observed throughout the dentinal tubule, suggesting that the process extends throughout the thickness of dentin.[337]

However, a study employing confocal microscopy found that odontoblast processes in rat molars do not extend to the outer dentin or DEJ, except during the early stages of tooth development.[48] It is likely that the walls of tubules contain many proteins originally derived from odontoblasts that no longer remain at that site. Because dentin matrix has no turnover, these antigens remain fixed in place. From a clinical perspective, it is important to remember that these processes in the tubules represent appendages from living odontoblasts in the pulp, which explains why the dentin must be considered a vital tissue, the destruction of which will affect the pulp.

The odontoblast is considered to be a fixed postmitotic cell in that once it has fully differentiated, it apparently cannot undergo further cell division. If this is indeed the case, the life span of the odontoblast coincides with the life span of the viable pulp. However, its metabolic activity can be dynamically altered (described under the heading Pulpal Repair).

Relationship of Odontoblast Structure to Secretory Function

Studies using radiolabeled chemicals have shed a great deal of light on the functional significance of the cytoplasmic organelles of the active odontoblast.[401,400] In experimental animals, intraperitoneal injection of a collagen precursor (e.g., ^{3}H-proline) is followed by autoradiographic labeling of the odontoblasts and predentin matrix[401] (Fig. 12-7). Rapid incorporation of the isotope in the RER soon leads to labeling of the Golgi complex in the area where the procollagen is packed and concentrated into secretory vesicles. Radiolabeled vesicles can then be followed along their migration pathway until they reach the base of the odontoblast process. Here they fuse with the cell membrane and release their tropocollagen molecules into the predentin matrix by the process of exocytosis.

It is now known that collagen fibrils precipitate from a solution of secreted tropocollagen and that the aggregation of fibrils occurs on the outer surface of the odontoblast plasma membrane. Fibrils are released into the predentin and increase in thickness as they approach the mineralized matrix. Whereas fibrils at the base of the odontoblast process are approximately 15 nm in diameter, fibrils in the region of the calcification front have attained a diameter of about 50 nm.

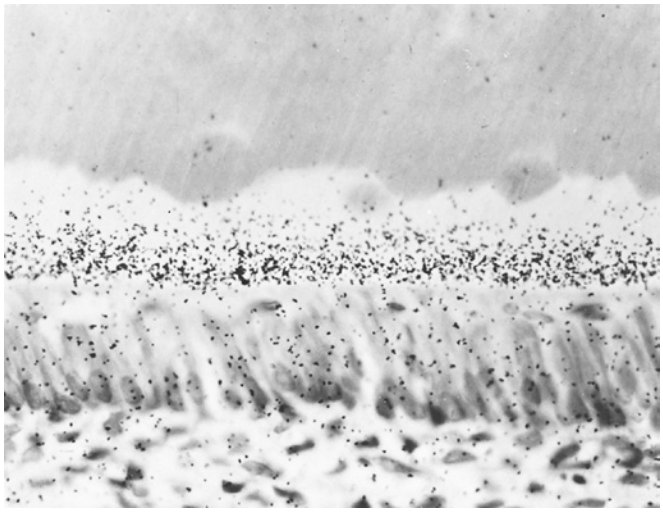

FIG. 12-7 Autoradiograph demonstrating odontoblasts and predentin in a developing rat molar 1 hour after intraperitoneal injection of ^{3}H-proline.

Similar tracer studies[400] have elucidated the pathway of synthesis, transport, and secretion of the predentin proteoglycans. The protein moiety of these molecules is synthesized by the RER of the odontoblast, whereas sulfation and addition of the glycosaminoglycan (GAG) moieties to the protein molecules take place in the Golgi complex. Secretory vesicles then transport the proteoglycans to the base of the odontoblast process, where they are secreted into the predentin matrix. Proteoglycans, principally chondroitin sulfate, accumulate near the calcification front. The role of the proteoglycans is speculative, but mounting evidence suggests that they act as inhibitors of calcification by binding calcium. It appears that just before calcification, the proteoglycans are removed, probably by lysosomal enzymes secreted by the odontoblasts.[84]

Pulp Fibroblast

Fibroblasts are the most numerous cells of the pulp. They appear to be tissue-specific cells capable of giving rise to cells that are committed to differentiation (e.g., odontoblast-like cells) if given the proper signal. These cells synthesize types I and III collagen, as well as proteoglycans and GAGs. Thus, they produce and maintain the matrix proteins of the ECM. Because they are also able to phagocytose and digest collagen, fibroblasts are responsible for collagen turnover in the pulp.

Although distributed throughout the pulp, fibroblasts are particularly abundant in the cell-rich zone. The early differentiating fibroblasts are polygonal and appear to be widely separated and evenly distributed within the ground substance. Cell-to-cell contacts are established between the multiple processes that extend out from each of the cells. Many of these contacts take the form of gap junctions that provide for electronic coupling or chemical signaling from one cell to another. In terms of ultrastructure, the organelles of the immature fibroblasts are generally in a rudimentary stage of development, with an inconspicuous Golgi complex, numerous free ribosomes, and sparse RER. As they mature, the cells become stellate in form, and the Golgi complex enlarges, the RER proliferates, secretory vesicles appear, and the fibroblasts take on the characteristic appearance of protein-secreting cells. In addition, collagen fibrils accumulate along the outer surface of the cell body. With an increase in the number of blood vessels, nerves, and collagen fibers, there is a relative decrease in the number of fibroblasts in the pulp.

Many fibroblasts of the pulp are characterized by being relatively undifferentiated. A more modern term for undifferentiated cells is *stem cells*. Many pulpal cells do seem to remain in a relatively undifferentiated modality, compared with fibroblasts of most other connective tissues.[136] This perception has been supported by the observation of large numbers of reticulin-like fibers in the pulp. Reticulin fibers have an affinity for silver stains and are similar to the argyrophilic fibers of the pulp. However, in a careful review, it appears that actual reticulin fibers may not be present in the pulp; instead the previously described fibers are actually *argyrophilic collagen fibers*.[15] The fibers apparently acquire a GAG sheath, and it is this sheath that is impregnated by silver stains. In the young pulp, the nonargyrophilic collagen fibers are sparse, but they progressively increase in number as the pulp ages.

Many experimental models have been developed to study wound healing in the pulp, particularly dentinal bridge formation after pulp exposure or pulpotomy. One study[98] demonstrated that mitotic activity preceding the differentiation of replacement odontoblasts appears to occur primarily among perivascular fibroblasts.

Pulpal fibroblasts seem to take active part in signaling pathways in the dental pulp. For example, fibroblast growth and synthesis are stimulated by neuropeptides; in turn, fibroblasts produce nerve growth factor (NGF) and proinflammatory cytokines during inflammation.[31,408,413] NGF plays an important role not only in tooth development but also in regulating neuronal and possibly odontoblast responses to injury via activation of similar neurotrophin receptors expressed on both cell types (see also Plasticity of Intradental Nerve Fibers in this chapter).[408]

Macrophage

Macrophages are monocytes that have left the bloodstream, entered the tissues, and differentiated into various subpopulations. The different subpopulations can be studied by their antigenic properties in immunohistochemical studies. Many are found in close proximity to blood vessels. A major subpopulation of macrophages is active in endocytosis and phagocytosis (Fig. 12-8). Because of their mobility and phagocytic activity, they are able to act as scavengers, removing extravasated red blood cells, dead cells, and foreign bodies from the tissue. Ingested material is destroyed by the action of lysosomal enzymes. Another subset of macrophages participates in immune reactions by processing antigen and presenting it to memory T cells.[281] The processed antigen is bound to class II major histocompatibility complex (MHC) molecules on the macrophage, where it can interact with specific receptors present on naive or memory T cells.[133] Such interactions are essential for T cell–dependent immunity. Similar to fibroblasts, macrophages take an active part in the signaling pathways in the pulp. When activated by the appropriate inflammatory stimuli, macrophages are capable of producing a large variety of soluble factors, including interleukin 1, tumor necrosis factor, growth factors, and other cytokines. One study showed that a subset of macrophages express lymphatic markers, indicating a link between macrophages and lymphatic function and development.[19]

Dendritic Cell

Dendritic cells are accessory cells of the immune system. Similar cells are found in the epidermis and mucous membranes, where they are called *Langerhans cells*.[173,280] Dendritic cells are primarily found in lymphoid tissues, but they are also widely distributed in connective tissues, including the pulp[319] (Fig. 12-9). These cells are termed *antigen-presenting cells* and are characterized by dendritic cytoplasmic processes and the presence of class II MHC complexes on their cell surface (Fig. 12-10). In the normal pulp they are mostly located in the periphery of the coronal pulp close to the predentin, but they migrate centrally in the pulp after antigenic challenge.[421] They are known to play a central role in the induction of T cell–dependent immunity. Like antigen-presenting macrophages, dendritic cells engulf protein antigens and then present an assembly of peptide fragments of the antigens and MHC class II molecules. It is this assembly that T cells can recognize. Then the assembly binds to a T-cell receptor and T-cell activation

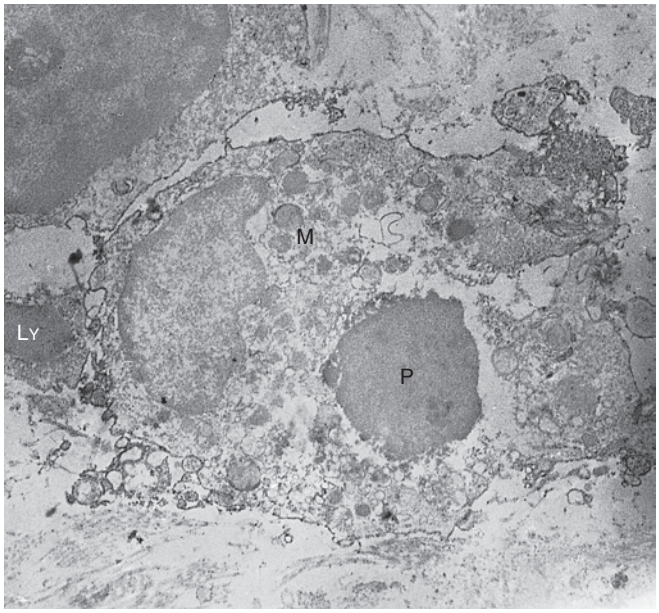

FIG. 12-8 Immunoelectron micrograph of an HLA-DR+ matured macrophage (M) in the human pulp, showing a phagosome (P). *Ly,* lymphocyte.

FIG. 12-9 Class II antigen-expressing dendritic cells in the pulp and dentin border zone in normal human pulp, as demonstrated by immunocytochemistry. *D,* dentin; *OB,* odontoblastic layer.

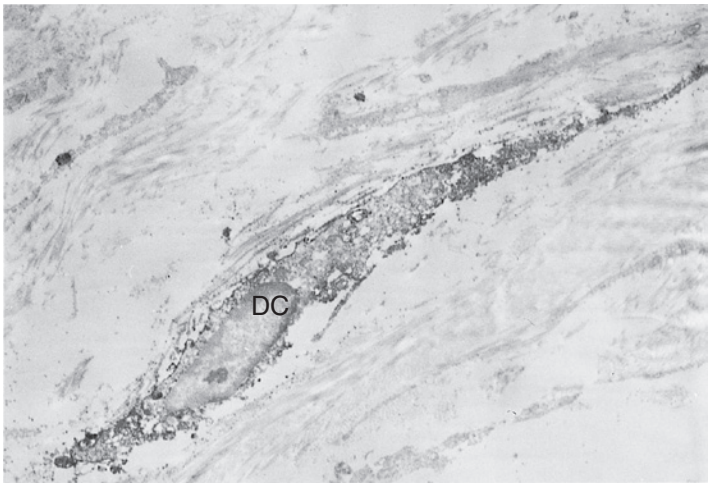

FIG. 12-10 Immunoelectron micrograph of a dendritic-like cell (DC) in the human pulp, showing a dendritic profile with a relatively small number of lysosomal structures.

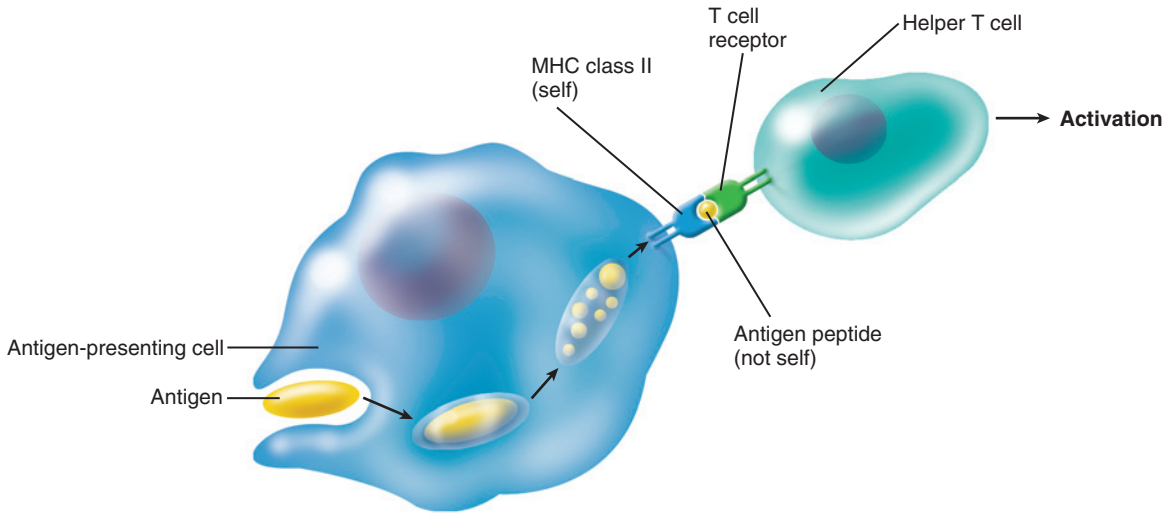

FIG. 12-11 Function of MHC class II molecule-expressing cells. They act as antigen-presenting cells that are essential for the induction of helper T cell–dependent immune responses.

occurs (Fig. 12-11). Fig. 12-12 shows a cell-to-cell contact between a dendritic-like cell and a lymphocyte.

Lymphocyte

Hahn and colleagues[133] reported finding T lymphocytes in normal pulps from human teeth. T8 (suppressor) lymphocytes were the predominant T-lymphocyte subset present in these samples. Lymphocytes have also been observed in the pulps of impacted teeth.[201] The presence of macrophages, dendritic cells, and T lymphocytes indicates that the pulp is well equipped with cells required for the initiation of immune responses.[173,319] B lymphocytes are scarcely found in the normal uninflamed pulp.

Mast Cell

Mast cells are widely distributed in connective tissues, where they occur in small groups in relation to blood vessels. Mast cells are seldom found in the normal pulp tissue, although they are routinely found in chronically inflamed pulps.[319] The mast cell has been the subject of considerable attention because of its dramatic role in inflammatory reactions. The granules of mast cells contain heparin, an anticoagulant, and histamine, an important inflammatory mediator, as well as many other chemical factors.

METABOLISM

The metabolic activity of the pulp has been studied by measuring the rate of oxygen consumption and the production of carbon dioxide or lactic acid.[26,95,96,97,135,320] An investigation employed an oxygen-sensitive microelectrode inserted into a rat incisor pulp with a micromanipulator.[418] The authors reported that odontoblasts consumed O_2 at the rate of 3.2 ± 0.2 ml/min/100 g of pulp tissue.[418]

Because of the relatively sparse cellular composition of the pulp, the rate of oxygen consumption is low in comparison with that of most other tissues. During active dentinogenesis, metabolic activity is much higher than after the crown is completed. As would be anticipated, the greatest metabolic activity is found in the region of the odontoblast layer, and the lowest is found in the central pulp, where most of the nerves and blood vessels are located.[25]

In addition to the usual glycolytic pathway, the pulp has the ability to produce energy through a phosphogluconate (i.e.,

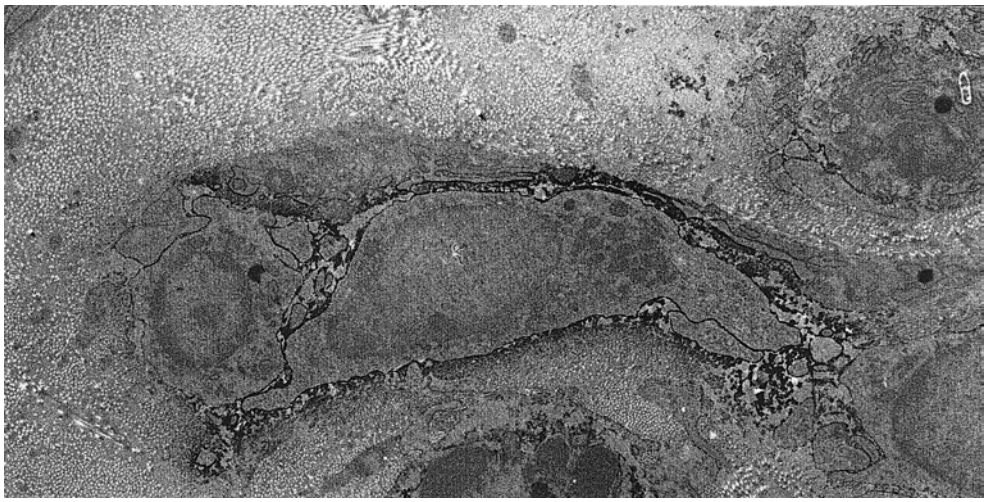

FIG. 12-12 Immunoelectron micrograph of a cell resembling a dendritic cell and a lymphocyte. They show cell-to-cell contact.

pentose phosphate) shunt type of carbohydrate metabolism,[97] a metabolic pathway that permits tissues to function under varying degrees of ischemia. This could explain how the pulp manages to withstand periods of low perfusion resulting from vasoconstriction induced by epinephrine-containing infiltration anesthesia.[190]

Several commonly used dental materials (e.g., eugenol, zinc oxide and eugenol, calcium hydroxide, silver amalgam) inhibit oxygen consumption by pulp tissue, indicating that these agents may be capable of depressing the metabolic activity of pulpal cells.[96,172] One study[135] found that application of orthodontic force to human premolars for 3 days resulted in a 27% reduction in respiratory activity in the pulp. This study utilized carbon-14-labeled succinic acid in the medium. As cells metabolize succinic acid, they produce $^{14}CO_2$ that can be trapped and quantitated by a liquid scintillation counter.[135] This technique requires only a few milligrams of tissue.

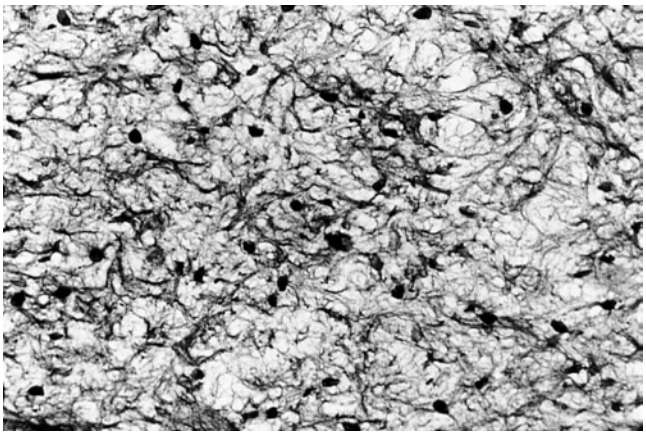

FIG. 12-13 Delicate network of pulpal collagen fibers as demonstrated by the Pearson silver impregnation method.

THE PULPAL INTERSTITIUM AND GROUND SUBSTANCE

The interstitium consists of the interstitial fluid and the interstitial (extracellular) matrix and occupies the extracellular and extravascular space. It is amorphous and generally regarded as a gel rather than a solid. Its constituents are similar in all tissues, but their relative amount varies. The major structural component of the interstitium is collagen (Fig. 12-13). The network of collagen fibers also supports the other components of the interstitium, the proteoglycans, hyaluronan, and elastic fibers. The two former components represent the glycosaminoglycans of the interstitial matrix.

Because of its content of polyanionic polysaccharides, the interstitium is responsible for the water-holding properties of connective tissues and acts as a molecular sieve in regulating the diffusion of substances through this space. The magnitude of the excluded volume has important consequences because the effective protein concentration in the interstitium is higher than the value that would be estimated from fluid volume per se.[404]

Connective tissue consists of cells and fibers, both embedded in ground substance or ECM. Cells that produce connective tissue fibers also synthesize the major constituents of the ECM. Whereas the fibers and cells have recognizable shapes, the ECM is described as being amorphous. It is generally regarded as a gel rather than a solid. Because of its content of polyanionic polysaccharides, the ECM is responsible for the water-holding properties of connective tissues.[404]

Nearly all proteins of the ECM are glycoproteins.[237] Proteoglycans are an important subclass of glycoproteins.[123] These molecules support cells, provide tissue turgor, and mediate a variety of cell interactions. They have in common the presence of GAG chains and a protein core to which the chains are linked. Except for heparan sulfate and heparin, the chains are composed of disaccharides. The primary function of GAG chains is to act as adhesive molecules that can bond to cell surfaces and other matrix molecules.

Fibronectin is a major surface glycoprotein that, together with collagen, forms an integrated fibrillary network that influences adhesion, motility, growth, and differentiation of cells. Laminin, an important component of basement membranes, binds to type IV collagen and cell surface receptors.[118] Tenascin is another substrate adhesion glycoprotein.

In the pulp, the principal proteoglycans include hyaluronic acid dermatan sulfate, heparan sulfate, and chondroitin sulfate.[130] The proteoglycan content of pulp tissue decreases approximately 50% with tooth eruption.[220] During active dentinogenesis, chondroitin sulfate is the principal proteoglycan, particularly in the odontoblast and predentin layer, where it is somehow involved with mineralization; with tooth eruption, hyaluronic acid and dermatan sulfate increase, and chondroitin sulfate decreases greatly.

The consistency of a connective tissue (e.g., the pulp) is largely determined by the proteoglycan components of the ground substance. The long GAG chains of the proteoglycan molecules form relatively rigid coils constituting a network that holds water, thus forming a characteristic gel. Hyaluronic acid in particular has a strong affinity for water and is a major component of ground substance in tissues with a large fluid content, such as Wharton's jelly of the umbilical cord. The water content of the young pulp is very high (approximately 90%), so the ground substance forms a cushion capable of protecting cells and vascular components of the tooth.

Ground substance also acts as a molecular sieve in that it excludes large proteins. Cell metabolites, nutrients, and wastes pass through the ground substance between cells and blood vessels. In some ways, ground substance can be likened to an ion exchange resin, because the polyanionic chains of the GAGs bind cations. In addition, osmotic pressures can be altered by excluding osmotically active molecules. Thus, proteoglycans can regulate the dispersion of interstitial matrix solutes, colloids, and water, and (in large measure) they determine the physical characteristics of a tissue, such as the pulp.

Degradation of ground substance can occur in certain inflammatory lesions that have a high concentration of macrophage lysosomal enzymes. Proteolytic enzymes, hyaluronidases, and chondroitin sulfatases of lysosomal and bacterial origin are examples of the hydrolytic enzymes that can attack components of the ground substance. The pathways of inflammation and infection are strongly influenced by the state of polymerization of the ground substance components.

Hyaluronan

Another major structural component of the interstitial matrix is hyaluronan. It is an unbranched, random-coil molecule from repeating nonsulfated disaccharide units and is found in the interstitium as free molecules or bound to cells, possibly via the connection to fibronectin.[205] Its large molecular weight together with its protein structure accounts for its unique properties. It has a high viscosity even at low concentration, exhibits exclusion properties, and has a strong affinity for water.

Hyaluronan is one of several types of GAGs in the pulp.[219,237] The hyaluronan receptor-1 is expressed on lymphatic vessels and also on immune cells in the dental pulp.[19] Hyaluronan is removed from the tissue by the lymphatics and metabolized in the lymph nodes[101] and by endothelial cells in the liver.[134,298]

Elastic Fibers

Elastic fibers constitute an elastin core and a surrounding microfibrillar network and provide elasticity to the tissue.[289] The amount of elastin in the interstitial matrix in most tissue is small. There is no evidence for elastic fibers in the matrix in the pulp.[134,298]

The Inflamed Interstitium

Hyaluronidases, and chondroitin sulfatases of lysosomal and bacterial origin, are examples of the hydrolytic enzymes that can attack components of the interstitium. During infection and inflammation, the physical properties of the pulp tissue may then be altered due to production of such degrading enzymes.[145,318] In addition to their own damaging effect, they may also pave the way for the deleterious effects of bacterial toxins, increasing the magnitude of the damage.[177]

The pathways of inflammation and infection are strongly influenced by the particular composition of the interstitium in every tissue and its degradation by either host or microbial enzymes.

CONNECTIVE TISSUE FIBERS OF THE PULP

Two types of structural proteins are found in the pulp: collagen and elastin. Elastin fibers are confined to the walls of arterioles and, unlike collagen, are not a part of the ECM.

A single collagen molecule, referred to as *tropocollagen,* consists of three polypeptide chains, designated as either alpha-1 or alpha-2 depending on their amino acid composition and sequence. In the human pulp, the amount of collagen is reported to be 26% to 32% of dry weight in premolars and molars.[378] Type I and type III collagen represent the major subtypes of collagen in the pulp, and type I is found in thick striated fibrils throughout the pulp tissue.[206,336] The different combinations and linkages of chains making up the tropocollagen molecule have allowed collagen fibers and fibrils to be classified into a number of types:

- Type I collagen is found in skin, tendon, bone, dentin, and pulp.
- Type II collagen is found in cartilage.
- Type III collagen is found in most unmineralized connective tissues. It is a fetal form found in the dental papilla and the mature pulp. In bovine pulp, it constitutes 45% of the total pulp collagen during all stages of development.[378]
- Types IV and VII collagen are components of basement membranes.
- Type V collagen is a constituent of interstitial tissues.
- Type VI collagen is a heterotrimer of three distinct chains, alpha 2 (VI) and alpha 3 (VI), and is widely distributed in low concentrations in soft tissues at interfibrillar filaments.

Type I collagen is synthesized by odontoblasts and osteoblasts; fibroblasts synthesize types I, III, V, and VII collagen.

In collagen synthesis, the protein portion of the molecule is formed by the polyribosomes of the RER of connective tissue cells. The proline and lysine residues of the polypeptide chains are hydroxylated in the cisternae of the RER, and the chains are assembled into a triple-helix configuration in the smooth endoplasmic reticulum. The product of this assembly is termed *procollagen,* and it has a terminal unit of amino acids known as the *telopeptide of the procollagen molecule*. When these molecules reach the Golgi complex, they are glycosylated and packaged in secretory vesicles. The vesicles are transported to the plasma membrane and secreted by way of exocytosis into the extracellular milieu, thus releasing the procollagen. Here the terminal telopeptide is cleaved by a hydrolytic enzyme, and the tropocollagen molecules begin aggregating to form collagen fibrils. It is believed that the GAGs somehow mediate aggregation of tropocollagen. The conversion of soluble collagen into

FIG. 12-14 Dense bundles of collagen fibers (CF) in the apical pulp.

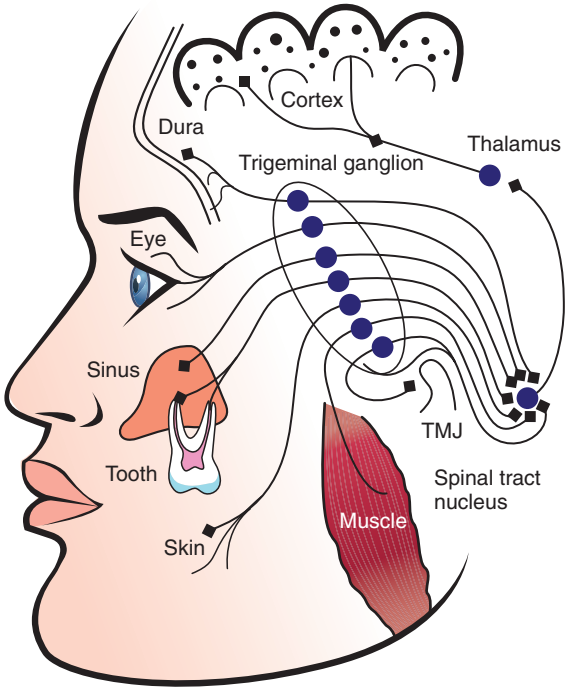

FIG. 12-15 Schematic drawing illustrating the convergence of sensory information from teeth to higher brain centers.

insoluble fibers occurs as a result of cross-linking of tropocollagen molecules. The presence of collagen fibers passing from the dentin matrix between odontoblasts into the dental pulp has been reported in fully erupted teeth.[28] Larger collagen fiber bundles are much more numerous in the radicular pulp than in the coronal pulp. The highest concentration of these larger fiber bundles is usually found near the apex (Fig. 12-14). Pulpectomy procedures should engage the pulp with a barbed broach in the region of the apex, as it generally affords the best opportunity to remove this tissue intact.[366]

THE TRIGEMINAL SYSTEM

Innervation

Pain is a subjective phenomenon involving not only sensory physiologic responses, but also emotional, conceptual, and motivational aspects of behavior. The existence of peripheral "nociceptive" (pain-detecting) sensory neurons forms the basis for pain, and pain sensations of varying qualities and intensities are evoked by activation of the intradental nerves innervating teeth. Noxious stimuli in teeth are transmitted in primary afferent neurons located in the trigeminal ganglion via second-order neurons in the brain stem to the brain (Fig. 12-15; see also Chapter 17 and later in this chapter). Transmission of sensory information consists of a cascade of events involving input, processing, and sensing,[329] so the control of dental pain should be based on an understanding of the origin of pain signals and the complex modulation that may take place locally and at higher levels. The sensory system of the pulp appears to be well suited for signaling potential damage to the tooth. The tooth is innervated by a large number of myelinated and unmyelinated axons. The number of axons entering a human premolar may reach 2000 or more, and each axon can arborize to form multiple points of innervation.[90,170,169]

Regardless of the nature of the sensory stimulus (i.e., thermal, mechanical, chemical, electric [e.g., pulp tester]), almost all afferent impulses generated from pulp tissue result in the sensation of pain. However, when the pulp is weakly stimulated by an electric pulp tester under carefully controlled experimental conditions, a nonpainful sensation (i.e., prepain) has been reported.[247] Thus, not all afferent neurons that innervate the pulp are nociceptors. The innervation of the pulp includes both *afferent neurons*, which conduct sensory impulses, and *autonomic or efferent neurons*,[163] which provide neurogenic modulation of the microcirculation, inflammatory reactions,[148] and perhaps regulate dentinogenesis.[46]

The sympathetic innervation of teeth derives from the superior cervical ganglion (SCG).[8,301] Postganglionic sympathetic nerves travel with the internal carotid nerve, join the trigeminal nerve at the ganglion, and supply teeth and supporting structures via the maxillary and mandibular division of the trigeminal nerve.[239] Sympathetic fibers appear with blood vessels at the time the vascular system is established in the dental papilla.[104] In the adult tooth pulp, sympathetic fibers form plexuses, usually around pulpal arterioles (Fig. 12-16). Stimulation of these fibers results in constriction of the arterioles and a decrease in blood flow.[1,82] The sympathetic neuron terminals contain the classic neurotransmitter, norepinephrine (NE), and neuropeptide Y (NPY) (see Neuropeptides later in this chapter). NPY is synthesized in sympathetic neurons and supplied to terminals by axonal transport. By contrast, NE is mainly produced locally in the terminals. Compared with the sensory nerves, these fibers are most often located in deeper parts of the pulp proper, but fibers have also been found in close relation to odontoblasts.[1,164]

The presence of parasympathetic cholinergic nerves in dental tissues has been and is still controversial, although it has been concluded that there is absence of parasympathetic vasodilation in the cat dental pulp.[286,323] It has been reported that the neuropeptide vasoactive intestinal polypeptide (VIP) is localized in the parasympathetic neurons.[225,226] The origin of VIP-containing fibers in the pulp is uncertain insofar as no form of surgical denervation has resulted in complete loss of these fibers from the dental pulp.[390]

Sensory nerve fibers are usually classified according to their diameter, conduction velocity, and function as shown in Table 12-1. The pulp contains two types of sensory nerve fibers: myelinated (A fibers) and unmyelinated (C fibers). It has been shown that there is some functional overlap between pulpal A and C fibers, as both fiber types can be nociceptors.[163,169,170,246,267] The A fibers include both A-beta and A-delta fibers. The A-beta fibers may be slightly more sensitive to stimulation than the A-delta fibers, but functionally these fibers are grouped together in the dental pulp, because both innervate the dentinal tubules, and both are stimulated by dentinal fluid movement (Fig. 12-17). Approximately 90% of the A fibers in dental pulp are A-delta fibers.[246] Table 12-2 summarizes the principal characteristics of the main sensory fibers.

During the bell stage of tooth development, "pioneer" nerve fibers enter the dental papilla following the path of blood vessels.[104] Although only unmyelinated fibers are observed in the dental papilla, a proportion of these fibers are probably A fibers that have lost or not developed their myelin sheath. Myelinated fibers are the last major structures to appear in the developing human dental pulp.[11] The number of nerve fibers gradually increases, and branching occurs as the fibers approach dentin. During the bell stage, very few fibers enter the predentin.[104]

The sensory nerves of the pulp arise from the trigeminal nerve and pass into the radicular pulp in bundles by way of

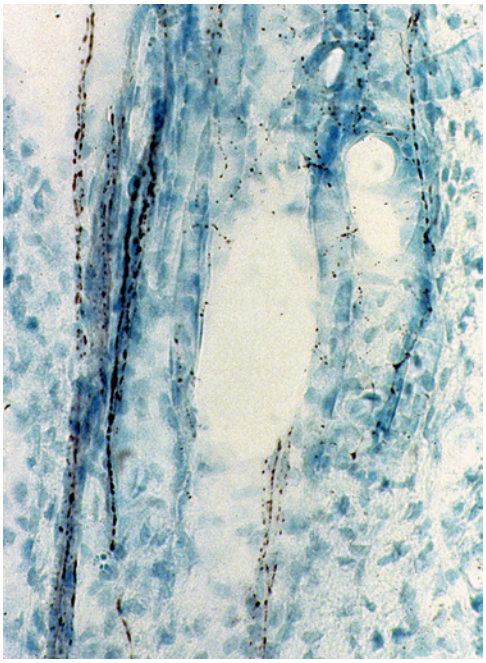

FIG. 12-16 Histologic section, immunohistologically stained for neuropeptide Y (NPY), shows the distribution of sympathetic nerves in the root pulp of a rat molar. NPY fibers are seen associated with blood vessels. (Courtesy Dr. Inge Fristad, Department of Clinical Dentistry, University of Bergen.)

TABLE 12-1
Classification of Nerve Fibers

Type of Fiber	Function	Diameter (μm)	Conduction Velocity (m/sec)
A-alpha	Motor, proprioception	12-20	70-120
A-beta	Pressure, touch	5-12	30-70
A-gamma	Motor, to muscle spindles	3-6	15-30
A-delta	Pain, temperature, touch	1-5	6-30
B	Preganglionic autonomic	< 3	3-15
C dorsal root	Pain	0.4-1	0.5-2
Sympathetic	Postganglionic sympathetic	0.3-1.3	0.7-2.3

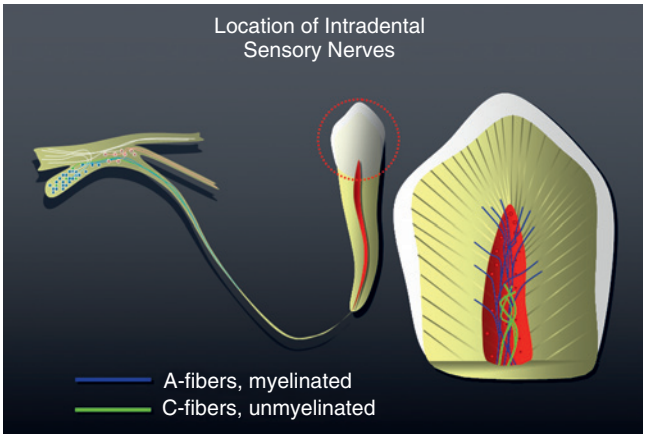

FIG. 12-17 Schematic drawing illustrating the location of A and C fibers in the dental pulp. Myelinated A fibers are located in the periphery of the pulp, penetrating the inner part of dentin. Unmyelinated C fibers are located in the deeper part of the pulp proper.

TABLE 12-2
Characteristics of Sensory Fibers

Fiber	Myelination	Location of Terminals	Pain Characteristics	Stimulation Threshold
A-delta	Yes	Principally in region of pulp-dentin junction	Sharp, pricking	Relatively low
C	No	Probably distributed throughout pulp	Burning, aching, less bearable than A-delta fiber sensations	Relatively high, usually associated with tissue injury

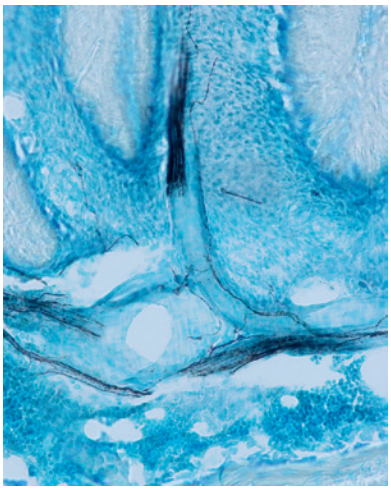

FIG. 12-18 Histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows distribution of sensory nerves in the apical area of a rat molar. Nerve fibers are seen associated with blood vessels and enter the dental pulp in nerve bundles. (Courtesy Dr. Inge Fristad, Department of Clinical Dentistry, University of Bergen.)

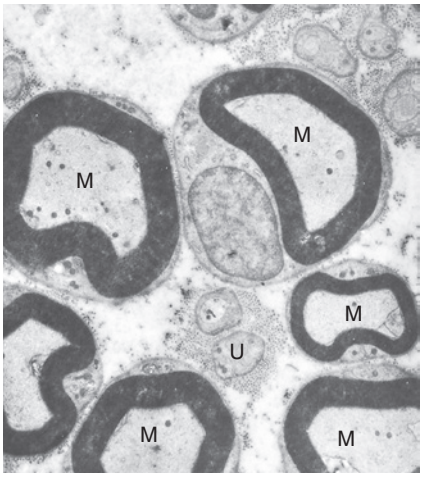

FIG. 12-19 Electron micrograph of the apical pulp of a young canine tooth, showing in cross section myelinated nerve axons (M) within Schwann cells. Smaller, unmyelinated axons (U) are enclosed singly and in groups by Schwann cells. (Courtesy Dr. David C. Johnsen, School of Dentistry, Case Western Reserve University.)

the foramen in close association with arterioles and venules (Fig. 12-18). Each of the nerves entering the pulp is invested within Schwann cells, and the A fibers acquire their myelin sheath from these cells. With the completion of root development, the myelinated fibers appear grouped in bundles in the central region of the pulp (Fig. 12-19). Most of the unmyelinated C fibers entering the pulp are located within these fiber bundles; the remainder is situated toward the periphery of the pulp (see Fig. 12-17).[307] It should be noted that single neurons have been reported to innervate the pulps of multiple teeth in animal studies.[155] Assuming a similar innervation pattern in humans, this finding may partially explain why patients often have difficulty localizing pulpal pain to a specific tooth. An alternative explanation to this clinical observation is that the pulp has a relatively low density of proprioceptors, and thus

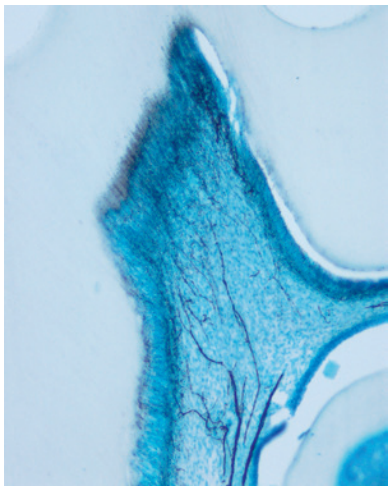

FIG. 12-20 Histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows the distribution of sensory nerves in a rat molar. Nerves enter the coronal pulp in bundles and ramify in a network beneath the odontoblasts (i.e., plexus of Raschkow), before entering between the odontoblasts and the inner part of dentin.

patients have difficulty identifying the inflamed tooth until the inflammation reaches the periradicular tissue, which is highly innervated with proprioceptors. This is discussed in greater detail in Chapter 17.

In the human premolar, the number of unmyelinated axons entering the tooth at the apex reached a maximal number shortly after tooth eruption.[170] At this stage, an average of 1800 unmyelinated axons and more than 400 myelinated axons were found, although in some teeth fewer than 100 myelinated axons were present. Five years after eruption, the number of A fibers gradually increased to more than 700. The relatively late appearance of A fibers in the pulp may help to explain why the electric pulp test tends to be unreliable in young teeth, as A fibers are more easily electrically stimulated than C fibers.[108]

A quantitative study of axons 1 to 2 mm coronal to the root apex of fully developed human canine and incisor teeth[170] reported a mean of about 360 myelinated axons in canines and incisors, whereas there were 1600 to 2200 unmyelinated axons. However, this does not reflect the actual number of neurons supporting a single tooth, because multiple branching of the axons may occur in the peripheral tissues. Overall, approximately 80% of the axons were unmyelinated fibers.[169,170]

The nerve bundles pass upward through the radicular pulp together with blood vessels (see Fig. 12-18). Once they reach the coronal pulp, they fan out beneath the cell-rich zone, branch into smaller bundles, and finally ramify into a plexus of single-nerve axons known as the *plexus of Raschkow* (Fig. 12-20). Full development of this plexus does not occur until the final stages of root formation.[90] It has been estimated that each axon entering the pulp sends at least eight branches to the plexus of Raschkow. There is prolific branching of the fibers in the plexus, producing a tremendous overlap of receptor fields.[143,267,268,269,273] It is in the plexus that the A fibers emerge from their encircling Schwann cells and branch repeatedly to form the subodontoblastic plexus. Finally, terminal axons pass between the odontoblasts as free nerve endings (Figs. 12-21 and 12-22). The extent to which dentin is innervated has been the subject of numerous investigations.[42,45,46,49,90,215] With the exception of the innervation of

FIG. 12-21 Detailed histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows the distribution of sensory nerves in the odontoblast layer of a rat molar. (Courtesy Dr. Inge Fristad, Department of Clinical Dentistry, University of Bergen.)

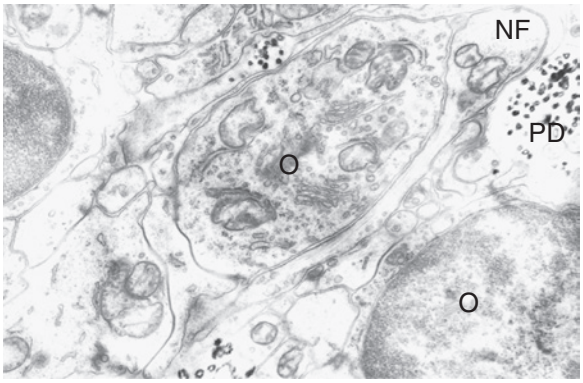

FIG. 12-22 Unmyelinated nerve fiber (NF) without a Schwann cell covering located between adjacent odontoblasts (O) overlying pulp horn of a mouse molar tooth. Predentin (PD) can be seen at upper right. Within the nerve, there are longitudinally oriented fine neurofilaments, microvesicles, and mitochondria. (From Corpron RE, Avery JK: The ultrastructure of intradental nerves in developing mouse molars, *Anat Rec* 175:585, 1973.)

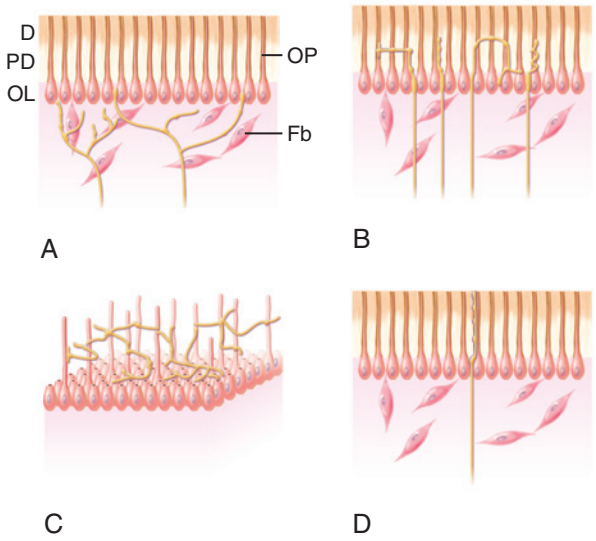

FIG. 12-23 Schematic drawing showing distribution of nerve fibers in the dentin-pulp border zone. **A,** Fibers running from the subodontoblastic plexus to the odontoblast layer. *D,* dentin; *Fb,* fibroblast; *OL,* odontoblast layer; *OP,* odontoblast process; *PD,* predentin. **B,** Fibers extending into the dentinal tubules in the predentin. **C,** Complex fibers that branch extensively in the predentin. **D,** Intratubular fibers extending into the dentin.

dentinal tubules, discussed later in this chapter, the bulk of dentin is devoid of sensory nerve fibers. This offers an explanation as to why pain-producing agents (e.g., potassium chloride) do not always elicit pain when applied to exposed dentin. Similarly, application of topical anesthetic solutions to dentin does not decrease its sensitivity. A high concentration of lidocaine solution is needed to block the response of intradental nerves to mechanical stimulation of the dentin.[4]

One investigator[131] studied the distribution and organization of nerve fibers in the dentin-pulp border zone of human teeth. On the basis of their location and pattern of branching, several types of nerve endings were described (Fig. 12-23). Some fibers were found running from the subodontoblastic nerve plexus toward the odontoblast layer. However, these fibers do not reach the predentin; they terminate in extracellular spaces in the cell-rich zone, the cell-poor zone, or the odontoblast layer. Other fibers extend into the predentin and run through a dentinal tubule in close association with an odontoblast process. Most of these intratubular fibers extend into the dentinal tubules for only a few micrometers, but a few may penetrate as far as 100 µm (see Fig. 12-20). The area covered by a single such terminal complex often reaches thousands of square micrometers.[131,268]

Intratubular nerve endings are most numerous in the area of the pulp horns, where as many as 40% of the tubules may contain fibers.[46,215] The number of intratubular fibers decreases in other parts of the dentin, and in root dentin only about 1% of dentinal tubules contain fibers. This notion has been challenged in a study that stained pulps for protein gene-product 9.5, a specific marker for nerves.[230] In that study, root dentin appeared to be as well innervated as coronal dentin. The anatomic relationship between the odontoblast processes and sensory nerve endings has led to much speculation as to the functional relationships between these structures, if any.[45] When present, nerve fibers lie in a groove or gutter along the surface of the odontoblast process, and toward their terminal ends, they twist around the process like a corkscrew. The cell membranes of the odontoblast process and the nerve fiber are closely approximated and run closely parallel for the length of their proximity, but they are not synaptically linked.[160]

Although it may be tempting to speculate that the odontoblasts and their associated nerve axons are functionally interrelated and that together they play a role in dentin sensitivity, there is a paucity of evidence supporting this hypothesis. If the odontoblast were acting as a classic receptor cell,* it would have chemical, electric, or mechanical communication with the adjacent nerve fiber. However, researchers have been unable to find classical anatomic structures (e.g., synaptic junctions) that could functionally couple odontoblasts and

*A receptor cell is a non–nerve cell capable of exciting adjacent afferent nerve fibers. Synaptic junctions connect receptor cells to afferent nerves.

nerve fibers together. With regard to the membrane properties of odontoblasts, it has been reported that the membrane potential of the odontoblast is low (−24 to −30 mV),[198,227] and that the cell does not respond to electric stimulation.[198,406] It would appear that the odontoblast does not possess the properties of an excitable cell. Further, the sensitivity of dentin is not diminished after disruption of the odontoblast layer.[37,216] It is still possible that odontoblasts could modulate neuronal function via alteration in sodium channel activity or the release of paracrine factors that diffuse to the closely approximated nerve terminal.

Another study showed that a reduction in pulpal blood flow, induced by stimulation of sympathetic fibers leading to the pulp, results in depressed excitability of pulpal A fibers.[82] The excitability of C fibers is less affected than that of A fibers by a reduction in blood flow.[365]

Of clinical interest is the evidence that nerve fibers of the pulp may be resistant to necrosis[85,255] because their cell bodies are found in ganglia outside the pulp. Because nerve bundles in general are more resistant to autolysis than other tissue elements, even in degenerating pulps, C fibers might still be able to respond to noxious stimulation. It may be that C fibers remain excitable even after blood flow has been compromised in the diseased pulp, as C fibers are often able to function in the presence of hypoxia.[365] This may explain why instrumentation of the root canals of apparently nonvital teeth sometimes elicits pain. On the other hand, histologic studies on nonvital teeth failed to demonstrate high levels of innervation, leading to the suggestion that pain may be due to the transfer of noxious chemicals to terminals located in periapical tissues.[255]

Steps and Mechanisms in Pain Perception

When activated by a stimulus sufficient to cause tissue damage or release of inflammatory mediators, nerve endings in the pulp and periradicular tissues begin to send bursts of messages to the central nervous system (CNS) that may eventually be perceived as pain. The anatomic pathway for this transmission of information has been fairly well established, and it is tempting to view the perception of pain of orofacial origin as a simple graded response to the intensity of the stimulus. However, researchers have come to realize that the pain system is a complex, multilevel system that begins with the *detection* of tissue-damaging stimuli in the periphery, the *processing* of that input at the level of the medullary spinal cord, and the *perception* of what is felt as pain in higher brain regions such as the cerebral cortex (Fig. 12-24). After a noxious stimulus is detected in the periphery, there is ample opportunity for a great deal of endogenous and possibly exogenous modification of the message prior to its ultimate perception. The clinician deals with all three levels of the pain system in diagnosing and treating odontalgia, and a practitioner with a basic understanding of each level will be able to recognize therapeutic opportunities and apply effective pain control methods.

Detection: The First Step in Pain Perception

Various types of peripheral neurons are found in the trigeminal system, including large-diameter, heavily myelinated Aα, Aβ, and Aγ fibers associated with motor, proprioception, touch, pressure, and muscle spindle stretch functions. But it is the

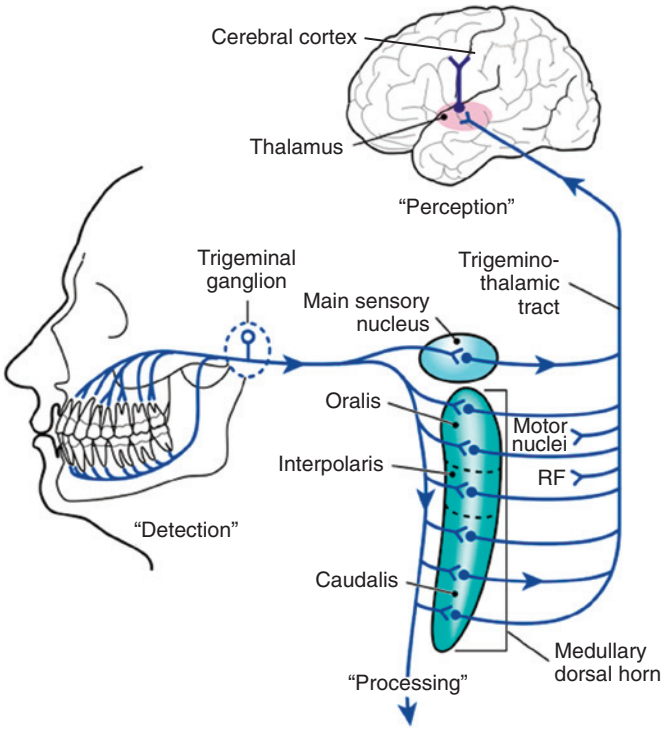

FIG. 12-24 Schematic diagram of the pathway of transmission of nociceptive information from the orofacial region. The trigeminal pain system is a complex multilevel system that begins with the *detection* of tissue damaging stimuli in the periphery, the *processing* of that input at the level of the medullary spinal cord, and the final *perception* of what is felt as pain in the cerebral cortex. There is growing appreciation for the concept that once a noxious stimulus is detected in the periphery, there is the opportunity for a great deal of modification of the message prior to ultimate perception.

smaller, less myelinated Aδ and yet smaller and unmyelinated C fibers that conduct information likely to be perceived as pain. These two classes of pain-sensing nerve fibers, or *nociceptors*, are both found in the tooth pulp, but there are three to eight times more unmyelinated C fibers than Aδ fibers.[41,45,170,371] It should be noted that this classification system is based purely on the size and myelination of the neurons and does not necessarily indicate function. For example, another class of pulpal C fibers are the postganglionic sympathetic efferents found in association with blood vessels, where they regulate pulpal blood flow[2,187,299,300] and may also influence the activity of peripheral nociceptors (for reviews, see Perl[296] or Hargreaves[137]). Because most pulpal sensory fibers are nociceptive, their terminal branches are free nerve endings, and physiologic stimulation by any modality (temperature, hyperosmotic fluids) results in the perception of pure pain, which can be difficult for patients to localize. Under experimental conditions, electrical stimulation can result in a *prepain* sensation that is also difficult to localize. Once inflammation has extended to the periodontal ligament, which is well endowed with Aβ discriminative touch receptors, localization of pain is more predictable with light mechanical stimuli such as the percussion test.

In the normal uninflamed pulp and periradicular tissues, a noxious stimulus causes depolarization of nociceptors sufficient to generate action potentials by means of the opening of voltage-gated sodium channels (Na_v). After generation of an action potential, not only is information sent to the CNS, but

also in an *antidromic* fashion (i.e., in the reverse direction of the impulse) in which proinflammatory neuropeptides such as substance P (SP), calcitonin gene-related peptide (CGRP), neurokinins, and the classic neurotransmitter, glutamate, are released from afferent terminals in the pulp and periradicular tissues.

Neuropeptides

Of immense importance in pulp biology is the presence of neuropeptides in pulpal nerves.[42,46,49,349] Pulpal nerve fibers contain neuropeptides such as calcitonin gene–related peptide (CGRP) (see Figs. 12-18, 12-20, and 12-21),[41] substance P (SP),[268,285] neuropeptide Y (see Fig. 12-16), neurokinin A (NKA),[12] and VIP.[228,389] In rat molars, the largest group of intradental sensory fibers contains CGRP. Some of these fibers also contain other peptides, such as SP and NKA.[42,45] Release of these peptides can be triggered by numerous stimuli, including tissue injury,[12] complement activation, antigen-antibody reactions,[280] or antidromic stimulation of the inferior alveolar nerve.[285,282] Once released, vasoactive peptides produce vascular changes that are similar to those evoked by histamine and bradykinin (i.e., vasodilation).[282] In addition to their neurovascular properties, SP and CGRP contribute to inflammation and promote wound healing.[31,369] The release of CGRP can be modified by sympathetic agonists and antagonists,[115,138] offering the promise of using such agonists to treat dental pain. This latter point is important because clinicians use sympathetic agonists every day—the vasoconstrictors present in local anesthetic solutions may have direct effects on inhibiting dental nerve activity. Local anesthetic reduction in pain may be due to the actions of both the local anesthetic and the vasoconstrictor.[137,138] In cats, capsaicin acutely activates and chronically blocks the TRPV1-expressing classes of C and A-delta nociceptors in the pulp.[163] In addition, the chronic application of capsaicin ointment to skin has been shown to relieve pain in patients, suggesting that clinical trials evaluating chronic application of capsaicin for treating pulpal or periradicular pain may be of value.

Antidromic stimulation of nerves (i.e., toward the peripheral terminals) simply means that the afferent barrage goes in the opposite direction of the orthograde stimulation (toward the CNS). Normally, sensory nerves are stimulated at their peripheral terminations, and their action potentials then travel toward the brain. In antidromic nerve stimulation, the sensory nerve is usually cut. The peripheral end of the nerve is then electrically stimulated. This causes an action potential to travel backward toward the periphery, which causes release of neuropeptides in the pulp.[285] All branches of the nerve also depolarize and release neuropeptides (the so-called axon reflex).[322]

It has been reported[246,285] that mechanical stimulation of dentin produces vasodilation within the pulp, presumably by causing the release of neuropeptides from intradental sensory fibers (neurogenic inflammation).[285] Electric stimulation of the tooth has a similar effect.[152] The pulpal concentrations of CGRP, SP, and NKA are elevated in painful human teeth over healthy control teeth extracted for orthodontic reasons.[12] These peptides are also elevated in pulps beneath advancing carious lesions.[310,311]

Pulp Testing

The electric pulp tester delivers a current sufficient to overcome the resistance of enamel and dentin and stimulate the sensory A fibers at the dentin-pulp border zone. Smaller C fibers of the pulp do not respond to the conventional pulp tester because significantly more current is needed to stimulate them.[267] Bender and associates[17] found that in anterior teeth, the optimal placement site of the electrode is the incisal edge, as the response threshold is lowest at that location and increases as the electrode is moved toward the cervical region of the tooth.

Cold tests using carbon dioxide (CO_2) snow or liquid refrigerants and heat tests employing heated gutta-percha or hot water activate hydrodynamic forces within the dentinal tubules, which in turn excite the intradental A fibers. C fibers are generally not activated by these tests unless they produce injury to the pulp. It has been shown that cold tests do not injure the pulp.[108] Heat tests have a greater potential to produce injury, but if the tests are used properly, injury is not likely.

Sensitivity of Dentin

The mechanisms underlying dentin sensitivity have been a subject of interest for many years. How are stimuli relayed from the peripheral dentin to the sensory terminals located in the region of the dentin-pulp border zone? Converging evidence indicates that movement of fluid in the dentinal tubules is the basic event in the arousal of dentinal pain.[35,37,263,385,387] It now appears that pain-producing stimuli, such as heat, cold, air blasts, and probing with the tip of an explorer, have in common the ability to displace fluid in the tubules.[35,246] This is referred to as the *hydrodynamic mechanism of dentin sensitivity*. The hydrodynamic theory suggests that dentinal pain associated with stimulation of a sensitive tooth ultimately involves mechanotransduction. Classical mechanotransducers have been recognized on pulpal afferents, providing a mechanistic support to this theory.[149] Thus, fluid movement in the dentinal tubules is translated into electric signals by receptors located in the axon terminals innervating dentinal tubules. Using single-fiber recording techniques, a positive correlation was found between the degree of pressure change and the number of nerve impulses leaving the pulp (Figs. 12-25 and 12-26).[246,266,384] Thus, the outward fluid movements (negative pressure) produce a much stronger nerve response than inward movements.[246,388]

In experiments on humans, brief application of heat or cold to the outer surface of premolar teeth evoked a painful response before the heat or cold could have produced temperature changes capable of activating sensory receptors in the underlying pulp.[266,372] The evoked pain was of short duration: 1 or 2 seconds. The thermal diffusivity of dentin is relatively low, yet the response of the tooth to thermal stimulation is rapid, often less than 1 second. Evidence suggests that thermal stimulation of the tooth results in a rapid movement of fluid into the dentinal tubules. This results in activation of the sensory nerve terminal in the underlying pulp. Presumably, heat expands the fluid within the tubules faster than it expands dentin, causing the fluid to flow toward the pulp, whereas cold causes the fluid to contract more rapidly than dentin, producing an outward flow. It is speculated that the rapid movement of fluid across the cell membrane of the axon terminal activates a mechanosensitive receptor, similar to how fluid movement activates hair cells in the cochlea of the ear. All axon terminals have membrane channels through which charged ions pass, and this initial receptor current, if sufficient, can trigger voltage-gated sodium channels to depolarize the cell, leading

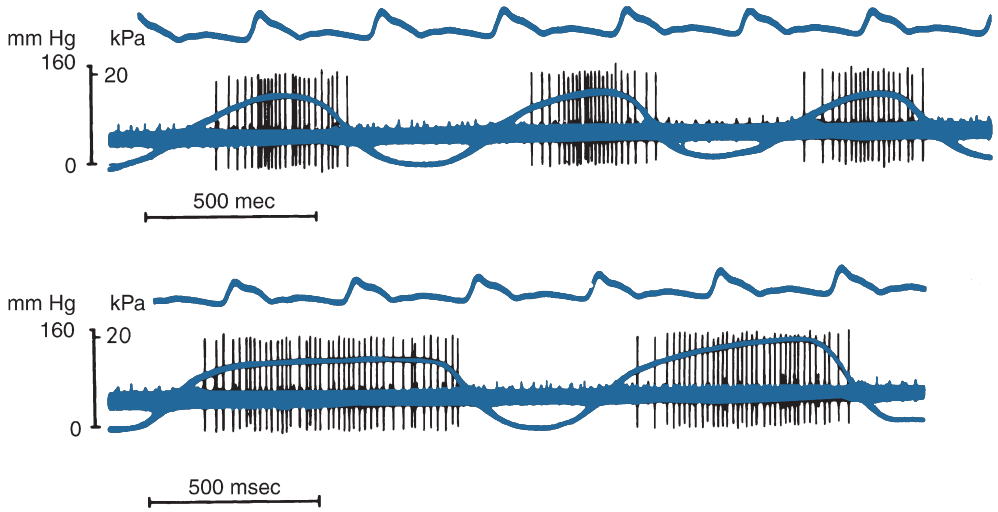

FIG. 12-25 Response of a single dog pulp nerve fiber to repeated hydrostatic pressure stimulation pulses. Lower solid wavy line of each recording indicates the stimulation pressure applied to the pulp. Upper line (kPa) is the femoral artery blood pressure curve recorded to indicate the relative changes in the pulse pressure and the heart cycle. (Modified from Närhi M: Activation of dental pulp nerves of the cat and the dog with hydrostatic pressure, *Proc Finn Dent Soc* 74[suppl 5]:1, 1978.)

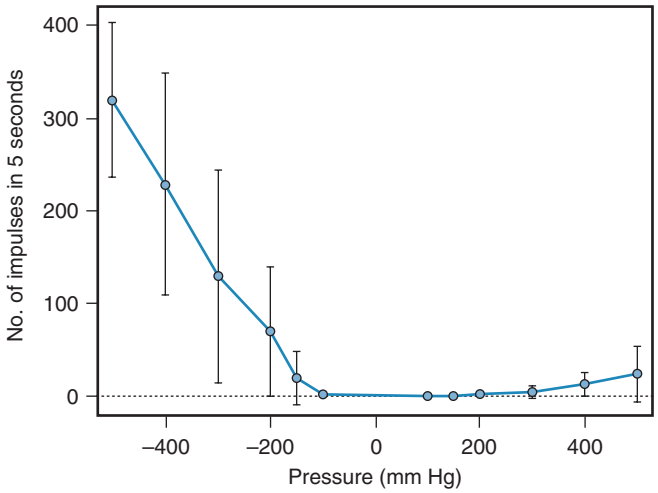

FIG. 12-26 The average number of impulses recorded from dentine after application of pressure stimuli to the dentine. More impulses are recorded after application of negative pressure (outward fluid flow) than after positive pressure (inward fluid flow). (From Vongsavan N, Matthews B: The relationship between the discharge of intradental nerves and the rate of fluid flow through dentine in the cat, *Arch Oral Biol.* 52:643, 2007.)

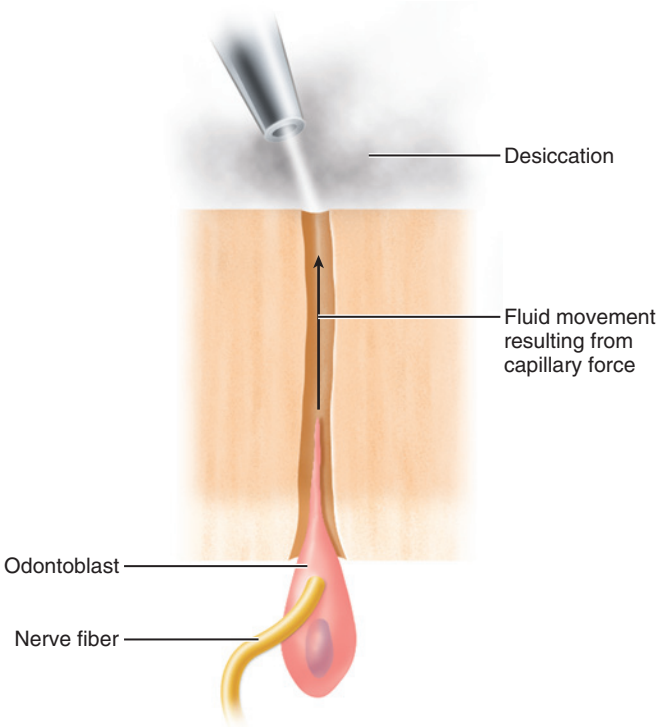

FIG. 12-27 Diagram illustrating movement of fluid in the dentinal tubules resulting from the dehydrating effect of a blast of air from an air syringe.

to a barrage of impulses to the brain. Some ion channels are activated by voltage, some by chemicals, and some by mechanical pressure.[246,263,268] In the case of pulpal nerve fibers that are activated by hydrodynamic forces, pressure would be transduced, gating mechanosensitive ion channels.

The dentinal tubule is a capillary tube with an exceedingly small diameter.*[111] The physical properties of capillarity are significant because fluid force increases as the diameter is reduced. If fluid is removed from the outer end of exposed dentinal tubules by dehydrating the dentinal surface with an air blast or absorbent paper, capillary forces produce a rapid outward movement of fluid in the tubule (Fig. 12-27). According to Brännström,[35] desiccation of dentin can theoretically cause dentinal fluid to flow outward at a rate of 2 to 3 mm/sec. In addition to air blasts, dehydrating solutions containing hyperosmotic concentrations of sucrose or calcium chloride can produce pain if applied to exposed dentin.

*To appreciate fully the dimensions of dentin tubules, understand that the diameter of the tubules (about 1 μm) is much smaller than that of red blood cells (about 7 μm). The thickness of coronal dentin is about 3 mm, so each tubule is 3000 μm long but only 1 μm in diameter. Thus, each tubule is 3000 diameters long.

Investigators have shown that it is the A fibers rather than the C fibers that are activated by hydrodynamic stimuli (e.g., heat, cold, air blasts) applied to exposed dentin.[265,273] However, if heat is applied long enough to increase the temperature of the dentin-pulp border by several degrees Celsius, then C fibers may respond, particularly if the heat produces injury. It seems that the A fibers are mainly activated by a rapid displacement of the tubular contents.[263] Slow heating of the tooth produced no response until the temperature reached 111° F (43.8° C), at which time C fibers were activated, presumably because of heat-induced injury to the pulp. These C fibers are called *polymodal nociceptors* because they contain numerous receptors that confer the ability to detect and respond to many different types of stimuli.[268,269] Capsaicin, the pungent active ingredient in hot peppers, is known to simulate C fibers and a subset of A-delta fibers.[163] Capsaicin activates a receptor termed the "transient receptor potential, subtype vanilloid 1" or TRPV1.[58] The TRPV1 receptor is expressed primarily on a major subclass of nociceptors and responds to heat (> 110° F [> 43° C]), certain inflammatory mediators, and acid (pH < 6). Thus TRPV1 has been considered a molecular integrator of polymodal noxious stimuli.[277] The ability of the TRPV1 antagonist, capsazepine, to inhibit acid-, heat-, and capsaicin-activated trigeminal neurons[222] has led to the development of new drugs (i.e., TRPV1 antagonists) for the treatment of pulpal pain. Eugenol is known to activate and ultimately desensitize TRPV1, and this may explain the anodyne action of zinc oxide eugenol temporary restorations.[415]

It has also been shown that pain-producing stimuli are more readily transmitted from the dentin surface when the exposed tubule apertures are open[157,171] and the fluid within the tubules is free to flow outward.[171,246,387] For example, acid treatment of exposed dentin to remove the smear layer opens the tubule orifices and makes the dentin much more responsive to stimuli such as air blasts and probing.[157,269]

Perhaps the most difficult phenomenon to explain is dentinal pain associated with light probing of dentin. Even light pressure of an explorer tip can produce strong forces.* These forces have been shown to mechanically compress dentin and close open tubule orifices with a smear layer that causes sufficient displacement of fluid to excite the sensory receptors in the underlying pulp (Fig. 12-28).[54,55] Considering the density of the tubules in which hydrodynamic forces would be generated by probing, multiple nerve endings would be simultaneously stimulated when a dental explorer is scratched across dentin. Another newly suggested explanation is that the nerves innervating teeth are special in nature and that low-threshold mechanoreceptors are signaling nociceptive input in teeth.[103] Similar low-threshold mechanoreceptors convey tactile sensation in skin. The authors used the term low-threshold "algoneurons" for these nerves. This theory is not in conflict with the hydrodynamic theory but may help to explain the sensation of pain felt after weak mechanical stimuli like air puffs and water spray.

Another example of the effect of strong hydraulic forces that are created within the dentinal tubules is the phenomenon of odontoblast displacement. In this reaction, the nuclei and cell

*A force of 44 cM (44 g) applied to an explorer having a tip 40 μm in diameter would produce a pressure of 2437 MPa on the dentin.[54] This is far in excess of the compressive strength of dentin, listed as 245MPa, as evidenced by the shallow grooves lined by smear layers created in dentin using this force.[55]

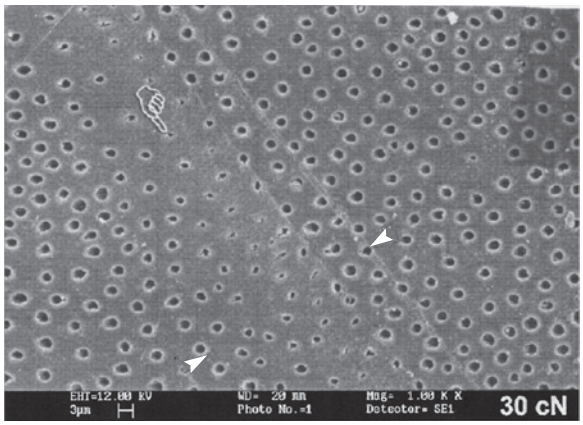

FIG. 12-28 Scanning electron micrograph of a shallow groove *(between white arrowheads)* created in polished dentin by a dental explorer tine under a force of 30 g (30 cN). Note the partial occlusion of the tubules by smeared matrix. (From Camps J, Salomon JP, Meerbeek BV, et al: Dentin deformation after scratching with clinically relevant forces, *Arch Oral Biol* 48:527, 2003.)

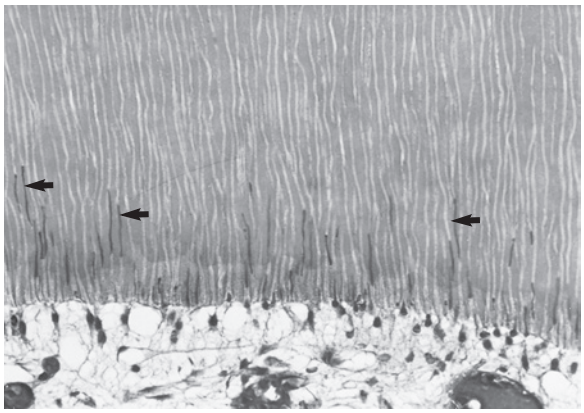

FIG. 12-29 Odontoblasts *(arrows)* displaced upward into the dentinal tubules.

bodies of odontoblasts are displaced upward in the dentinal tubules, presumably by a rapid movement of fluid in the tubules produced when exposed dentin is desiccated, as with the use of an air syringe or cavity-drying agents (Fig. 12-29).[79,202] Such cellular displacement results in the destruction of odontoblasts, because cells thus affected soon undergo autolysis and disappear from the tubules. Displaced odontoblasts may eventually be replaced by stem cells that migrate from the cell-rich zone of the pulp, as discussed later in the chapter.

The hydrodynamic theory can also be applied to an understanding of the mechanism responsible for hypersensitive dentin.[36,37] There is controversy regarding whether exposed dentin is simply sensitive or becomes truly hypersensitive.[268,269,273] Growing evidence indicates that new sodium channels responsible for activating nerves are expressed in nerve tissue exposed to inflammation.[119,137,308] An increase in the density of sodium channels or their sensitivity may contribute to dentinal hypersensitivity. Hypersensitive dentin is also associated with the exposure of dentin normally covered by cementum or enamel. The thin layer of cementum is frequently lost as gingival recession exposes cementum to the oral environment. Cementum is subsequently worn away by brushing, flossing, or using toothpicks. Once exposed, the dentin

may respond to the same stimuli to which any exposed dentin surface responds (e.g., mechanical pressure, dehydrating agents). Although the dentin may at first be very sensitive, within a few weeks the sensitivity usually subsides. This desensitization is thought to occur as a result of gradual occlusion of the tubules by mineral deposits, thus reducing hydrodynamic forces. In addition, deposition of reparative dentin over the pulpal ends of the exposed tubules probably also reduces sensitivity, because reparative dentin is less innervated by sensory nerve fibers.[44] However, some hypersensitive dentin does not spontaneously desensitize, so hypersensitivity may be caused by either inflammatory changes in the pulp or mechanical changes in the patency of dentinal tubules.

Currently, the treatment of hypersensitive teeth is directed toward reducing the functional diameter of the dentinal tubules to limit fluid movement. Four possible treatment modalities[287,373,389] can accomplish this objective:

1. Formation of a smear layer on the sensitive dentin by burnishing the exposed root surface[157,268,269]
2. Application of agents, such as oxalate compounds, that form insoluble precipitates within the tubules[157,292]
3. Application of agents such as hydroxyethyl methacrylate (HEMA) with or without glutaraldehyde that are thought to occlude tubules with precipitated plasma proteins in dentinal fluid[325]
4. Application of dentin bonding agents to seal off the tubules[303]

Dentin sensitivity can be modified by laser irradiation, but clinicians must be concerned about its effect on the pulp.[346,357]

Peripheral Sensitization

Following repeated noxious stimuli, both A and polymodal C fiber nociceptors undergo a process of sensitization manifested by three obvious changes in response patterns. First, firing thresholds may decrease, so that previously non-noxious stimuli may trigger discharges, contributing to the sensation of pain *(allodynia)*. Second, after-discharges may occur, so that noxious stimuli may produce an even greater increase in the perceived intensity of pain *(hyperalgesia)*. And third, firing may occur spontaneously, contributing to the development of spontaneous pain. These changes are often seen in endodontic pain patients and may be explained in part by the effects of chemical mediators released into inflamed pulp and periradicular tissues. Such mediators include substances produced from damaged tissues, agents of vascular origin, and peptides released from the nerve fibers themselves (Table 12-3). Other mechanisms of peripheral sensitization are listed in Box 12-1.

Hyperalgesia and Allodynia

Three characteristics of hyperalgesia are (1) spontaneous pain, (2) a decreased pain threshold, and (3) an increased response to a painful stimulus.[137] The peripheral mechanisms for these symptoms include a decrease in firing threshold, an increase in responsiveness to noxious stimuli, and development of spontaneous discharges of nociceptors. All three of these characteristics can be seen in patients experiencing inflammatory pain of pulpal origin (Table 12-4). It is recognized that hyperalgesia can be produced by sustained inflammation, as in the case of sunburned skin. Clinical observation has shown that the sensitivity of dentin often increases when the underlying pulp becomes acutely inflamed, and the tooth may be more difficult to anesthetize. This is due in part to the upregulation of tetrodotoxin-resistant (TTX-resistant) sodium channels in

BOX 12-1

Peripheral Mechanisms Contributing to Hyperalgesia and Allodynia

Mechanism

- Composition and concentration of inflammatory mediators[142]
- Changes in afferent fiber: activation and sensitization[199,243,324]
- Changes in afferent fiber: sprouting[51]
- Changes in afferent fiber: proteins[51,119,409]
- Tissue pressure[264,265]
- Tissue temperature[250]
- Sympathetic primary afferent fiber interactions[166,210,295]
- Aβ fiber plasticity[272]

Modified from Hargreaves KM, Swift JQ, Roszkowski MT, et al: Pharmacology of peripheral neuropeptide and inflammatory mediator release, *Oral Surg Oral Med Oral Pathol* 78:503, 1994.

TABLE 12-3

Effect of Inflammatory Mediators on Nociceptive Afferent Fibers

Mediator	Effect on Nociceptors	Effect on Human Volunteers
Potassium[174]	Activate	++
Protons[218,343]	Activate	++
Serotonin[16,174]	Activate	++
Bradykinin[16,174,211]	Activate	+++
Histamine[174]	Activate	+
Tumor necrosis factor-α	Activate	?
Prostaglandins[27]	Sensitize	±
Leukotrienes[27,229]	Sensitize	±
Nerve growth factor[213,297]	Sensitize	++
Substance P[132]	Sensitize	±
Interleukin 1[99]	Sensitize (?)	?

+, Positive; ++, very positive; +++, extremely positive; ±, equivalent; ?, unknown.
Modified from Fields H: *Pain,* New York, 1987, McGraw-Hill.

TABLE 12-4

Signs of Hyperalgesia and Allodynia and Endodontic Diagnostic Tests

Signs of Hyperalgesia	Related Diagnostic Tests or Symptoms
Spontaneous pain	Spontaneous pain
Reduced pain threshold	Percussion test, palpation test, throbbing pain
Increased response to painful stimuli	Increased response to pulp test (electric or thermal test)

From Hargreaves KM, Swift JQ, Roszkowski MT, et al: Pharmacology of peripheral neuropeptide and inflammatory mediator release, *Oral Surg Oral Med Oral Pathol* 78:503, 1994.

inflamed neural tissue.[119,137] NGF seems to play an important role in hyperalgesia. NGF regulates chronic inflammatory hyperalgesia by controlling gene expression in sensory neurons,[274] including genes involved in inflammatory hyperalgesia in the dental pulp.[74] Although a precise explanation for hyperalgesia is lacking, apparently localized elevations in tissue pressure and inflammatory mediators that accompany acute inflammation play an important role.[151,154,344,362,379] Clinically, we know that when the pulp chamber of a painful tooth with an abscessed pulp is opened, drainage of exudate soon produces a reduction in the level of pain. This suggests that mechanical stimuli may contribute substantially to pain during inflammatory hyperalgesia.

From a clinical point of view, *thermal allodynia* is the term that best describes a patient whose chief complaint is "I have pain when I drink cold beverages." *Mechanical allodynia* is involved when the chief complaint is "It now hurts when I bite on this tooth." These previously non-noxious stimuli now cause the perception of pain. Hyperalgesia is manifested in endodontic pain patients when noxious stimuli (e.g., refrigerant sprays or carbon dioxide snow used in the cold test) produce much more pain than they would in teeth with normal pulp tissues. Spontaneous pain involves episodes of pain that seem to be unprovoked. All these changes can be partly explained by sensitization of peripheral nerve endings in the pulp and periradicular tissues.

Many silent nerve fibers are present in the normal pulp[268,269] and are termed *silent* because they are not excited by ordinary external stimuli. Once they are sensitized through pulpal inflammation, they begin to respond to hydrodynamic stimuli.[46,268,269,273] This phenomenon may provide an additional mechanism for dentin hypersensitivity. The molecular mechanisms of this activation are not known in detail but involve upregulation of numerous genes and their products.[12,45,119]

Inflammatory Mediators

Among the best characterized of the inflammatory mediators are the prostaglandins (PGs), which are derived from arachidonic acid via the action of the cyclooxygenase (COX) enzyme systems. The human COX enzyme is known to exist in at least two forms, COX-1 and COX-2. COX-1 is constitutively expressed and produces PGs that are involved in basic housekeeping functions such as cytoprotection in the stomach, regulation of blood flow in the kidneys, and the formation of thromboxane A_2. The formation of thromboxane A_2 can ultimately lead to platelet aggregation; therefore, inhibition of thromboxane A_2 should decrease platelet aggregation. COX-2 is inducible, synthesized in inflamed tissues (including dental pulp),[262] and is important in the production of the proinflammatory PGs as well as the vasodilating prostacyclin (PGI_2). Although they do not produce pain if applied alone, PGs are known to sensitize peripheral nociceptors, which increases the algogenic (pain-producing) properties of serotonin and bradykinin.[76,124] The exact mechanism by which PGs increase neuronal excitability is not clear, but there is a growing body of evidence to suggest that they activate the PG E receptor subtypes **EP2** and EP3 in the trigeminal system[293] and exert their effects by regulating the activity of certain ion channels,[394] including voltage-gated sodium channels (for a review, see England[86]). For example, application of prostaglandin E_2 (PGE_2) to isolated dorsal root ganglion neuronal somata more than doubles the responsiveness of certain sodium channels found predominantly on nociceptors—channels thought to be relatively resistant to lidocaine.[9,120] When administered to rats before an inflammatory insult, ibuprofen, a nonselective COX inhibitor, has been shown to block the increased expression of $Na_v1.7$ and $Na_v1.8$.[127,128] Therefore, if the concentrations of PGs in inflamed pulp and periradicular tissues can be decreased with nonsteroidal antiinflammatory drugs (NSAIDs) or corticosteroids, postoperative pain may be relieved; in addition, more profound local anesthesia may be achieved in patients with hyperalgesia of pulpal origin.[140,161,254] It is interesting to note that sensory neurons themselves are a source of PGs; during inflammation, the levels of PGE_2 appear to increase in dorsal root ganglia and spinal cord, suggesting that NSAIDs also have a central site of action (discussed in Chapter 4).[235,405]

Bradykinin (BK) is a proinflammatory mediator derived from circulating plasma proteins and also causes direct activation of nociceptive neurons, resulting in pain. Increased levels of BK have been demonstrated in the inflamed dental pulp,[207] and the presence of growth factors associated with inflammation (e.g., nerve growth factor) have been reported to cause an increase in the expression of mRNA encoding B1 and B2 receptors in primary cultures of rat trigeminal ganglia,[363] as well as other receptors such as TRPV1 and TRPA1.[74,168] The *transient receptor potential subtype V1* (TRPV1) is the "capsaicin receptor"; it plays a key role in mediating inflammatory pain. TRPA1 is expressed on capsaicin-sensitive neurons[74] and interacts with TRPV1.[314] Bradykinin likely increases the excitability of nociceptive neurons through its action on TRPV1 and TRPA1 (for review, see Tominaga et al[358]).

Cytokines are a diverse group of regulatory proteins synthesized and secreted by a variety of cell types, such as leukocytes, neurons, and glia. In particular, tumor necrosis factor α (TNF-α) and the interleukins IL-1β, IL-6, and IL-8 are thought to play a role in the neuroplastic changes that occur in nociceptors innervating inflamed tissues, leading to hyperalgesia.[197] Application of TNF-α rapidly sensitizes TRPV1,[183] contributing to activation of the capsaicin-sensitive class of nociceptors. All of the above are thought to exist in the inflamed pulp (for review, see Fouad[100]) and are thought to act at least in part by causing increased release of prostanoids.[347]

Painful Pulpitis

From the foregoing, it is apparent that pain associated with the stimulation of the A fibers does not necessarily signify that the pulp is inflamed or that tissue injury has occurred. Clinically, pain produced by A fibers in response to the hydrodynamic mechanism has a sharp or bright quality as contrasted with the dull, boring, or throbbing pain associated with C fibers. A fibers have a relatively low threshold of excitability to external stimuli,[246,267] and painful pulpitis is more likely to be associated with nociceptive C fiber activity indicative of pulpal tissue injury.[267-269,273] The clinician should carefully examine symptomatic teeth to rule out the possibility of hypersensitive dentin, cracked or leaky fillings, or fracture lines—each of which may initiate hydrodynamic forces—before establishing a diagnosis of reversible or irreversible pulpitis (see also Chapters 1 and 21).

Pain associated with an inflamed or degenerating pulp may be either provoked or spontaneous. The hyperalgesic pulp may demonstrate a lowered threshold of pain by responding to stimuli that usually do not evoke pain (allodynia), or the

pain may be exaggerated and persist longer than normal (hyperalgesia).[6] On the other hand, the tooth may commence to ache spontaneously in the absence of any external stimulus.[137] Spontaneous, unprovoked pain generally indicates a pulp that is seriously damaged and generally will not respond to noninvasive therapy.

Plasticity of Intradental Nerve Fibers

It has become apparent that the innervation of the tooth is a dynamic process in which the number, size, and cytochemistry of nerve fibers can change because of aging,[102,224,349] tooth injury,[42,45,46,49] and dental caries.[310] For example, in rats, nerve fibers sprout into inflamed tissue surrounding sites of pulpal injury, and the content of CGRP and SP increases in these sprouting fibers.[42,45,46,49,53] When inflammation subsides, the number of sprouts decreases. Fig. 12-30 compares the normal distribution of CGRP-immunoreactive sensory fibers in an adult rat molar with those beneath a shallow cavity preparation. The innervational pattern in normal and inflamed teeth is governed by neuronal growth factors. Neurotrophic and target-derived factors regulate neuronal structure, survival, and function and are important for the maintenance of neuronal phenotype characteristics. During development, all dental fibers appear to require nerve growth factor (NGF) and express its receptor, TrkA, at some stages,[244] whereas in adult teeth the large trigeminal neurons are potentially dependent only on dental pulp–derived, glial cell line–derived neurotrophic factor (GDNF); the smaller trigeminal neurons remain dependent on NGF.[200,305] This suggests that GDNF may function as a neurotrophic factor for the subset of larger neurons supporting the tooth, which apparently mediate mechanosensitive stimuli, whereas NGF is suggested to support neurons responsible for nociception.[259] NGF is the most extensively investigated among the trophic factors.[209] Binding of target-derived NGF is dependent on specific TrkA receptors located on the axonal surface, with subsequent internalization and transport to the cell body, where the effects are mediated.

Regulation of neural changes during inflammation seems to be a function of NGF expression.[47,53] NGF receptors are found on intradental sensory fibers and Schwann cells.[45] Evidence indicates that NGF is synthesized by fibroblasts in the coronal subodontoblastic zone (i.e., cell-rich zone), particularly in the tip of the pulp horn.[45] Maximal sprouting of CGRP- and SP-containing nerve fibers corresponds to areas of the pulp where there is increased production of NGF.[53] Fig. 12-31 shows the expression of NGF-mRNA in a pulp horn subjacent to cavity preparation.

It has been suggested that neuroimmune interactions take place in the dental pulp, because a coordinated increase of pulpal nerves and immune cells has been demonstrated.[173,319] In addition, recruitment of immunocompetent cells has been demonstrated in the dental pulp after electrical tooth stimulation.[66,106] Similar responses have been seen in periapical bone in rats with radicular pulpitis.[195] Neurogenic inflammation is generally thought to enhance healing, because denervated teeth show poorer healing following pulp exposures than innervated teeth.[42,50]

Another consideration in the neural response to inflammation is the possibility of a change in the distribution and activity of voltage-gated sodium channels. In particular, mice lacking the gene for $Na_v1.7$ show reduced painlike behaviors when treated with a variety of proinflammatory agents.[270] Also implicated in the altered firing characteristics of nociceptors innervating inflamed tissues are the sodium channels that are resistant to tetrodotoxin (TTX), the biotoxin found in the tetraodon pufferfish. The two main TTX-R sodium channels are $Na_v1.8$ and $Na_v1.9$, and both have been shown to be increased two- to fourfold in inflamed dental pulp collected

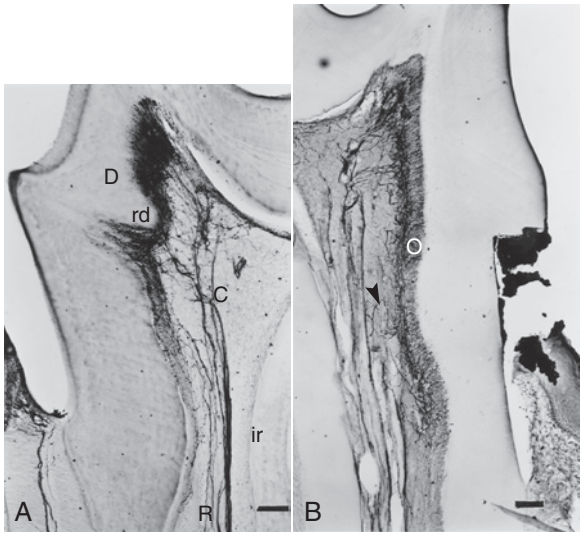

FIG. 12-30 A, Normal distribution of calcitonin gene–related peptide (CGRP)-immunoreactive sensory fibers in adult rat molar. Nerve fibers typically are unbranched in the root (R), avoid interradicular dentin (ir), and form many branches in coronal pulp (C) and dentin (D). Nerve distribution is often asymmetric, with endings concentrated near the most columnar odontoblasts (in this case on the left side of the crown). When reparative dentin (rd) forms, it alters conditions so that dentinal innervation is reduced (magnification ×75). B, Shallow class I cavity preparation on the cervical root of a rat molar was made 4 days earlier. Primary odontoblast (O) layer survived, and many new CGRP-immunoreactive terminal branches spread beneath and into the injured pulp and dentin. Terminal arbor can be seen branching *(arrowhead)* from a larger axon and growing into the injury site. Scale bar: 0.1 mm (magnification. *A,* ×45). (From Taylor PE, Byers MR and Redd PE: Sprouting of CGRP nerve fibers in response to dentin injury in rat molars. Brain Res 461:371-376, 1988.)

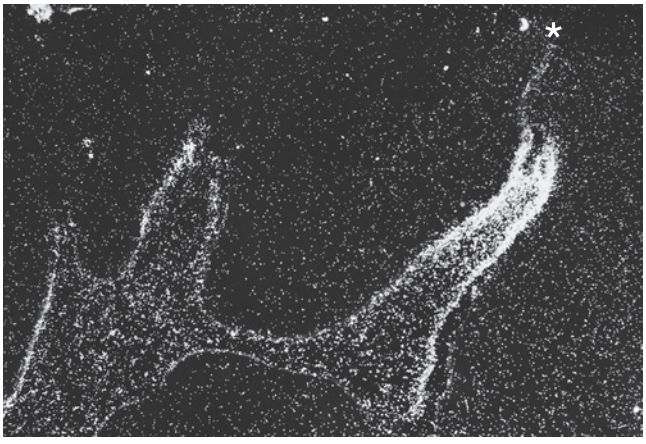

FIG. 12-31 NGF-mRNA is upregulated in the mesial pulp horn 6 hours after cavity preparation. (From Byers MR, Wheeler EF, and Bothwell M: Altered expression of NGF and p75 NGF-Receptor mRNA by fibroblasts of injured teeth precedes sensory nerve sprouting. Growth Factors 6:41-45, 1992.)

from patients with a diagnosis of irreversible pulpitis.[396,402] When exposed to PGE_2, neurons isolated from dorsal root ganglia cells have been shown to increase TTX-resistant sodium channel currents within minutes,[120] indicating an increased activation of existing channels, rather than de novo protein synthesis. These sodium channels are relatively resistant to lidocaine,[313] and this may explain the difficulty in achieving profound anesthesia in inflamed tissues (see also Chapter 4).[140]

Tissue Injury and Deafferentation

When a peripheral nerve is cut or crushed, an interruption of the afferent input to the CNS occurs, which is called *deafferentation*. It would be logical to assume that the result of deafferentation would be anesthesia of the formerly innervated area, but occasionally other symptoms may occur, which may surprisingly include pain. Following nerve injury, a dramatic shift in transcription of neuropeptides, receptors, and sodium channels has been documented. The bidirectional contact between the nerve cell and the peripheral target tissue is lost, and the neurons change into a state of either regeneration or neuronal cell death. The impact on neurons in the trigeminal ganglion is dependent on the injury site. A peripheral injury has less effect than a more centrally located one. However, even a small pulp exposure induces neuronal changes, both in the trigeminal ganglion and at the second-order neuronal level in the brain stem.[43,391] Because each single-rooted tooth contains about 2000 nerve fibers,[169,170] extirpation of the pulp is shown to cause both neurochemical and degenerative changes of their cell bodies in the gasserian (trigeminal) ganglion.[129,186,334] The central projection of these nerves to the spinal nucleus of the trigeminal nerve is also affected,[367] and there is evidence for transsynaptic changes[330,334] that are reflected in the sensory cortex. Even larger responses would be expected from tooth extraction, where both periodontal ligament and pulpal innervation are destroyed.

When an axon is severed peripherally, a complete degeneration of the cell bodies may not always occur.[392] Attempted regeneration by axonal sprouting may result in altered expression of various receptors, resulting in sensitivity to norepinephrine (via increased adrenergic receptor activity)[278] or acetylcholine (via increased cholinergic receptor activity),[72] sensitizing sensory neurons to autonomic activity. In addition, dorsal horn neurons, deprived of their normal sensory input, may begin to respond to other nearby afferents. Thus, normal inhibitory influences are reduced, and a widening of the sensory receptive field is produced, which can produce central sensitization (see Central Sensitization later in this chapter). *Phantom tooth pain* is another term often used synonymously with pain following deafferentation. Different reports suggest an incidence of persistent pain following pulpectomy to be in the range of 3% to 6%.[238,383]

Following tissue injury or tissue inflammation, extensive changes occur in the gene expression of sensory ganglion neurons and, by way of transsynaptic mechanisms, in their central projections.[45,268,269,334] One example is the upregulation of inducible gene transcription factors such as c-*fos*[43] and different subsets of sodium channels.[119] This is thought to result in alterations in threshold properties and the size of receptor fields. C-*fos* is not normally expressed in neurons in the brain stem, but chronic pulpitis causes prolonged increases in c-*fos* expression is some brain stem neurons (Fig. 12-32).[43]

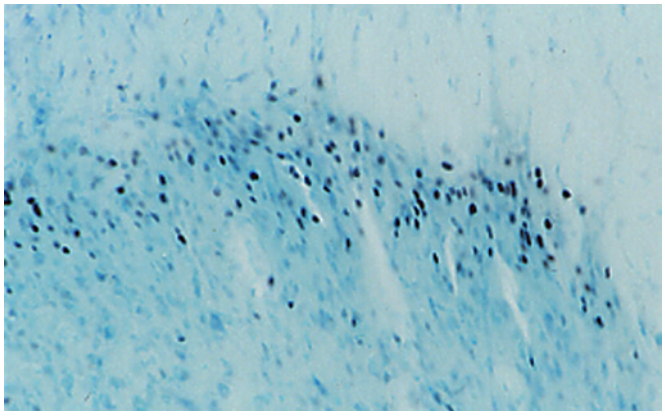

FIG. 12-32 The interstitial cells of the trigeminal tract of medullary dorsal horn express c-*fos* during chronic tooth pulp inflammation in rat. (Adapted from Byers MR, Chudler EH, Ladarola MJ: Chronic tooth pulp inflammation causes transient and persistent expression of Fos in dynorphin-rich regions of rat brainstem. Brain Res 861:191-207, 2000.)

If such changes also occur in humans, they may help to explain why certain patients may complain of vague, poorly described pain for months following endodontic treatment. If their pulpitis caused sprouting of periapical nerves,[49,50,53,195] these nerves may have taken part in transport of peripheral signaling molecules to the cell body by way of retrograde axoplasmic flow.[42,53] This could induce changes in the expression of many genes, resulting in central sensitization[6,41] that may require many months to correct.[351] Reports of nerve sprouting in human inflamed pulps have been confirmed in different studies.[310,312,319] Such reactions might contribute to increases in dentin sensitivity as well as expansion of receptive fields.[268,269,273] Sprouting of sympathetic fibers has also been reported,[147] but the timing appears to be different. The functional implications and how this relates to pain mechanisms are unknown, but it has been suggested that these reactions are involved in healing and nociception following pulpal inflammation.[115,147,148]

Processing: The Second Step in Pain Perception

The Medullary Dorsal Horn

After activation of peripheral nociceptors, nerve impulses in the form of action potentials convey information about the intensity (encoded by firing frequency), quality (encoded by type of neuron activated), and temporal features (encoded by onset, duration, and offset of depolarization) of the peripheral stimuli to the CNS. In the trigeminal pain system, these action potentials arrive at the trigeminal spinal tract nuclear complex located in the medulla.[141,214,329] Three distinct subnuclei can be found in this complex. Named for their anatomic position, they are the *subnuclei oralis, interpolaris,* and *caudalis* (see Fig. 12-24). Although the more rostral subnuclei (oralis and interpolaris) receive some nociceptive input from oral tissues,[70] most such input is received at the level of the subnucleus caudalis.[89,233,329] Because of its organizational similarity to the dorsal horn of the spinal cord (which receives nociceptive input from the somatosensory system), the subnucleus caudalis has been termed the *medullary dorsal horn*.

Components of the Medullary Dorsal Horn

The medullary dorsal horn relays information to higher centers in the brain and serves as the site of much potential processing of the signals from primary afferent sensory nerve fibers. Output from this region can be increased (hyperalgesia), decreased (analgesia), or misinterpreted (referred pain). Understanding the functional components involved in such processing not only helps explain some of these clinical phenomena but also allows evaluation of potential therapeutic modalities currently under investigation. Functional components include the central terminals of primary nociceptors (Aδ and C fiber afferents), the second-order projecting neurons, interneurons, the terminals of descending neurons, and glial[241] cells (for review, see Hargreaves[137]).

Primary afferent fibers (whose cell bodies are located in the trigeminal ganglion) transmit signals to projection neurons via the release of transmitters such as the excitatory amino acid, glutamate, and the neuropeptide, substance P. Receptors for these neurotransmitters are found on postsynaptic membranes and include the N-methyl-D-aspartate receptor (NMDA) and α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPA) classes of glutamate receptors and the neurokinin 1 (NK1) class of substance P receptors. Antagonists to these receptors have been shown to reduce hyperalgesia in animal studies.[59] In a human clinical trial using an oral surgery model, the AMPA/kainate antagonist, LY293558, was shown to be antihyperalgesic.[117] NK1 antagonists have shown promising results in animal studies, but in general, they have displayed limited analgesic efficacy in humans.[156]

The cell bodies of the second-order (projection) neurons in the trigeminal pain system are found in the medullary dorsal horn; their processes cross the midline and project rostrally to the thalamus via the trigeminothalamic tract (Fig. 12-33). From the thalamus, third-order neurons relay information to the cerebral cortex via a thalamocortical tract. Once signals have reached the cortex, the input may be perceived as pain. Evidence exists that referred pain is caused by convergence of afferent input from different areas onto the same projection neurons.

Approximately 50% of subnucleus caudalis neurons are estimated to receive convergence of sensory input from cutaneous and deep structures.[329] In one study of a cat, a single nucleus caudalis neuron received input from sensory neurons innervating the cornea, the skin overlying the maxilla, a maxillary premolar tooth, and a mandibular canine and premolar tooth on one side.[333] Subnuclei oralis and interpolaris also receive converging input from orofacial and muscle afferents.[332] This would explain the clinical observation of patients who perceive pain in a particular tooth that actually originates from either a different tooth or structure (see also Chapter 17). In such cases, anesthetizing the tooth suspected by the patient would afford no relief. However, if an anesthetic is delivered selectively to the suspected primary source of pain, the patient's discomfort should greatly diminish.[279] Likewise, if the source of a perceived toothache were located in a muscle of mastication, palpation of that muscle should aggravate the pain.[410]

In the medullary dorsal horn, local circuit interneurons have the potential to affect transmission of nociceptive input from primary afferents to projection neurons. Depending on the transmitter released, these neurons have the ability to enhance or diminish the signal. Typically, excitatory interneurons release glutamate or substance P, whereas inhibitory interneurons release the amino acid, glycine, or gamma amino butyric acid (GABA).[214,331]

The terminals of neurons that descend from brain structures such as the locus coeruleus and nucleus raphe magnus tend to inhibit nociceptive transmission at the level of the medullary dorsal horn.[14] These terminals release a variety of neuroeffective agents, including the endogenous opioid peptides (EOPs). The EOPs, similar in three-dimensional structure to many of the exogenous opiates from which their name derives, are released in response to nociceptive input and act to suppress the pain system. The EOPs likely are partly responsible for the placebo effect seen in pain control studies, because this effect can be reversed by administration of the opioid antagonist, naloxone.[139,212]

The final component of the medullary dorsal horn complex to be considered is the glial cell population. Historically considered to be solely supportive in function, they are now recognized to play an important role in the pain processing system.[302,397] Following nociceptive input from primary afferents, glia release cytokines such as TNF-α and IL-1, as well as certain PGs that may facilitate the activity of projection neurons. Glial modulating agents have been shown to be effective in experimental models of neuropathic pain,[348] and NSAIDs potentially could exert part of their analgesic mechanism by acting at this level.

Central Sensitization

Central sensitization can be defined as an increased responsiveness of central nociceptive neurons to peripheral stimulation that occurs in addition to *peripheral sensitization* of the primary afferent nociceptors. Central sensitization is thought to be a major cause of hyperalgesia and allodynia.[204] Clinical trials implicate central sensitization in patients reporting pain due to irreversible pulpitis. In one survey of nearly 1000 patients, 57% of patients with irreversible pulpitis reported mechanical allodynia (pain due to percussion).[288] This appears to be due at least in part to central sensitization, as both the ipsilateral (pulpitis) tooth and a contralateral (normal) tooth demonstrated mechanical allodynia to a force transducer.[185] Thus, central sensitization contributes to a spread of endodontic pain, and the clinical application of bite force transducers may provide a novel method for diagnosing pain mechanisms.[184,185]

Different studies shed light on the molecular mechanisms involved in central sensitization (for review, see Cousins and Power[63] and Hargreaves[137]), but the process is generally initiated by a barrage of nociceptive impulses from peripheral C fibers. The level and duration of pain prior to endodontic intervention have been cited in several studies as predictors of postoperative endodontic pain,[364,393] and this may be due to such a prolonged and intense input from C nociceptors. Any reduction of such a barrage should limit the occurrence of central sensitization and the development of pain of longer duration after tissue injury (including surgical and nonsurgical endodontic procedures). The use of long-acting local anesthetics following tonsillectomies and third molar extractions has been shown to provide pain relief far beyond the duration of the peripheral tissue anesthesia.[125,167]

A reduction in the chemical mediators of inflammation at the level of the medullary dorsal horn also should reduce sensitization of second-order neurons. Decreasing the synthesis of proinflammatory PGs, cytokines, nitric oxide, or the use of drugs that block the receptors of such agents probably will

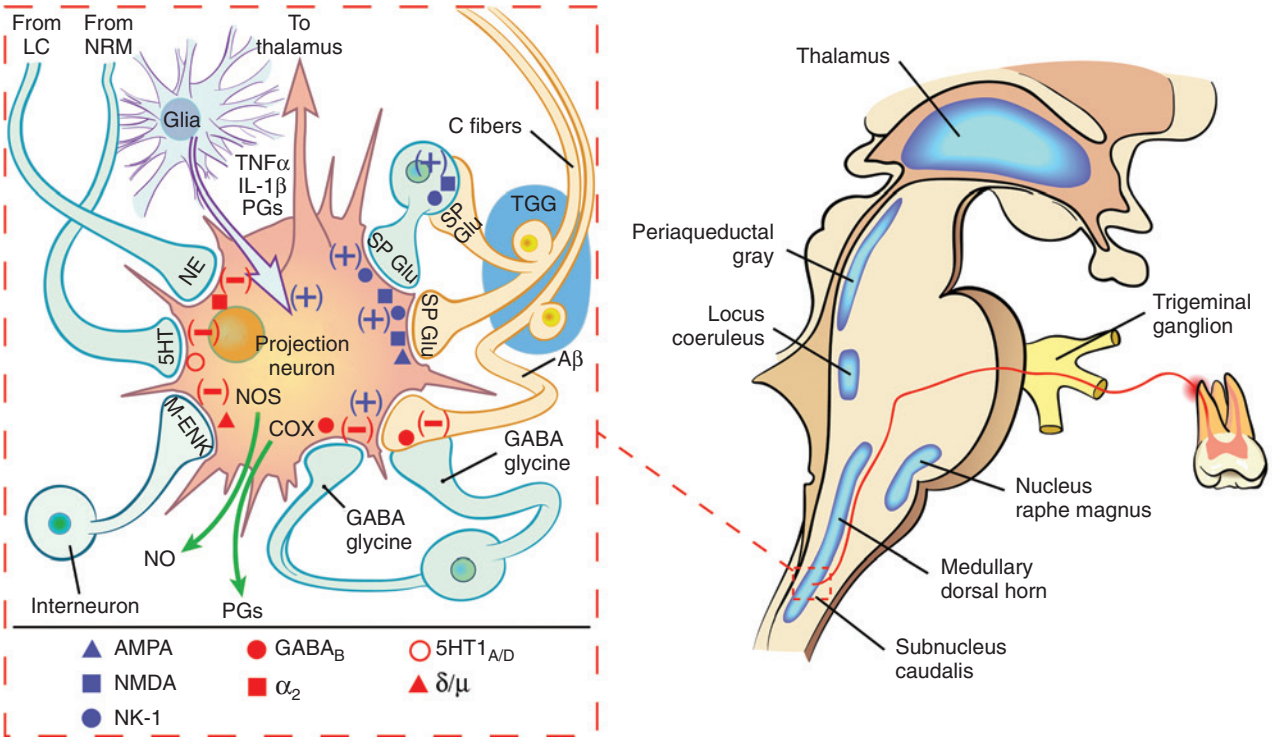

FIG. 12-33 Schematic diagram of the perception and modulation of orofacial pain. Activation of primary afferent fibers (in this example from an inflamed maxillary molar) leads to the entry of a nociceptive signal that is conveyed across a synapse in the subnucleus caudalis of the trigeminal spinal nucleus. The second-order neuron projects to the thalamus; the information is then relayed to the cortex. A great deal of processing of nociceptive input can occur at the level of the medullary dorsal horn (MDH). The *inset* depicts a typical wide dynamic range (WDR) projection neuron and its relationship with other components of the MDH. Primary afferent fibers release the excitatory amino acid, glutamate—which binds and activates either AMPA or NMDA receptors—and substance P—which activates NK-1 receptors on the WDR neuron or excitatory interneurons. Descending fibers from the locus coeruleus (LN) and nucleus raphe magnus (NRM) secrete serotonin (5HT) and norepinephrine (NE), respectively, which inhibit transmission. Release of γ amino butyric acid (GABA), the amino acid, glycine, and endogenous opioid peptides such as met-enkephalin (M-ENK) also inhibit transmission of nociceptive information. Projection neurons may have autocrine or paracrine effects by the synthesis and release of prostaglandins (PGs) and nitric oxide (NO) via the action of cyclooxygenase (COX) and nitric oxide synthase (NOS), respectively. Glial cells can modulate nociceptive processing by the release of cytokines such as tumor necrosis factor alpha (TNF-α) and interleukin 1 beta (IL-1β). The + sign indicates an excitatory action, whereas the – sign denotes an inhibitory action.

become accepted pharmacotherapy in the future. For example, application of an inflammatory agent to the tooth pulp of rat maxillary molars results in an increased receptive field of Aβ touch receptors on the face. This can be blocked by pretreatment with a glutamate NMDA receptor antagonist, indicating that such centrally acting drugs may offer highly efficacious means of treating odontogenic pain.[59] A similar investigation implicated nitric oxide synthesis at the level of the subnucleus caudalis in the development of tactile hypersensitivity following dental injury.[416] Reduction in nitric oxide synthase levels may also provide protection from central sensitization.[234,248]

Perception: Thalamus to Cortex

The final anatomic step in the trigeminal pain pathway relies on neurons that leave the thalamus and extend to the cerebral cortex (see Fig. 12-24). The patient actually perceives a stimulus as painful at the cortical level. It is interesting to note (but likely of no surprise to the experienced clinician) that a disproportionately large portion of the sensory cortex in humans is devoted to input from orofacial regions.[294]

It is becoming increasingly obvious that higher-order (i.e., cortical) perceptual processes have a profound effect on the ultimate state of pain the patient experiences (for a review, see Yaksh[411]). Memories of previous pain experiences provide a framework by which similar new experiences are judged and serve to shape the patient's response to a given stimulus. In the field of dentistry, the anxiety level of the patient at the time of treatment has been shown to affect not only the patient's response to pain experienced during treatment,[78,407] but also the tendency of the patient to recall the experience as painful or unpleasant even 18 months after treatment.[112] The clinician should do everything possible to control a patient's anxiety level prior to endodontic treatment (see also Chapter 28). One simple pretreatment method is to provide patients with positive written information regarding the control of pain during their endodontic treatment. In a placebo-controlled clinical

trial, 437 endodontic patients were given one of five informative paragraphs to read prior to treatment. One of the paragraphs contained positive information about pain during treatment. Patients completed questionnaires following treatment that evaluated their dental anxiety and dental fear. Subjects given positive information were shown to be less fearful of pain during endodontic therapy.[381] Along with a positive and caring attitude, pharmacologic intervention may help reduce anxiety. Nitrous oxide has been shown to be effective in a dental setting,[75] but it may interfere with radiography procedures during endodontic therapy. In a placebo-controlled clinical trial in patients undergoing the extraction of impacted third molars, 0.25 mg of oral triazolam (a benzodiazepine) provided comparable anxiolysis to intravenous diazepam titrated to a typical clinical endpoint.[176] Of course, the patient so medicated must be provided transportation to and from the dental office, and the potential drug-drug interactions with other centrally acting agents such as opioids, barbiturates, and alcohol must be considered. One interaction that should be considered is the capacity of grapefruit juice to prolong the half-life of triazolam.[217] It has been shown that furanocoumarins in grapefruit juice inhibit cytochrome P450 3A4,[290] which is the enzyme responsible for the metabolism of triazolam in the liver. Patients should be told not to take oral triazolam with grapefruit juice.

VASCULAR SUPPLY

Blood from the dental artery enters the tooth by way of arterioles having diameters of 100 μm or less. These vessels pass through the apical foramen or foramina with nerve bundles. Smaller vessels may enter the pulp by way of lateral or accessory canals. They are richly innervated by autonomic and sensory nerves, and the regulation of blood flow seems to be dominated by neuronal control[2,20,188,283,359] (Fig. 12-34).

The arterioles course up through the central portion of the radicular pulp and give off branches that spread laterally toward the odontoblast layer, beneath which they ramify to form a capillary plexus[196] (Fig. 12-35). As the arterioles pass into the coronal pulp, they fan out toward the dentin, diminish in size, and give rise to a capillary network in the subodontoblastic region[351] (Fig. 12-36). This network provides the odontoblasts with a rich source of metabolites.

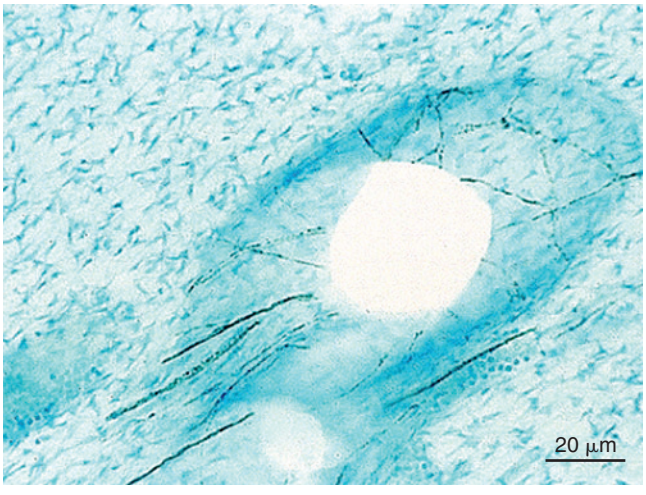

FIG. 12-34 Substance P–positive nerve fibers in the wall of pulpal blood vessels. (Courtesy Dr. K.J. Heyeraas.)

Capillary blood flow in the coronal portion of the pulp is nearly twice that in the root portion.[192] Moreover, blood flow in the region of the pulp horns is greater than in all other areas of the pulp.[249] In young teeth, capillaries commonly extend into the odontoblast layer, thus ensuring an adequate supply of nutrients for the metabolically active odontoblasts (Fig. 12-37). In the subodontoblastic capillaries, fenestrations are observed in the vessel wall.[306] These fenestrations are thought to promote rapid transport of fluid and metabolites from the capillaries to the adjacent odontoblasts. The average capillary density is about 1400/mm^2, which is greater than in most other tissues of the body.[386]

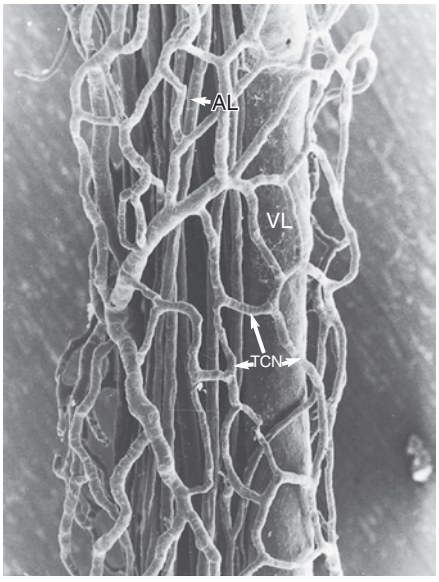

FIG. 12-35 High-power scanning electron micrograph of vascular network in the radicular pulp of a dog molar showing the configuration of the subodontoblastic terminal capillary network (TCN). Venules (VL) and arterioles (AL) are indicated. (Courtesy Dr. Y. Kishi, Kanagawa Dental College, Kanagawa, Japan.)

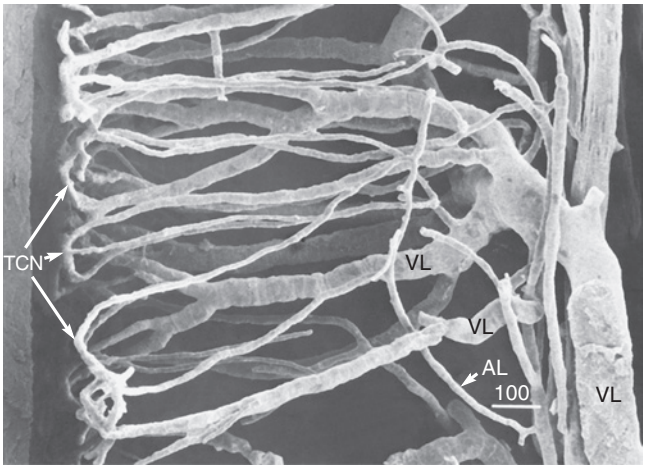

FIG. 12-36 Subodontoblastic terminal capillary network (TCN), arterioles (AL), and venules (VL) of young canine pulp. Dentin would be to the far left and the central pulp to the right. Scale bar: 100 μm. (From Takahashi K, Kishi Y, Kim S: A scanning electron microscopic study of the blood vessels of dog pulp using corrosion resin casts, *J Endod* 8:131, 1982.)

Blood passes from the capillary plexus, first into postcapillary venules (see Figs. 12-36 and 12-38) and then into larger venules.[196] Venules in the pulp have unusually thin walls, and the muscular layer is discontinuous,[68] which may facilitate the movement of fluid in or out of the vessel. The collecting venules become progressively larger as they course to the central region of the pulp. The largest venules have a diameter that may reach a maximum of 200 μm, considerably larger than the arterioles of the pulp.

The resting pulpal blood flow is relatively high, averaging 0.15 to 0.60 ml/min/g tissue,[245,360] and blood volume represents about 3% of pulpal wet weight,[30] approximately the same as in mammary tumor tissue.[403] As would be anticipated, pulpal blood flow is greater in the peripheral layer of the pulp (i.e., the subodontoblastic capillary plexus)[196] where the oxygen consumption has been shown to be higher than in the central pulp.[26]

Changes in pulpal blood flow can be measured through dentin using laser Doppler flowmeters. Sensitivity to movement requires that they are stabilized in an occlusal stent or a modified rubber dam clamp.[93,321] Because up to 80% of the Doppler signal originates from periodontal tissue, it is helpful to cover periodontal tissues with a black rubber dam.[144] Laser Doppler flowmetry can be used to detect revascularization of traumatized teeth.[79,88] Although measurement of pulpal blood flow would be an ideal tool for determining pulp vitality, the use of laser Doppler and other techniques is limited due to sensitivity, specificity, reproducibility, and costs.

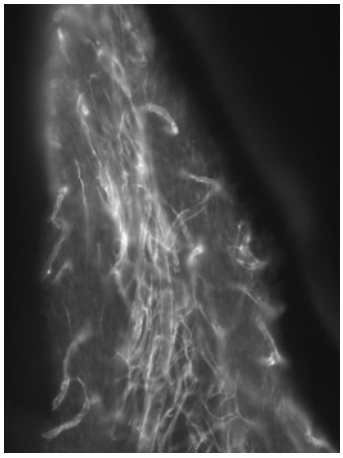

FIG. 12-37 Blood vessels in the pulp horn fan out into the odontoblast layer. (Courtesy Dr. S.R. Haug.)

Regulation of Pulpal Blood Flow

Under normal physiologic conditions, pulpal vascular tone is controlled by neuronal, paracrine, and endocrine mechanisms that keep the blood vessels in a state of partial constriction. The pulpal blood flow is also influenced by vascular tone in neighboring tissues. Vasodilatation in these tissues has been shown to cause a drop in pulpal blood flow due to reduction in local arterial pressure of the teeth and thereby reduced pulpal perfusion pressure.[361] The "stealing" of dental perfusion pressure makes the dental pulp vulnerable in clinical situations with inflammatory processes in the adjacent tissues, as in gingivitis and periodontitis.

Neuronal regulation of blood flow is extensive in the pulp. There is little or no vasoconstrictor tone of sympathetic origin in the dental pulp during resting conditions,[165,359] but a neuronal vasodilator tone caused by release of sensory neuropeptides has been demonstrated (Fig. 12-39).[21,20]

There are α-adrenergic receptors in the pulp,[162] and stimulation of the cervical sympathetic trunk causes vasoconstriction and fall in pulpal blood flow that can be partially reversed by α-receptor blockade.[188,359] NPY, colocalized with norepinephrine in pulpal sympathetic nerve fibers, contributes also to vasoconstriction in the pulp.[81,194]

Increase in pulpal blood flow is observed after electrical tooth stimulation and is caused by the release of sensory neuropeptides followed by vasodilatation.[20,153,181] CGRP released from sensory nerve fibers is mainly responsible for the observed vasodilatation.[21,20]

Glutamate, present in CGRP negative sensory afferent nerve fibers in the pulp, also has a vasodilatory effect when applied in the pulp during experimental conditions.[419]

There is evidence for sympathetic modulation of sensory neuropeptide release in the dental pulp[138]; presynaptic adrenoceptors are found on the sensory nerve terminals and attenuate the release of vasodilators from the sensory nerves.[34,180]

Muscarinic receptors have been identified in the pulp,[32] and the parasympathetic neurotransmitter acetylcholine (ACh)

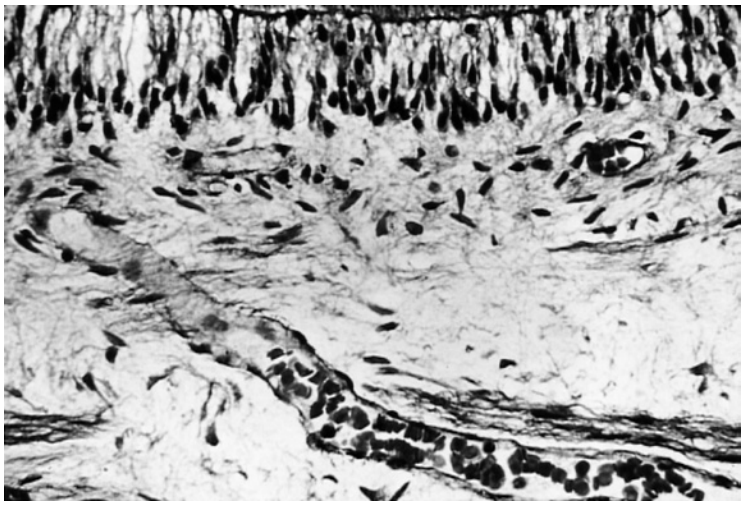

FIG. 12-38 Postcapillary venule draining blood from subodontoblastic capillary plexus.

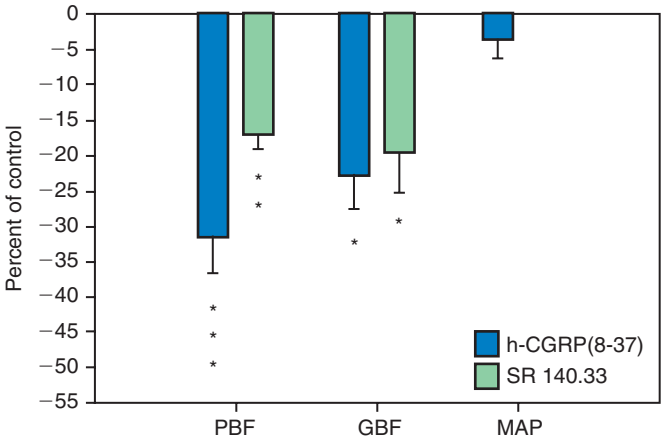

FIG. 12-39 Effect of antagonist infusion of h-CGRP(8-37) (calcitonin gene–related peptide inhibitor) and SR 140.33 (substance P inhibitor) on basal pulpal blood flow (PBF) and gingival blood flow (GBF). (From Berggreen E, Heyeraas KJ: Effect of the sensory neuropeptide antagonists h-CGRP[8-37] and SR 140.33 on pulpal and gingival blood flow in ferrets, *Arch Oral Biol* 45:537, 2000.)

causes vasodilatation and increases blood flow in the tissue.[417] The vasodilation evoked by acetylcholine has been demonstrated to be partly dependent on nitric oxide (NO) production.

VIP, which coexists with ACh in postganglionic neurons, is found in the dental pulp[105,377] and has been demonstrated to cause vasodilatation and increase in pulpal blood flow in cats.[283]

On the other hand, Sasano and coworkers[323] failed to demonstrate parasympathetic nerve-evoked vasodilatation in the cat dental pulp, leaving pulpal vascular responses to parasympathetic neurotransmitters with some uncertainty.

Local Control of Blood Flow

The microvascular bed in the dental pulp has the ability to regulate hemodynamics in response to local tissue demands. Endothelin-1 is located in the endothelium of pulpal vasculature,[57] and close intraarterial infusions of endothelin-1 reduce pulpal blood flow.[22,116,417] However, endothelin-1 does not seem to influence blood vessel vascular tone under basal, resting conditions.[22]

The endothelium in pulpal blood vessels modulates vascular tone by release of vasodilators such as prostacyclin and NO. A basal synthesis of NO provides a vasodilator tone on pulpal vessels.[20,223] The shear forces that blood flow exert on endothelial cells seem to regulate the release of NO.[73]

Adenosine is released from ischemic and hypoxic tissue and is probably important in the metabolic regulation of blood flow in periods of low pulpal oxygen tension. When applied from the extraluminal side of the vessel wall, adenosine mediates vasodilatation in pulpal vessels.[417]

Humoral Control of Blood Flow

Evidence for humoral control of pulpal blood flow exists and takes place when vasoactive substances transported by the bloodstream reach the receptors in the pulp tissue. Angiotensin II is produced by activation of the renin/angiotensin system and exerts a vasoconstrictive basal tone on pulpal blood vessels.[22] The angiotensin II receptors, AT1 and AT2, have been identified in the rat pulp.[341]

Similarly to the effect of norepinephrine released from sympathetic nerve fibers in the pulp, epinephrine released from the adrenal medulla will cause vasoconstriction due to activation of α-adrenergic receptors in the pulp. Another catecholamine, dihydroxyphenylalanine (DOPA), also induces vasoconstriction in pulpal arterioles when applied intraarterially.[417]

Fluid Drainage

Interstitial fluid, which accumulates in the tissue during normal conditions through net filtration out of the blood vessels or during inflammation where the net filtration is increased, must be removed in order to maintain normal fluid balance. In most tissues in the body, the lymphatic vessels drain excess fluid from the peripheral tissue and return it into the blood vessel system. In addition, the lymphatic system is important because it transports captured antigen and presents it in the lymph nodes. The existence of lymphatics in the pulp has been a matter of debate because it is difficult to distinguish between blood and lymphatic vessels by ordinary microscopic techniques without specific lymphatic markers.

Several specific lymphatic markers have now been applied, and contradictory conclusions have been drawn. One of the markers tested is vascular endothelial growth factor receptor (VEGFR-3) known to be expressed by lymphatic endothelial cells in adult tissue.[114,242] The receptor expression was reported in human and mouse pulp tissue, but to identify lymphatic vessels, use of more than one lymphatic marker is recommended. However, other studies have failed to demonstrate lymphatic markers in the pulp [114,242] and one study showed lymphatic vessel endothelium receptor-1 (LYVE-1) staining in immune cells in human pulp, but not in vascular structures (Fig. 12-40). Taken together it seems that the pulp is not supplied with draining lymphatic vessels.

Transcapillary Fluid Exchange

In all tissues in the body, the fluid transport between the blood vessels and the interstitial space is regulated by differences in colloid osmotic and hydrostatic pressures in the plasma and the interstitium, and by properties in the capillary membrane (Fig. 12-41). From the interstitium excess fluid is transported back to the blood circulation through the lymphatic system. The pulp seems to be an exception along with, for example, the brain and bone marrow, as lymphatic vessels are not detected in the tissue (see the previous section). During normal conditions, a steady state is achieved as the fluid filtered into the interstitial space equals the amount of fluid transported out of the same compartment. Using radioisotopes, the interstitial fluid volume in the pulp was measured and averaged 0.6 ± 0.03 ml/g wet weight,[30] demonstrating that as much as 60% of the extracellular fluid in dental pulp is located outside the vascular system. Measurements of interstitial fluid pressure in the pulp with the micropuncture method have given values that range from 6 to 10 mm Hg,[20,150] but higher values measured with different methods have also been reported.[39,380,385]

Colloid osmotic pressure (COP) measurements in interstitial fluid isolated from rat incisors have shown a relatively high pulpal COP, reaching 83% of plasma COP.[30] The high value may imply that the normal permeability of pulpal vessels to plasma proteins is relatively high or the drainage of plasma proteins is ineffective.

Because lymphatic vessels are lacking inside the pulp, excess interstitial fluid and proteins must be transported

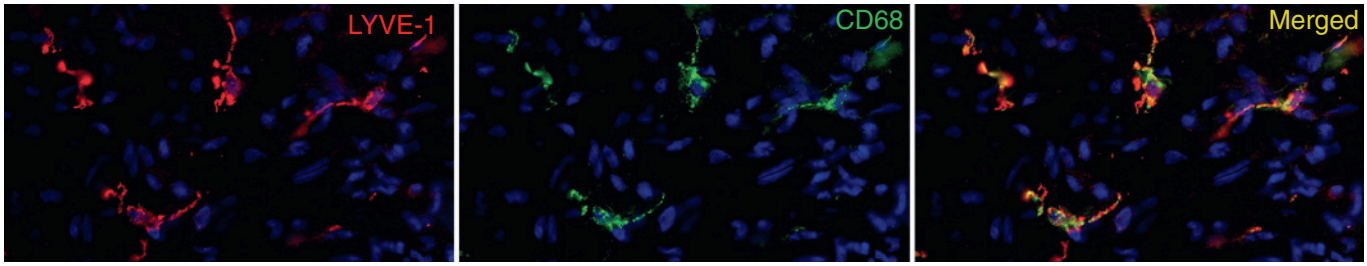

FIG. 12-40 Immune cells in normal human pulp are immunopositive to LYVE-1, known as a lymphatic vessel marker. The $CD68^+/LYVE\text{-}1^+$ cells derive from the monocytic lineage of cells. Immunostaining demonstrated the lack of $LYVE\text{-}1^+$ lymphatic vessels in the pulp. (Courtesy Dr. A Virtej.)

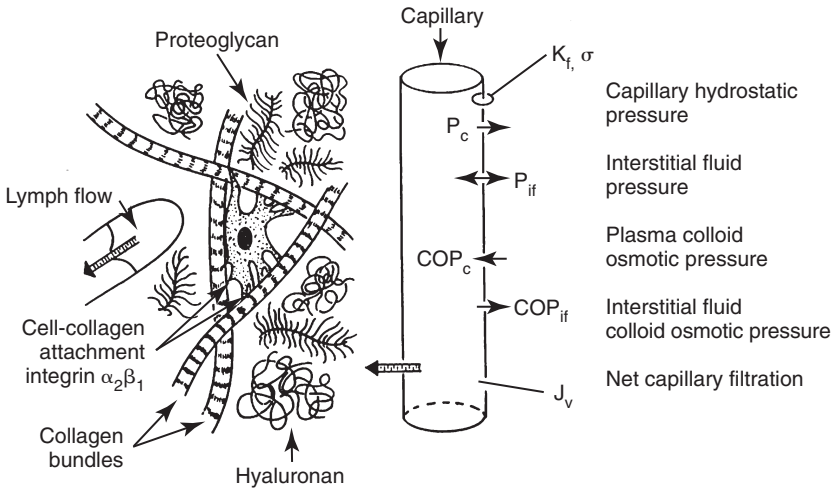

FIG. 12-41 Interstitial structure and pressures that govern transcapillary fluid transport. K_f, Capillary filtration coefficient; σ, capillary reflection coefficient for plasma proteins. (From Wiig H, Rubin K, Reed RK: New and active role of the interstitium in control of interstitial fluid pressure: potential therapeutic consequences, *Acta Anaesthesiol Scand* 47:111, 2003.)

out of the pulp by other transport routes in order to achieve a steady-state situation. Two possibilities exists: (1) transport of fluid in the interstitial compartment toward the apical part of the pulp and furthermore out of the apex, and (2) a combination of fluid reabsorption into pulpal blood vessels in addition to transport of protein-rich fluid toward the apex.

Circulation in the Inflamed Pulp

Inflammation in the pulp takes place in a low-compliance environment composed of rigid dentinal walls. *Compliance* is defined as the relationship between volume (V) and interstitial pressure (P) changes: $C = \Delta V / \Delta P$. Consequently, in the low-compliant pulp, an increase in blood or interstitial volume will lead to a relatively large increase in the hydrostatic pressure in the pulp. The acute vascular reactions to an inflammatory stimulus are vasodilatation and increased vascular permeability, both of which will increase pulp interstitial fluid pressure[151,154,359,379] and may tend to compress blood vessels and counteract a beneficial blood flow increase (Fig. 12-42).

Classical studies have demonstrated that an increase in intrapulpal tissue pressure promoted absorption of tissue fluid back into the circulation, thereby reducing the pressure.[151,154] This observation can explain why pulpal tissue pressure in inflamed pulps may persist in local regions for long observation periods,[362] contradicting the old concept of a wide,

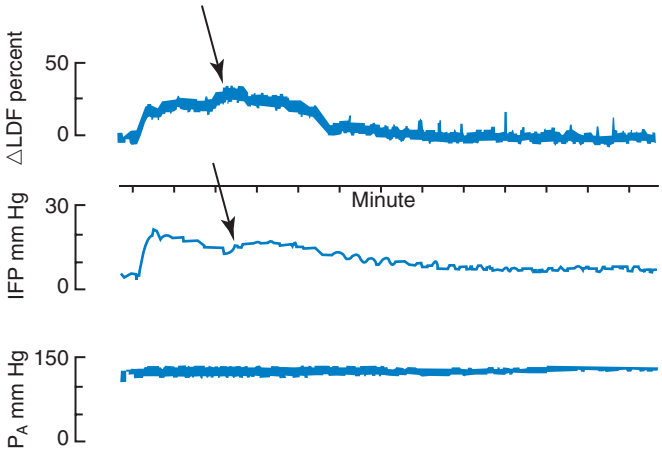

FIG. 12-42 Original simultaneous recordings of percent change in pulpal blood flow (ΔLDF%), interstitial fluid pressure (IFP), and systemic blood pressure (P_A mm Hg) in a cat during electrical tooth stimulation. Note that when IFP is first decreasing after an initial rise, the pulpal blood flow reaches its maximal level *(arrows),* demonstrating compression of vessels in the first phase. (Courtesy Dr. K.J. Heyeraas.)

generalized collapse of pulpal venules and cessation of blood flow (pulpal strangulation theory).

The delivery of dental restorative procedures may lead to substantial increases or decreases in pulpal blood flow, depending on the precise procedure and time point sampled.[189] Vasoactive mediators are locally released upon an inflammatory insult, and in the pulp, prostaglandin E2, bradykinin, SP, and histamine have all been demonstrated to increase pulpal blood flow after application.[191,284] In contrast, serotonin (5-HT) is released primarily from the platelets, and given intraarterially, it has been shown to reduce pulpal blood flow.[193,417]

Acute inflammation in the dental pulp induces an immediate rise in blood flow and can reach a magnitude of up to nearly 200% of control flow followed by increased vascular permeability.[152,154]

A common outcome of pulpal inflammation is development of tissue necrosis. One study found circulatory dysfunction developed in the pulp after exposure to lipopolysaccharide (LPS) from gram-negative bacteria.[30]

In addition, the inflammatory cytokines IL-1 and TNF-α are elevated in the inflamed pulp. When the endothelium is exposed to endotoxin, it expresses cytokines, chemokines, and thromboxane A2. The latter has been demonstrated to be produced in the pulp exposed to LPS[281] and induces vasoconstriction. The set of changes in endothelial function have been called *endothelial perturbation* and were first described in endothelial cells exposed to endotoxin or to cytokines such as IL-1, TNF-α, and IL-6.[24,271] The activated endothelium also participates in procoagulant reactions that promote fibrin clot formation.[345] A reduced pulpal perfusion due to endothelial perturbation might be the consequence of bacterial infection impairing pulpal defense mechanisms and promoting necrosis. Downregulation of vascular endothelial growth factor (VEGF) expression in stromal cells and reduced microvessel density have been observed in human dental pulps with irreversible pulpitis.[10] VEGF is an essential proangiogenic factor, and the reduced microvessel density might also lead to reduced pulpal perfusion and contribute to development of pulpal necrosis.

Vascular Permeability

Increased vascular permeability takes place as a result of acute inflammation, and vascular leakage has been demonstrated in the pulp after release of inflammatory mediators such as prostaglandin, histamine, bradykinin, and the sensory neuropeptide, SP.[182,191,236]

LPS and lipoteichoic acid () from gram-negative and gram-positive bacteria, respectively, cause upregulation of VEGF in activated pulpal cells.[33,353] VEGF increases vascular permeability,[91,328] and it is likely that it also causes leakage in pulpal vessels. It is a potent agent, because its ability to enhance microvascular permeability is estimated to be 50,000 times higher than that of histamine.[335] Cytokines such as IL-1 and TNF-α are released into the pulp interstitial fluid during inflammation[30] and upregulate VEGF mRNA gene expression in pulpal fibroblasts.[60]

The resulting increased vascular permeability allows increased transport of proteins through the capillary vessel wall and results in increased COP in the tissue. In acute pulpitis induced by LPS, it has been shown that COP in the pulp can reach the level of plasma COP, meaning that a protein transport barrier between plasma and interstitium can be eliminated.[30]

Clinical Aspects

The influence of posture on pulpal blood flow has been observed in humans.[60] Significantly greater pulpal blood flow was measured when subjects changed from an upright to a supine position. The supine position increases venous return from all tissues below the level of the heart, thereby increasing cardiac output and producing a transient increase in systemic blood pressure. The increase in blood pressure stimulates baroreceptors that reflexively decrease sympathetic vasoconstriction to all vascular beds, thereby increasing peripheral blood flow.

Patients with pulpitis often report an inability to sleep at night because they are disturbed by throbbing tooth pain. In addition to the lack of distractions normally present during the day, the following mechanism may be operative in patients with inflamed pulps. When these patients lie down at the end of the day, their pulpal blood flow probably increases due to the cardiovascular postural responses described earlier. This may increase their already elevated pulpal tissue pressure,[151,154,344,362,379] which is then sufficient to activate sensitized pulpal nociceptors and initiate spontaneous pulpal pain. Thus, the "throbbing" sensation of toothache was previously considered as an effect of the pulsation in the pulp that follows heart contractions (systole), but a study investigating the rhythm of toothache and the patient pulse showed lack of synchrony between the two parameters.[251] The authors raised an alternative hypothesis: that the throbbing quality is not a primary sensation but rather an emergent property, or perception, whose "pacemaker" lies within the CNS causing intermittent increases in pulpal tissue pressure.

PULPAL REPAIR

The inherent healing potential of the dental pulp is well recognized. As in all other connective tissues, repair of tissue injury commences with débridement by macrophages, followed by proliferation of fibroblasts, capillary buds, and the formation of collagen. Local circulation is of critical importance in wound healing and repair. An adequate supply of blood is essential to transport immune cells into the area of pulpal injury and to dilute and remove deleterious agents from the area. It is also important to provide fibroblasts with nutrients from which to synthesize collagen. Unlike most tissues, the pulp has essentially no collateral circulation; for this reason, it is theoretically more vulnerable than most other tissues. In the case of severe injury, healing would be impaired in teeth with a limited blood supply. It seems reasonable to assume that the highly cellular pulp of a young tooth, with a wide-open apical foramen and rich blood supply, has a much better healing potential than an older tooth with a narrow foramen and a restricted blood supply.

Dentin can be classified as primary, secondary, or tertiary, depending on when it was formed. *Primary dentin* is the regular tubular dentin formed before eruption, including mantle dentin. *Secondary dentin* is the regular circumferential dentin formed after tooth eruption, whose tubules remain continuous with that of primary dentin. *Tertiary dentin* is the irregular dentin that is formed in response to abnormal stimuli, such as excess tooth wear, cavity preparation, restorative materials, and caries.[64,65] In the past, tertiary dentin has been called *irregular dentin, irritation dentin, reparative dentin,* and *replacement dentin*. Much of the confusion was

caused by a lack of understanding of how tertiary dentin is formed.

If the original odontoblasts that made *secondary dentin* are responsible for focal tertiary dentin formation, that particular type of tertiary dentin is termed *reactionary dentin*.[339] Generally, the rate of formation of dentin is increased, but the tubules remain continuous with the secondary dentin.[342] However, if the provoking stimulus caused the destruction of the original odontoblasts, the new, less tubular, more irregular dentin formed by newly differentiated odontoblast-like cells is called *reparative dentin*. In this dentin the tubules are usually not continuous with those of secondary dentin. Initially, the newly formed cells tend to be cuboidal in shape, without the odontoblast process that is necessary to form dentinal tubules. They seem to form in response to the release of a host of growth factors that were bound to collagen during the formation of secondary dentin.[92,309,339] The loss of the continuous layer of odontoblasts exposes unmineralized predentin that is thought to contain both soluble and insoluble forms of TGF-β, insulin-like growth factor (IGF)-1 and IGF-2, bone morphogenetic proteins (BMPs), VEGF, and other growth factors that attract and cause proliferation and differentiation of mesenchymal stem cells to form reparative dentin and new blood vessels. During caries progression, bacterial acids may solubilize these growth factors from mineralized dentin, liberating them to diffuse to the pulp, where they could stimulate reactionary dentin formation. This is also thought to be the mechanism of action of calcium hydroxide during apexification treatment. Despite its high pH, calcium hydroxide has a slight demineralizing effect on dentin and has been shown to cause the release of TGF-β.[338] TGF-β and other growth factors stimulate and accelerate reparative dentinogenesis. Other researchers have attempted to apply growth factors to dentin to allow it to diffuse through the tubules to the pulp.[316,317,340] Although this has been successful, the remaining dentin thickness must be so thin that this approach may not be practical from a therapeutic perspective. Others have inserted deoxyribonucleic acid (DNA)-sequenced BMP-7 into retroviruses to transfect ferret pulpal fibroblasts to stimulate increased BMP-7 production. Although this was successful in normal pulps,[316] it was unsuccessful in inflamed pulps.[315] Specific amelogenin gene splice products, A+4 and A-4, adsorbed onto agarose beads and applied to pulp exposures, induced complete closure and mineralization of the root canal in rat molars.[122] The regulation of peritubular dentin formation is not well understood. Some have claimed that this is a passive process resulting in occlusion of the tubules over time, but it has also been claimed that this is a mechanism under odontoblast control. If odontoblasts could be stimulated to form excessive peritubular dentin by the application of an appropriate biologic signaling molecule to the floor of cavity preparations, then the tubules of the remaining dentin could be occluded, rendering this dentin impermeable and protecting the pulp from the inward diffusion of noxious substances that might leak around restorations.[291] These are examples of how molecular biology may be used in future restorative dentistry.

The term most commonly applied to irregularly formed dentin is *reparative dentin,* presumably because it frequently forms in response to injury and appears to be a component of the reparative process. It must be recognized, however, that this type of dentin has also been observed in the pulps of normal, unerupted teeth without any obvious injury.[276]

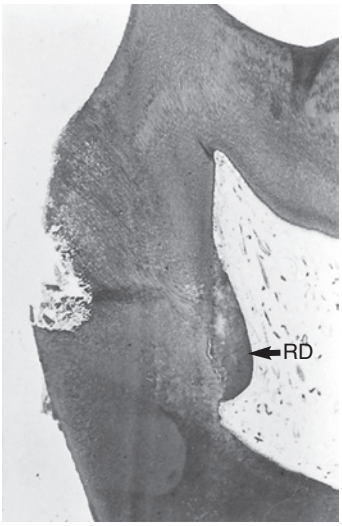

FIG. 12-43 Reparative dentin (RD) deposited in response to a carious lesion in the dentin. (From Trowbridge HO: Pathogenesis of pulpitis resulting from dental caries, *J Endod* 7:52, 1981.)

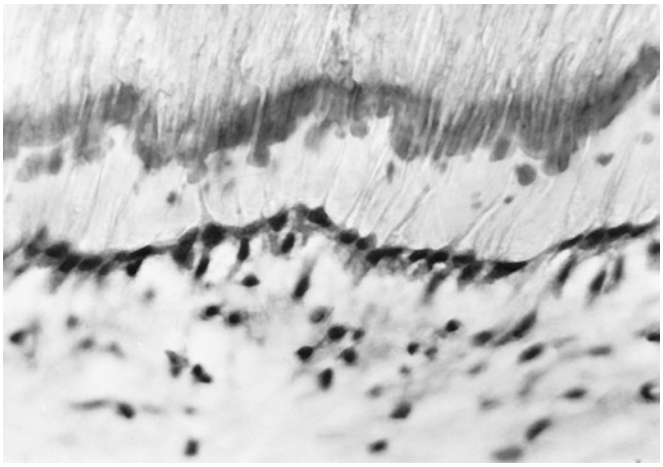

FIG. 12-44 Layer of cells forming reparative dentin. Note the decreased tubularity of reparative dentin compared with the developmental dentin above it.

It will be recalled that secondary dentin is deposited circumpulpally at a slow rate throughout the life of the vital tooth.[342] In contrast, when a carious lesion has invaded dentin, the pulp usually responds by depositing a layer of tertiary dentin over the dentinal tubules of the primary or secondary dentin that communicate with the carious lesion (Fig. 12-43). Similarly, when occlusal wear removes the overlying enamel and exposes the dentin to the oral environment, tertiary dentin is deposited on the pulpal surface of the exposed dentin. Thus, the formation of tertiary dentin allows the pulp to retreat behind a barrier of mineralized tissue.[370]

Compared with primary or secondary dentin, tertiary dentin tends to be less tubular, and the tubules tend to be more irregular with larger lumina. In some cases, particularly when the original odontoblasts are destroyed, no tubules are formed. The cells that form reparative dentin are often cuboidal and not as columnar as the primary odontoblasts of the coronal pulp (Fig. 12-44). The quality of tertiary dentin (i.e., the extent to which it resembles primary or secondary dentin) is quite

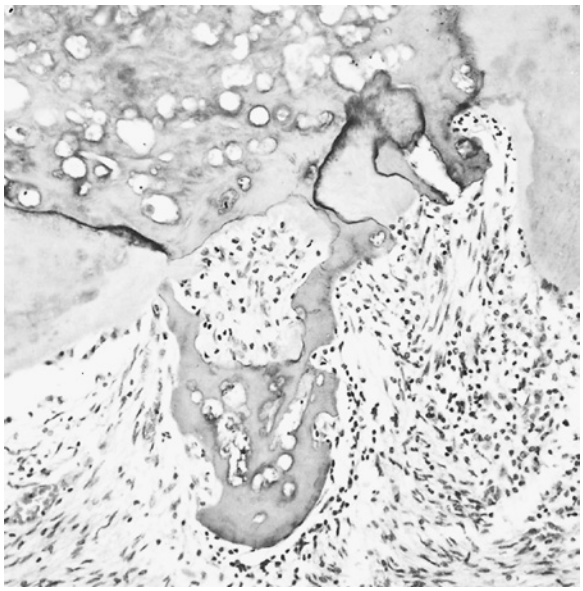

FIG. 12-45 Swiss-cheese type of reparative dentin. Note the numerous areas of soft-tissue inclusion and infiltration of inflammatory cells in the pulp.

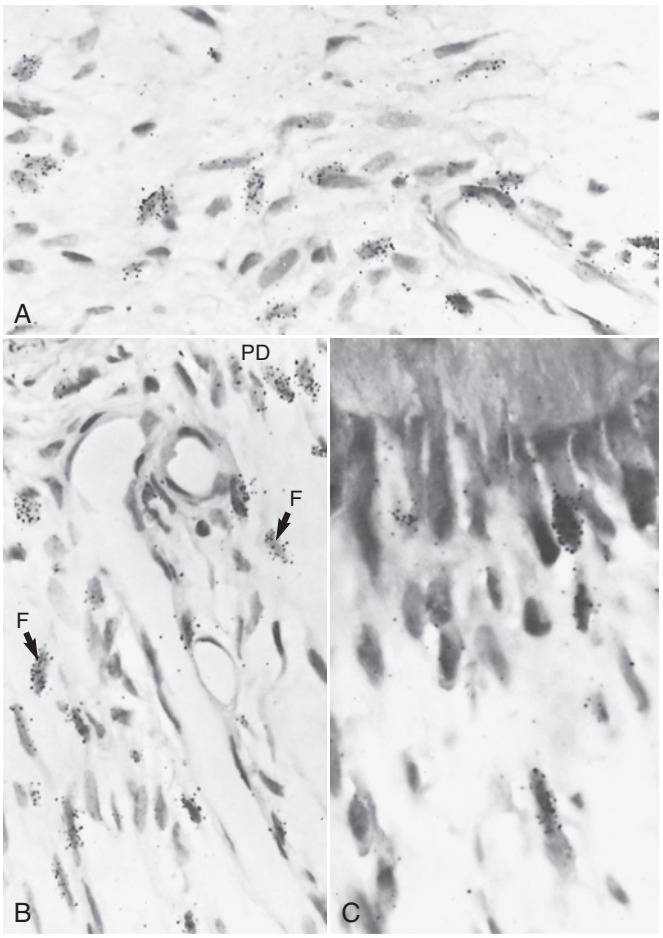

FIG. 12-46 Autoradiographs from dog molars illustrating uptake of ^{3}H-thymidine by pulp cells preparing to undergo cell division after pulpotomy and pulp capping with calcium hydroxide. **A,** Two days after pulp capping. Fibroblasts, endothelial cells, and pericytes beneath the exposure site are labeled. **B,** By the fourth day, fibroblasts (F) and preodontoblasts adjacent to the predentin (PD) are labeled, which suggests that differentiation of preodontoblasts occurred within 2 days. **C,** Six days after pulp capping, new odontoblasts are labeled, and tubular dentin is being formed. (Titrated thymidine was injected 2 days after the pulp capping procedures in **B** and **C**.) (From Yamamura T, Shimono M, Koike H, et al: Differentiation and induction of undifferentiated mesenchymal cells in tooth and periodontal tissue during wound healing and regeneration, *Bull Tokyo Dent Coll* 21:181, 1980.)

variable. If irritation to the pulp is relatively mild, as in the case of a superficial carious lesion, then the tertiary dentin formed may resemble primary dentin in terms of tubularity and degree of mineralization. On the other hand, dentin deposited in response to a deep carious lesion may be relatively atubular and poorly mineralized, with many areas of interglobular dentin. The degree of irregularity of this dentin is probably determined by numerous factors, such as the amount of inflammation present, the extent of cellular injury, and the state of differentiation of the replacement odontoblasts.

The poorest quality of reparative dentin is usually observed in association with marked pulpal inflammation.[64,370] In fact, the dentin may be so poorly organized that areas of soft tissue are entrapped within the dentinal matrix. In histologic sections, these areas of soft-tissue entrapment impart a Swiss-cheese appearance to the dentin (Fig. 12-45). As the entrapped soft tissue degenerates, products of tissue degeneration are released that further contribute to the inflammatory stimuli assailing the pulp.[370]

It has been reported that trauma caused by cavity preparation that is too mild to result in the loss of primary odontoblasts does not lead to reparative dentin formation, even if the cavity preparation is relatively deep.[73] This has been confirmed both in rat teeth[258] and human teeth.[256] However, chronic pulpal inflammation associated with deep caries produces reparative dentin. This reparative dentin is formed by new odontoblast-like cells. For many years, it has been recognized that destruction of primary odontoblasts is soon followed by increased mitotic activity within fibroblasts of the subjacent cell-rich zone. It has been shown that the progeny of these dividing cells differentiate into functioning odontoblasts.[98] Investigators[414] have studied dentin bridge formation in healthy teeth of dogs and found that pulpal fibroblasts appeared to undergo dedifferentiation and revert to undifferentiated mesenchymal stem cells (Fig. 12-46). The similarity of primary odontoblasts to replacement odontoblasts was established by D'Souza and colleagues.[77] They were able to show that cells forming reparative dentin synthesize type I (but not type III) collagen, and they are immunopositive for dentin sialoprotein.

Destruction of primary odontoblasts can occur from cutting cavity preparations dry,[80,202] from bacterial products such as endotoxins shed from deep carious lesions,[18,395] or from mechanical exposure of pulps.[257] Such pulpal wounds do not heal if the tissue is inflamed.[64] Local fibroblast-like cells divide, and the new cells then redifferentiate in a new direction to become odontoblasts. Recalling the migratory potential of ectomesenchymal cells from which the pulpal fibroblasts are derived, it is not difficult to envision the differentiating odontoblasts moving from the subodontoblastic zone to the area of injury to constitute a new odontoblast layer. Activation of antigen-presenting dendritic cells by mild inflammatory processes may also promote osteoblast/odontoblast-like differentiation and expression of molecules implicated in mineralization.

Recognition of bacteria by specific odontoblast and fibroblast membrane receptors triggers an inflammatory and immune response within the pulp tissue that would also modulate the repair process.[121]

Although many animal studies have shown dentin bridge formation in healthy pulps following pulp capping with adhesive resins,[64] such procedures fail in normal human teeth.[61]

When small mechanical pulp exposures are inadvertently made in healthy teeth, the recommendation has been to place a small, calcium hydroxide–containing dressing on the wound. After setting, the surrounding dentin can be bonded using a no-rinse, self-etching primer adhesive.[175] Calcium silicate cements like mineral trioxide aggregate (MTA) have also been recognized to promote hard-tissue formation, and available information indicates that the dentin bridge formed under these cements is more dense and has fewer defects compared with calcium hydroxide–containing dressings.[3,5,261,368]

The formation of atubular "fibrodentin" is another potential product of newly differentiated odontoblasts, provided that a capillary plexus develops beneath the fibrodentin.[15] This is consistent with the observation made by other researchers[64,98] that the newly formed dentin bridge is composed first of a thin layer of atubular dentin on which a relatively thick layer of tubular dentin is deposited. The fibrodentin was lined by cells resembling mesenchymal cells, whereas the tubular dentin was associated with cells closely resembling odontoblasts.

Other researchers[342] studied reparative dentin formed in response to relatively traumatic experimental class V cavity preparations in human teeth. They found that seldom was reparative dentin formed until about the 30th postoperative day. The rate of dentin formation was 3.5 µm/day for the first 3 weeks after the onset of dentinogenesis, after which it decreased markedly. By postoperative day 132, dentin formation had nearly ceased. Assuming that most of the odontoblasts were destroyed during traumatic cavity preparation, as was likely in this experiment, the 30-day delay between cavity preparation and the onset of reparative dentin formation is thought to reflect the time required for the proliferation, migration, and differentiation of new replacement odontoblasts.

Does reparative dentin protect the pulp, or is it simply a form of scar tissue? To serve a protective function, it would have to provide a relatively impermeable barrier that would exclude irritants from the pulp and compensate for the loss of developmental dentin. The junction between developmental and reparative dentin has been studied using a dye diffusion technique, which demonstrated the presence of an atubular zone situated between secondary dentin and reparative dentin (Fig. 12-47).[94] In addition to a dramatic reduction in the number of tubules, the walls of the tubules along the junction were often thickened and occluded with material similar to peritubular matrix.[326] Taken together, these observations would indicate that the junctional zone between developmental and reparative dentin is an atubular zone of low permeability. Moreover, the accumulation of pulpal dendritic cells was reduced after reparative dentin formation, which may indicate the reduction of incoming bacterial antigens.[319]

One group[356] studied the effect of gold foil placement on human pulp and found that this was better tolerated in teeth in which reparative dentin had previously been deposited beneath the cavity than in teeth that lacked this deposit. It would thus appear that reparative dentin can protect the pulp,[18] but it must be emphasized that this is not always the case. It is well known that reparative dentin can be deposited in a pulp that is irreversibly injured and that its presence does not necessarily signify a favorable prognosis (see Fig. 12-45). The quality of the dentin formed, and hence its ability to protect the pulp, to a large extent reflects the environment of the cells producing the matrix. The presence of a single tunnel defect[64] through reparative dentin would circumvent the protective effect of atubular reparative dentin. Therefore, any clinical attempt at pulp therapy must include sealing dentin with bonding agent.

FIG. 12-47 Diffusion of dye from the pulp into reparative dentin. Note atubular zone between reparative dentin (RD) and primary dentin on the left. (From Fish EW: *Experimental investigation of the enamel, dentin, and dental pulp,* London, 1932, John Bale Sons & Danielson.)

Periodontally diseased teeth have smaller root canal diameters than teeth that are periodontally healthy.[203] The root canals of such teeth are narrowed by the deposition of large quantities of reactionary dentin along the dentinal walls.[327] The decrease in root canal diameter with increasing age, in the absence of periodontal disease, is more likely to be the result of secondary dentin formation.

One study showed that in a rat model, frequent scaling and root planing resulted in reparative dentin formation along the pulpal wall subjacent to the instrumented root surface.[146] However, given that normal rat root dentin is only 100 µm thick, these procedures are probably more traumatic to the pulp in the rat model than in humans, where normal root dentin is more than 2000 µm thick.

Not uncommonly, the cellular elements of the pulp are largely replaced by fibrous connective tissue over a span of 5 decades. It appears that in some cases, the pulp responds to noxious stimuli by accumulating large fiber bundles of collagen, rather than by elaborating reparative dentin (Fig. 12-48). However, fibrosis and reparative dentin formation often go hand in hand, indicating that both are expressions of a reparative potential. In periodontally diseased teeth, the pulp tissue is found to be the site of an enhanced process of collagenous fibrosis associated with an inflammatory infiltrate.[56]

With the expanding knowledge of tooth regeneration and biologic mechanisms of functional dental tissue repair, current treatment strategies are beginning to give way to evolving fields such as tissue engineering and biomimetics. Pulpal stem cells in scaffolds have been shown to produce pulplike tissues with

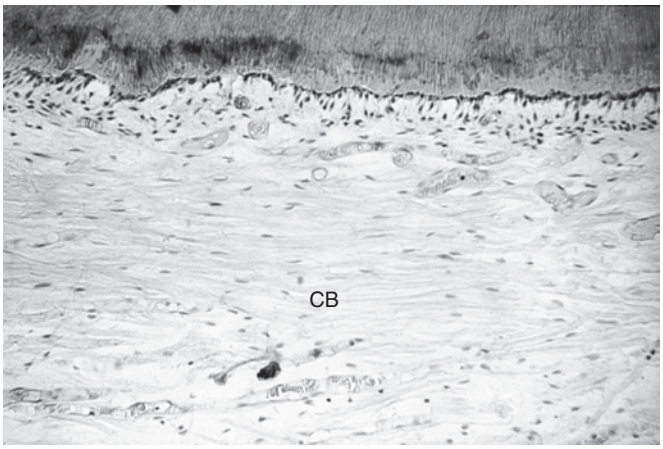

FIG. 12-48 Fibrosis of dental pulp showing replacement of pulp tissue by large collagen bundles (CB).

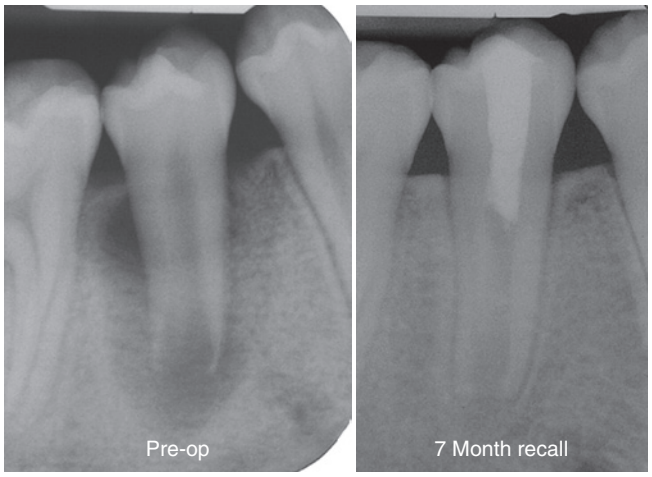

FIG. 12-49 Immature tooth with a necrotic infected canal with apical periodontitis. The canal is disinfected with copious irrigation with sodium hypochlorite and an antibiotic paste. Seven months after treatment, the patient is asymptomatic, and the apex shows healing of the apical periodontitis and some closure of the apex. (From Banchs F, Trope M: Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol? *J Endod* 30:196, 2004.)

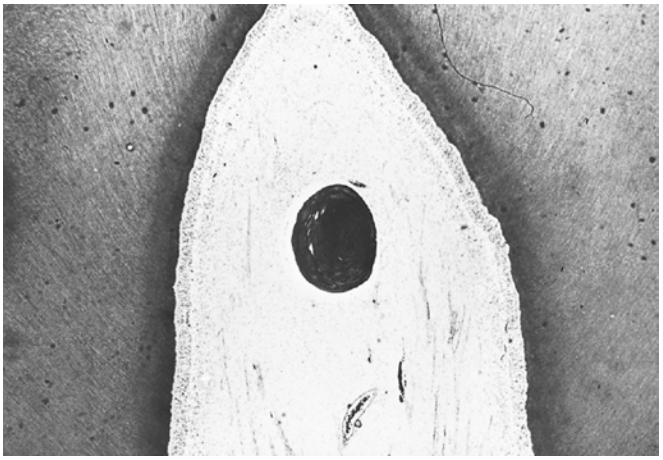

FIG. 12-50 Pulp stone with a smooth surface and concentric laminations in the pulp of a newly erupted premolar extracted in the course of orthodontic treatment.

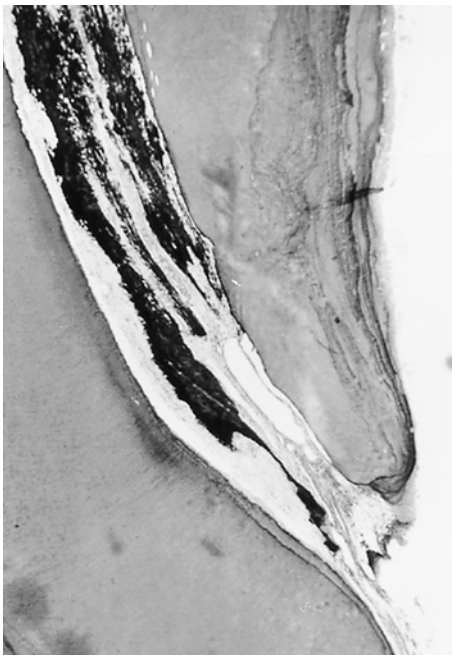

FIG. 12-51 Diffuse calcification near the apical foramen.

tubular-like dentin,[87] and in animal models, root perforations have been treated with scaffolds of collagen, pulpal stem cells, and dentin matrix protein 1, resulting in organized matrix similar to that of pulpal tissue.[304]

Studies investigating new possibilities for regeneration of the pulp/dentin complex are now frequently reported and are described in detail in Chapter 10. A first interesting case report, which has been followed up by others, has led to new strategies for treatment of necrotic immature roots (Fig. 12-49; see also Chapter 10).[13] In the future, the field of pulpal repair will probably develop rapidly, and new treatment strategies will appear.

PULPAL CALCIFICATIONS

Calcification of pulp tissue is a common occurrence. Although estimates of the incidence of this phenomenon vary widely, it is safe to say that one or more pulp calcifications are present in at least 50% of all teeth. In the coronal pulp, calcification usually takes the form of discrete, concentric pulp stones (Fig. 12-50), whereas in the radicular pulp, calcification tends to be diffuse (Fig. 12-51).[374] There is no clear evidence as to whether pulp calcification is a pathologic process related to various forms of injury or a natural phenomenon. The clinical significance of pulp calcification is that it may hinder root canal treatment.

Pulp stones (denticles) range in size from small, microscopic particles often seen in association with the wall of arterioles to accretions that occupy almost the entire pulp chamber (Fig. 12-52). The mineral phase of pulp calcifications has been shown to consist of typical carbonated hydroxyapatite.[374] Histologically, two types of stones are recognized: (1) those that are round or ovoid, with smooth surfaces and concentric laminations (see Fig. 12-50), and (2) those that assume no

particular shape, lack laminations, and have rough surfaces (Fig. 12-53). Laminated stones appear to grow by the addition of collagen fibrils to their surface, whereas unlaminated stones develop by way of the mineralization of preformed collagen fiber bundles. In the latter type, the mineralization front seems to extend out along the coarse fibers, making the surface of the stones appear fuzzy (Fig. 12-54). Often these coarse fiber bundles appear to have undergone hyalinization, thus resembling old scar tissue.

Pulp stones may also form around epithelial cells (i.e., remnants of Hertwig's epithelial root sheath). Presumably the epithelial remnants induce adjacent mesenchymal stem cells to differentiate into odontoblasts. Characteristically these pulp stones are found near the root apex and contain dentinal tubules.

The cause of pulpal calcification is largely unknown. Calcification may occur around a nidus of degenerating cells, blood thrombi, or collagen fibers. Many authors believe that this represents a form of dystrophic calcification. In this type of calcification, calcium is deposited in tissues that are degenerating. Calcium phosphate crystals may be deposited within the cells themselves. Initially this takes place within the mitochondria because of the increased membrane permeability to calcium resulting from a failure to maintain active transport systems within the cell membranes. Thus, degenerating cells serving as a nidus may initiate calcification of a tissue. In the absence of obvious tissue degeneration, the cause of pulpal calcification is enigmatic. It is often difficult to assign the term *dystrophic calcification* to pulp stones because they so often occur in apparently healthy pulps, suggesting that functional stress need not be present for calcification to occur. Calcification in the mature pulp is often assumed to be related to the aging process, but in a study involving 52 impacted canines from patients between 11 and 76 years of age, there was a constant incidence of concentric denticles for all age groups, indicating no relation to aging.[276] Diffuse calcifications, on the other hand, increased in incidence to age 25 years; thereafter they remained constant in successive age groups.

At times, numerous concentric pulp stones with no apparent cause are seen in all the teeth of young individuals. In such cases, the appearance of pulp stones may be ascribed to individual biologic characteristics (e.g., tori, cutaneous nevi).[276]

Although soft-tissue collagen does not usually calcify, it is common to find calcification occurring in old hyalinized scar tissue in the skin. This may be due to the increase in the extent of cross-linking between collagen molecules (because increased cross-linkage is thought to enhance the tendency for collagen

FIG. 12-52 Pulp stones occupying much of the pulp chamber.

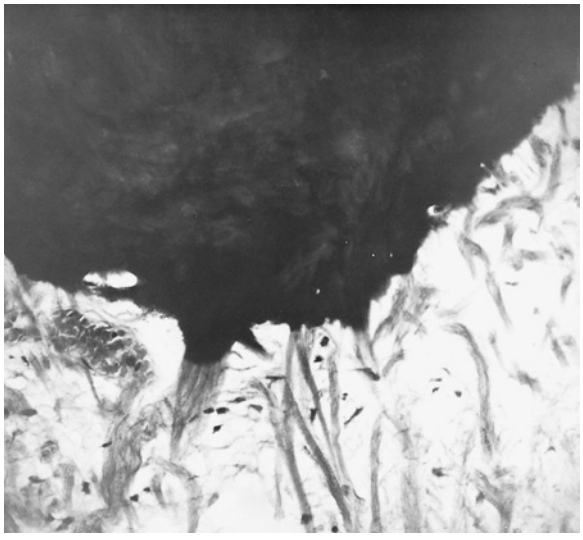

FIG. 12-54 High-power view of a pulp stone from Fig. 12-53, showing the relationship of mineralization fronts to collagen fibers.

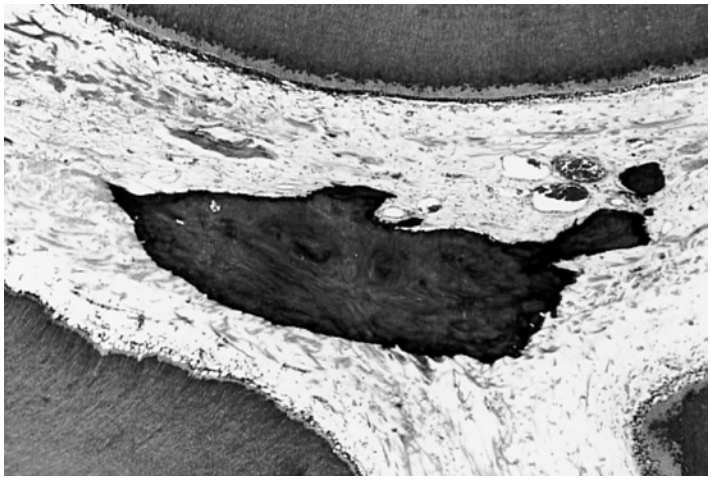

FIG. 12-53 Rough surface form of pulp stone. Note hyalinization of collagen fibers.

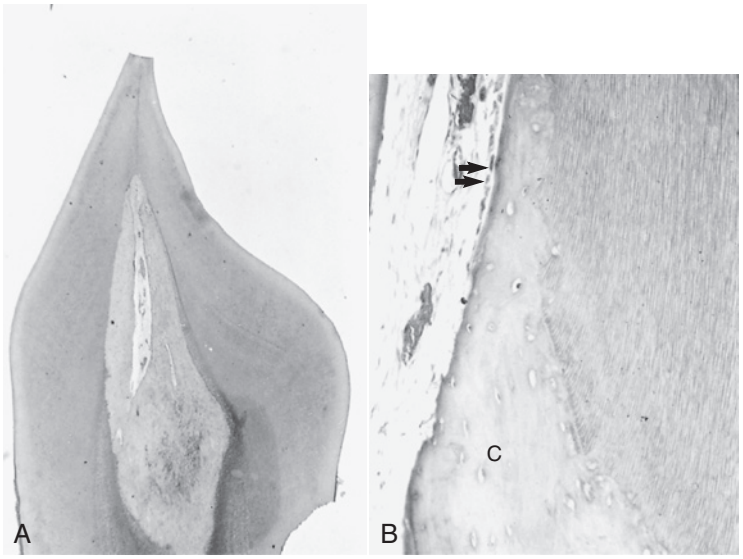

FIG. 12-55 A, Calcific metamorphosis of pulp tissue after luxation of tooth as a result of trauma. Note presence of soft-tissue inclusion. B, High-power view showing cementoblasts *(arrows)* lining cementum (C), which has been deposited on the dentin walls.

fibers to calcify). A relationship may exist between pathologic alterations in collagen molecules within the pulp and pulpal calcification.

Calcification replaces the cellular components of the pulp and may possibly hinder the blood supply, although concrete evidence for this strangulation theory is lacking. Idiopathic pulpal pain was classically attributed to the presence of pulp stones. Modern knowledge of mechanisms of nociceptor activation coupled with the observation that pulp stones are so frequently observed in teeth lacking a history of pain have largely discounted this hypothesis. Therefore, from a clinical perspective, it would be unlikely that a patient's unexplained pain symptoms are due to pulpal calcifications, no matter how dramatic they may appear on a radiograph.

Luxation of teeth as a result of trauma may result in calcific metamorphosis, a condition that can, in a matter of months or years, lead to partial or complete radiographic obliteration of the pulp chamber. The cause of radiographic obliteration is excessive deposition of mineralized tissue resembling cementum or, occasionally, bone on the dentin walls, also referred to as *internal ankylosis* (Fig. 12-55). Histologic examination invariably reveals the presence of some soft tissue, and cells resembling cementoblasts can be observed lining the mineralized tissue. This calcific metamorphosis of the pulp has also been reported in replanted teeth of the rat.[275]

Clinically, the crowns of teeth affected by calcific metamorphosis may show a yellowish hue compared with adjacent normal teeth. This condition usually occurs in teeth with incomplete root formation. Trauma results in disruption of blood vessels entering the tooth, thus producing pulpal infarction. The wide periapical foramen allows connective tissue from the periodontal ligament to proliferate and replace the infarcted tissue, bringing with it cementoprogenitor and osteoprogenitor cells capable of differentiating into either cementoblasts or osteoblasts or both.

When calcific metamorphosis is noted on a patient's radiograph, it is sometimes suggested that the tooth be treated endodontically because the pulp is expected to be secondarily infected, and endodontic therapy should be performed while the pulp canal is still large enough to instrument. In a classic study of luxated teeth, Andreasen[7] found that only 7% of the pulps that underwent calcific metamorphosis exhibited secondary infection. Because the success rate for nonsurgical endodontic therapy, not only in general[399] but also for obliterated teeth,[67] is considered high, prophylactic intervention does not seem to be warranted.

AGE CHANGES

Continued formation of secondary dentin throughout life gradually reduces the size of the pulp chamber and root canals, although the width of the cementodentinal junction appears to stay relatively the same.[109,342] In addition, certain regressive changes in the pulp appear to be related to the aging process (see also Chapter 26). There is a gradual decrease in the cellularity and a concomitant increase in the number and thickness of collagen fibers, particularly in the radicular pulp. The thick collagen fibers may serve as foci for pulpal calcification (see Fig. 12-53). The odontoblasts decrease in size and number, and they may disappear altogether in certain areas of the pulp, particularly on the pulpal floor over the bifurcation or trifurcation areas of multirooted teeth.

With age there is a progressive reduction in the number of nerves[102] and blood vessels.[23,25] Evidence also suggests that aging results in an increase in the resistance of pulp tissue to the action of proteolytic enzymes,[420] hyaluronidase, and sialidase,[25] suggesting an alteration of both collagen and proteoglycans in the pulps of older teeth. The main changes in dentin associated with aging are an increase in peritubular dentin, dentinal sclerosis, and the number of dead tracts.*[342] Dentinal sclerosis produces a gradual decrease in dentinal permeability as the dentinal tubules become progressively reduced in diameter.[350]

*The term *dead tract* refers to a group of dentinal tubules in which odontoblast processes are absent. Dead tracts are easily recognized in ground sections because the empty tubules refract transmitted light, and the tract appears black in contrast to the light color of normal dentin.

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CHAPTER 13

Pulpal Reactions to Caries and Dental Procedures

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CHAPTER OUTLINE

Pulpal Reaction to Caries
Neurogenic Mediators
Correlation Between Clinical Symptoms and Actual Pulpal Inflammation
Dentin Hypersensitivity and Its Management
Pulpal Reactions to Local Anesthetics
Pulpal Reactions to Restorative Procedures
The Degree of Inflammation of the Pulp Preoperatively
The Amount of Physical Irritation Caused by the Procedure
The Proximity of the Restorative Procedures to the Dental Pulp and the Surface Area of Dentin Exposed
The Permeability of Dentin and the Odontoblastic Layer Between the Area Being Restored and the Pulp
The Age of the Patient
Pulpal Reactions to Restorative Materials
Direct Pulp Capping with Mineral Trioxide Aggregate
The Use of Hemostatic Agents and Disinfectants on Direct Pulp Exposures
Pulpal Reactions to Laser Procedures
Lasers in the Prevention, Diagnosis, and Treatment of Caries
Lasers in the Treatment of Dentin Hypersensitivity
Use of Lasers as a Protective Measure for Dentin, Under Traditional Cavity Preparation
Pulpal Reactions to Vital Bleaching Techniques
Pulpal Reactions to Periodontal Procedures
Mechanical Irritants: Orthodontic Movement
Pulpal Reactions to Orthodontic/Orthognathic Surgery
Biomechanical Irritation: Parafunctional Habits
Pulpal Reactions to Implant Placement and Function

The dental pulp is a dynamic tissue that responds to external stimuli in many ways. However, there are certain unique features about the dental pulp response that distinguish it from other connective tissues in the body. The pulp's exposure to dental caries, a prevalent chronic infectious disease, its encasement in an unyielding environment after complete tooth maturation, and the scarcity of collateral circulation render it susceptible to injury and complicate its regeneration. Moreover, the pulp is endowed with a rich neurovascular supply that regulates the effects of inflammation that may ultimately lead to rapid degeneration and necrosis. The treatment of dental caries and other tooth abnormalities involves removal of the enamel and dentin, the hardest tissues in the body, thus adding to the irritation of the pulp. This chapter discusses the response of the pulp to all of these variables and presents advances in our understanding of dental procedures and their effects on the pulp.

PULPAL REACTION TO CARIES

Dental caries is a localized, destructive, and progressive infection of dentin, which, if left unchecked, can result in pulpal necrosis and potential tooth loss. Both bacterial by-products and products from the dissolution of the organic and inorganic constituents of dentin mediate the effects of dental caries on the pulp. Three basic reactions tend to protect the pulp against caries: (1) a decrease in dentin permeability, (2) tertiary dentin formation, and (3) inflammatory and immune reactions.[133] These responses occur concomitantly, and their robustness is highly dependent on the aggressive nature of the advancing lesion as well as host responses such as the age of the patient (see also Chapter 26).

In the advancing infection front of the carious lesion, multiple intrinsic and extrinsic factors are released that stimulate nearby pulpal tissue. Bacterial proteolytic enzymes, toxins, and metabolic by-products have been thought to initiate pulpal reactions, yet the buffering capacity of dentin and dentinal fluid likely attenuate these deleterious effects. This protective function is significantly reduced when the remaining dentin thickness is minimal.[237] When relatively unhindered access to pulpal tissue is present, both bacterial metabolites and their cell wall components induce inflammation. In initial-to-moderate lesions, current evidence suggests that acidic by-products of the carious process act indirectly by degrading the dentin matrix and thereby liberating bioactive molecules previously sequestered during dentinogenesis. Once liberated, these molecules again assume their role in dentin formation, this time stimulatory for *tertiary* dentinogenesis.[238] This theory is

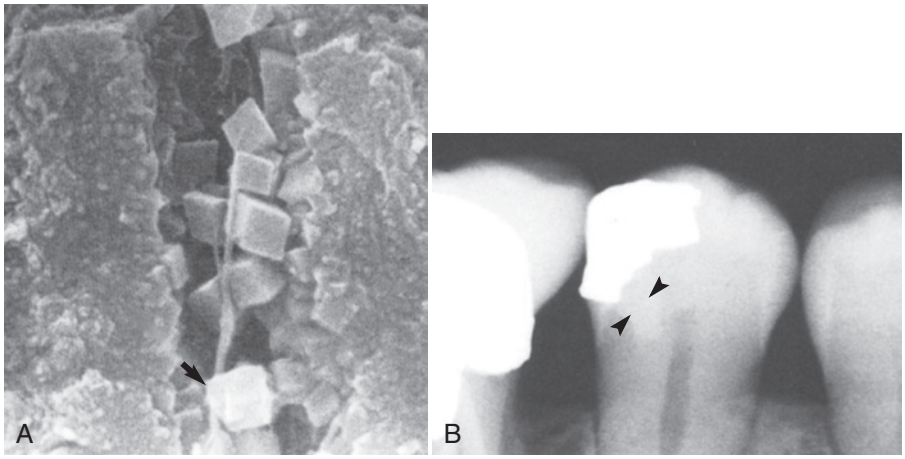

FIG. 13-1 A, Whitlockite crystals occlude the dentinal tubules in sclerotic dentin. B, Dentinal sclerosis is radiographically apparent beneath a deep class II lesion. (*A,* From Yoshiyama M, Masada J, Uchida A, Ishida H: Scanning electron microscopic characterization of sensitive vs. insensitive human radicular dentin, *J Dent Res* 68:1498, 1989.)

supported by the findings that demineralized dentin matrix implanted at the site of pulpal exposure can induce dentinogenesis.[266] Furthermore, placement of purified dentin matrix proteins on exposed dentin or exposed pulp stimulates tertiary dentin formation, indicating that these molecules can act directly or across intact dentin.[239,265] More information on growth factors embedded in dentin is reviewed in the chapter on regenerative endodontics (see Chapter 10).

Evidence offers several candidate molecules that stimulate reparative dentinogenesis. Heparin-binding growth factor, transforming growth factor (TGF)-β1, TGF-β3, insulin-like growth factors I and II, platelet-derived growth factor, bone morphogenetic protein-2 (BMP-2) and angiogenic growth factors have been shown to be embedded in dentin and stimulatory for dentinogenesis in vitro. The TGF-β superfamily in particular seems to be important in the signaling process for odontoblast differentiation from mesenchymal stem or progenitor cells as well as primary and tertiary dentinogenesis. As the predominant isoform, TGF-β1 is equally distributed in the soluble and insoluble fractions of dentin matrix.[41] During the carious dissolution of dentin, it is believed that the soluble pool of TGF-β1 can diffuse across intact dentin while the insoluble pool is immobilized on insoluble dentin matrix and serves to stimulate odontoblasts much like membrane-bound TGF-βs during odontogenesis.[237,240]

Despite the research interest in tertiary dentinogenesis, it is neither the first nor necessarily the most effective pulpally mediated defense against invading pathogens. A combination of an increased deposition of intratubular dentin and the direct deposition of mineral crystals into the narrowed dentin tubules to decrease dentin permeability is the first defense to caries and is called *dentin sclerosis*. It occurs by a combination of increased deposition of intratubular dentin and tubule occlusion by precipitated crystals. This results in an effective decrease in dentin permeability underneath the advancing carious lesion.[206] In vitro studies with cultured tooth slices implicate TGF-β1 as a central player in the increased deposition of intratubular dentin.[236] The deposition of whitlockite crystals in the tubular lumen most likely results from a similar stimulation of vital associated odontoblasts, possibly in combination with precipitation of mineral released during the demineralization process[156,262] (Fig. 13-1).

FIG. 13-2 Reactionary dentinogenesis (TD). Note the tubular morphology and the discontinuity of the tubules at the interface of secondary and reactionary dentin. Resident odontoblasts are still present.

The formation of tertiary dentin occurs over a longer period than does that of sclerotic dentin, and its resultant character is highly dependent on the stimulus. Mild stimuli activate resident quiescent odontoblasts whereupon they elaborate the organic matrix of dentin. This type of tertiary dentin is referred to as *reactionary dentin* and can be observed when initial dentin demineralization occurs beneath the noncavitated enamel lesion.[151] Mediators present during the carious process induce a focal upregulation of matrix production by resident odontoblasts. The resultant dentin is similar in morphology to physiologic dentin and may only be apparent due to a change in the direction of the new dentinal tubules (Fig. 13-2). In contrast, in aggressive lesions the carious process may prove cytocidal to subjacent odontoblasts and require repopulation of the

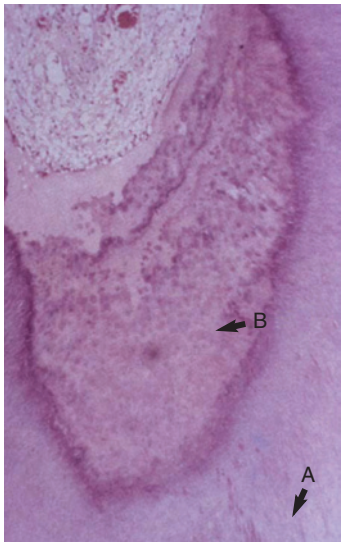

FIG. 13-3 Reparative dentin; the strong stimulus of the impinging infection is cytocidal for odontoblasts. The resultant dentin is irregular with soft tissue inclusions.

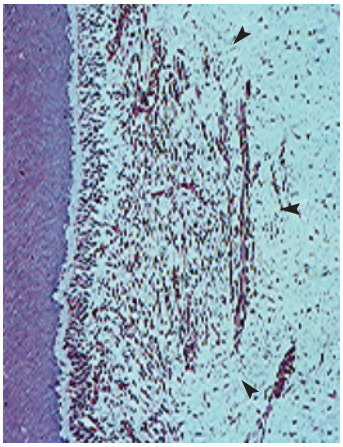

FIG. 13-4 The early pulpal response to caries is represented by a focal accumulation of chronic inflammatory cells. Note that peripheral to the inflammation the pulpal parenchyma is relatively unaffected.

disrupted odontoblast layer with differentiating progenitors. The organization and composition of the resultant matrix are a direct reflection of the differentiation state of the secretory cells. This accounts for the heterogeneity of *reparative dentin,* where the morphology can range from organized tubular dentin to more disorganized irregular *fibrodentin*. Fibrodentin, due to its irregular configuration and tissue inclusions, is more permeable than physiologic dentin[275] (Fig. 13-3).

Although dentin can provide a physical barrier against noxious stimuli, the pulpal immune response provides humoral and cellular challenges to invading pathogens. In the progressing carious lesion, the host immune response increases in intensity as the infection advances. It has been shown that titers of T-helper cells, B-lineage cells, neutrophils, and macrophages are directly proportional to lesion depth in human teeth.[118] The disintegration of large amounts of dentin, however, is not necessary to elicit a pulpal immune response. This is supported by the observation that a pulpal inflammatory response can be seen beneath noncavitated lesions and noncoalesced pits and fissures.[34]

The early inflammatory response to caries is characterized by the focal accumulation of chronic inflammatory cells (Fig. 13-4). This is mediated initially by odontoblasts and later by dendritic cells. As the most peripheral cell in the pulp, the odontoblast is positioned to encounter foreign antigens first and initiate the innate immune response. Pathogen detection in general is accomplished via specific receptors called pattern recognition receptors (PRRs).[120] These receptors recognize pathogen associated molecular patterns (PAMPs) on invading organisms and initiate a host defense through the activation of the NF-κB pathway.[101] One class of the PAMP recognition molecules is the Toll-like receptor family (TLRs). Odontoblasts have been shown to increase expression of certain TLRs in response to bacterial products. Under experimental conditions, odontoblasts expression of TLR3, 5, and 9 increased in response to lipoteichoic acid, whereas lipopolysaccharide increased TLR2 and 4 expression.[66,107,183] It was also shown that TGFβ-1 inhibits the expression of TLR2 and 4 by odontoblasts in response to gram-positive and gram-negative bacteria.[27,107]

Once the odontoblast TLR is stimulated by a pathogen, proinflammatory cytokines, chemokines, and antimicrobial peptides are elaborated by the odontoblast, resulting in recruitment and stimulation of immune effector cells as well as direct bacterial killing.[74,76,129]

Many cells produce chemokines at low levels constitutively. Unstimulated odontoblasts express genes coding for CCl2, CXCL12, and CXCL14, three genes known to code for factors chemotactic for immature dendritic cells.[42] They also produce CCL26, a natural antagonist for CCR1, CCR2, and CCR5 that are chemokines normally produced by monocytes and dendritic cells.[287] Stimulation with bacterial cell wall constituents has been shown to upregulate the expression of multiple chemokine genes including CXCL12, CCL2, CXCL9, CX3CL1, CCL8, CXCL10, CCL16, CCL5, CXCL2, CCL4, CXCL11, and CCL3, and nine chemokine receptor genes including CXCR4, CCR1, CCR5, CX3CR1, CCR10, and CXCR3, suggesting that odontoblasts sense pathogens and express factors that recruit immune effector cells[42,75,106,152] (Fig. 13-5). These data suggest a scenario whereby stimulated odontoblasts express high levels of chemokines such as IL-8 (CXCL8) that act in concert with the release of formerly sequestered growth factors from carious dentin that induce a focal increase in dendritic cell numbers with the additional release of chemotactic mediators.[77,240] The subsequent influx of immune effector cells is composed of lymphocytes, macrophages, and plasma cells. This cellular infiltrate is accompanied by localized capillary sprouting in response to angiogenic factors as well as co-aggregation of nerve fibers and human leukocyte antigen-DR (HLA-DR)-positive dendritic cells.[280,281]

As the carious lesion progresses, the density of the chronic inflammatory infiltrate as well as that of dendritic cells in the odontoblast region increases. Pulpal dendritic cells are responsible for antigen presentation and stimulation of T lymphocytes. In the uninflamed pulp they are scattered throughout the pulp. With caries progression they aggregate initially in the pulp and subodontoblastic regions, then extend into the odontoblast layer, and eventually migrate into the entrance to tubules beside the odontoblast process[282] (Fig. 13-6). Two distinct populations of dendritic cells have been identified in the dental pulp. CD11c+ is found in the pulp/dentin border and

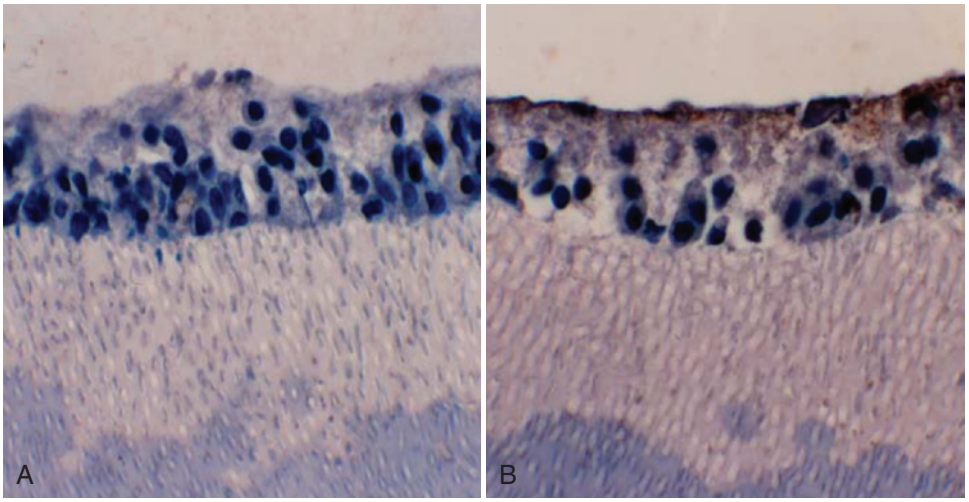

FIG. 13-5 Odontoblasts exposed to LPS in an in vitro culture model express IL-8, as evidenced by immunostaining with anti–IL-8 antibodies.

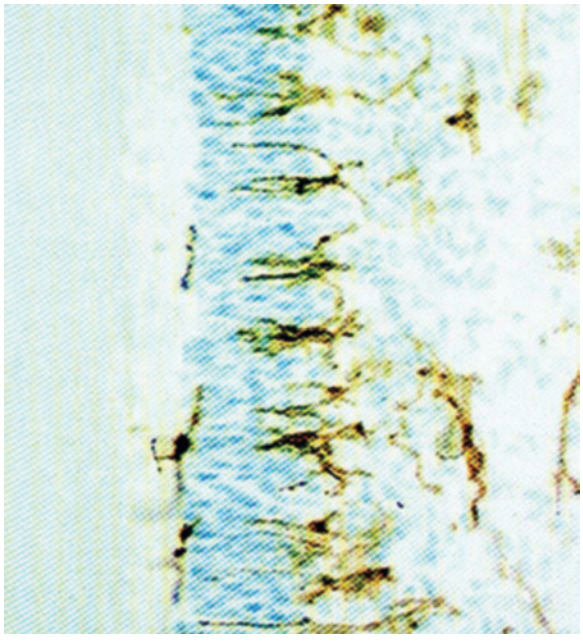

FIG. 13-6 Dental caries stimulates the accumulation of pulpal dendritic cells in and around the odontoblastic layer. (Reprinted with permission from Mats Jontell.)

subjacent to pits and fissures. F4/80+ dendritic cells are concentrated in the perivascular spaces in the subodontoblastic zone and inner pulp.[287] CD11c+ dendritic cells express Toll-like receptors 2 and 4 and are CD205 positive. F4/80+ dendritic cells have migratory ability. As they migrate from the central pulp they increase in size and become CD86 positive. The close spatial relationship between odontoblasts and dendritic cells under the carious lesion has led to speculation that dendritic cells may play a role in odontoblast differentiation or secretory activity in the immune defense and in dentinogenesis. Recent studies have demonstrated that pulp dendritic cell can migrate to regional lymph nodes, for antigen presentation.[24a] In vitro studies have suggested that the secretion of granulocyte-macrophage colony-stimulating factor (GM-CSF) and osteopontin by dendritic cells and macrophages represents a mechanism whereby they contribute to odontoblast differentiation.[227] Pulpal Schwann cells have also been shown to produce molecules in response to caries, which indicates the acquisition of the ability for antigen presentation.

Evidence suggests that odontoblasts also play a role in the humoral immune response to caries. IgG, IgM, and IgA have been localized in the cytoplasm and cell processes of odontoblasts in human carious dentin, suggesting that these cells actively transport antibodies to the infection front.[189] In the incipient lesion, antibodies accumulate in the odontoblast layer and with lesion progression can be seen in the dentinal tubules. Eventually this leads to a focal concentration of antibodies beneath the advancing lesion.[188]

In the most advanced phase of carious destruction, the humoral immune response is accompanied by immunopathologic destruction of pulpal tissue. In animal studies where monkeys were hyperimmunized to bovine serum albumin (BSA), there was an observed increase in pulpal tissue destruction subsequent to antigenic challenge across freshly cut dentin.[20] The odontoblasts also appear to be involved in the production of innate antimicrobial molecules such as human beta defensing-2 (HBD2). Therefore, interleukin (IL)-1 and tumor necrosis factor (TNF)-alpha as well as bacterial lipopolysaccharide (LPS) were responsible for significant increases in HBD2 in response to caries.[106] In summary, it appears the odontoblasts play a central role in orchestrating local and chemotactic inflammatory responses to dental caries (Fig. 13-7).

Pulpal exposure in primary and immature permanent teeth can lead to a proliferative response or *hyperplastic pulpitis*. Exuberant inflammatory tissue proliferates through the exposure and forms a "pulp polyp" (Fig. 13-8). It is presumed that a rich blood supply allows this proliferative response. Conventional root canal therapy or progressive vital pulp therapy is indicated.

NEUROGENIC MEDIATORS

Neurogenic mediators are involved in the pulpal response to irritants and, like immune components, they can mediate pathology as well as the healing response (see also Chapter 12).

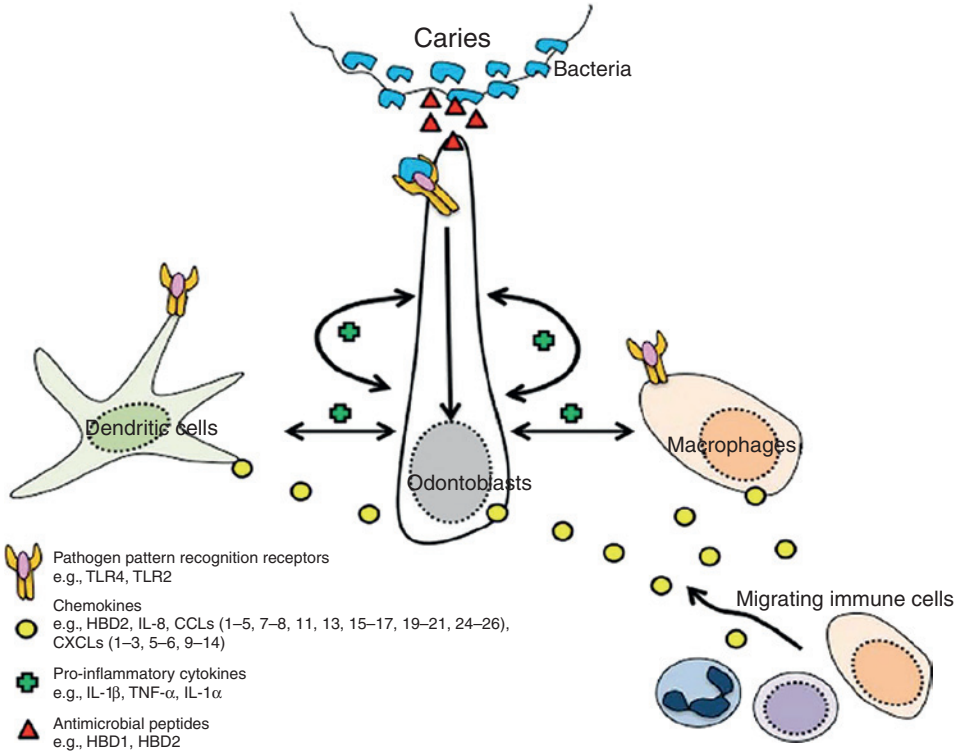

FIG. 13-7 Innate immunity in the odontoblast layer (ODL). Bacterial components from caries activate cytokine/chemokine release from odontoblasts, dendritic cells, or macrophages via Toll-like receptors (TLRs). Proinflammatory cytokines released from these cells act as autocrine and paracrine signals to amplify cytokine responses. including antimicrobial peptide, cytokine, and chemokine production. The release of chemokines creates a migration gradient for immune cells to ODL while antimicrobial peptides reduce bacterial load. (From Horst OV, Horst JA, Samudrala R, Dale BA: Caries induced cytokine network in the odontoblast layer of human teeth, *BMC Immunol* 12:9, Fig. 4C, 2011. Reprinted with permission.)

FIG. 13-8 A proliferative response to caries in a young tooth, typically referred to as proliferative pulpitis, hyperplastic pulpitis, or pulp polyp. (Courtesy Dr. Howard Strassler, University of Maryland, with permission.)

External stimulation of dentin causes the release of proinflammatory neuropeptides from pulpal afferent nerves.[36,130] Substance P (SP), calcitonin gene–related peptide (CGRP), neurokinin A (NKA), neurokinin Y, and vasoactive intestinal peptide are released and effect vascular events such as vasodilation and increased vascular permeability. This results in a net increase in tissue pressure that can progress to necrosis in extreme and persistent circumstances. Stimulation of sympathetic nerves in response to the local release of mediators such as norepinephrine, neuropeptide Y, and adenosine triphosphate (ATP) has been shown to alter pulpal blood flow. Receptor field studies as well as anatomic studies have shown sprouting of afferent fibers in response to inflammation.[36]

Neuropeptides can act to modulate the pulpal immune response. It has been demonstrated that SP acts as a chemotactic and stimulatory agent for macrophages and T lymphocytes. The result of this stimulation is increased production of arachidonic acid metabolites, stimulation of lymphocytic mitosis, and production of cytokines. CGRP demonstrates immunosuppressive activity, which is evidenced by a diminution of class II antigen presentation and lymphocyte proliferation.

SP and CGRP are mitogenic for pulpal and odontoblast-like cells; thereby they initiate and propagate the pulpal healing response.[260] CGRP has been shown to stimulate the production of bone morphogenic protein by human pulpal cells. The result of this stimulation has been postulated to induce tertiary dentinogenesis.[38] Substance P appears to increase in the dental pulp and periodontal ligament as a result of acutely induced occlusal trauma,[45] which may be related to the pain associated with concussion traumatic injury.

It has been shown that there may be gender differences in CGRP release in the dental pulp.[29,159] In one study, serotonin (a peripheral pronociceptive mediator) induced a significant increase in capsaicin-evoked CGRP release in dental pulps

obtained from female but not male patients.[159] This interplay of inflammatory mediators may explain some of the gender differences in clinical presentation with dental pain.

CORRELATION BETWEEN CLINICAL SYMPTOMS AND ACTUAL PULPAL INFLAMMATION

From a clinical perspective, it would be most helpful to the clinician to be able to diagnose pulpal conditions from a profile of symptoms with which a patient presents. If symptoms are not conclusive, a number of objective tests should aid the clinician in reaching a definitive diagnosis of the pulpal pathologic status. In actuality, combinations of subjective and objective findings are frequently insufficient in reaching definitive diagnosis of the status of the dental pulp. This is particularly true in cases of vital inflamed pulp, where it is difficult for the practitioner to determine clinically whether the inflammation is reversible or irreversible.

Many practitioners rely on painful symptoms to determine the status of the pulp. Several studies have examined this question in some detail. A number of classic studies were performed in which the subjective and objective clinical findings related to carious teeth were recorded prior to extracting the teeth and examining them histologically. The underlying hypothesis in these studies was that the more severe the clinical symptoms, the more intense pulpal inflammation and destruction was evident histologically. These studies showed that in the vital pulp, clinical symptoms generally did not correlate with gross histologic findings.[100,169,232] Furthermore, carious pulp exposure was associated with severe inflammatory response or liquefactive necrosis, regardless of symptoms (Fig. 13-9). These histologic changes ranged in extent from being present only at the site of the exposure to deep into the root canals.[232] In a few studies prolonged or spontaneous severe symptoms were associated with chronic partial, total pulpitis, or pulp necrosis.[64,232] However, in these as well as other studies it was common to find cases with evidence of severe inflammatory responses including partial necrosis histologically, but with little or no clinical symptoms—the so-called painless pulpitis.[64,100,169,232] Moreover, the density of nerve fibers[224] and the vascularity[225] in inflamed pulp do not correlate with clinical symptoms in primary and permanent teeth. It has been reported that the incidence of painless pulpitis that leads to pulp necrosis and asymptomatic apical periodontitis is about 40% to 60% of all pulpitis cases.[168]

FIG. 13-9 Histologic photomicrograph of a molar with carious pulp exposure. The exposure had been capped but had failed and the patient presented with symptoms. The photomicrograph shows an area of necrosis and extensive inflammation throughout the coronal pulp. (Courtesy Dr. Larz Spangberg, University of Connecticut, with permission.)

Objective clinical findings are essential for determining the vitality of the pulp and whether the inflammation has extended into the periapical tissues (see also Chapter 1). Lack of response to electric pulp testing generally indicates that the pulp has become necrotic.[221,232] Thermal pulp testing is valuable for reproducing a symptom of thermal sensitivity and allowing the practitioner to assess the reaction of the patient to a stimulus and the duration of the response. However, pulp testing cannot determine the degree of pulpal inflammation.[64,232] These studies show that irreversible pulpal inflammation can be diagnosed with some certainty only in cases where, in addition to being responsive to pulp testing, the pulp develops severe spontaneous symptoms. Pulp necrosis could be predictably diagnosed by a consistent negative response to pulp tests, preferably to both cold and electrical tests to avoid false responses.[210,211] Pulp necrosis could be verified by a test cavity or lack of hemorrhagic pulp tissue upon access preparation. It should be noted, however, that the latter sign should be assessed cautiously. Occasionally, the pulp space is very small, such as in older individuals with calcified canals, and hemorrhage upon access to the pulp may not be clinically appreciable. Conversely, cases with pulp necrosis and acute periapical infections may have hemorrhagic purulent drainage through the large pulp space upon access preparation, particularly after initial instrumentation.

The lack of correlation between the histologic status of the pulp and clinical symptoms may be explained by advances in the science of pulp biology. Studies have shown that numerous molecular mediators may act in synchrony to initiate, promote or modulate the inflammatory response in the dental pulp. The nature and quantity of these inflammatory mediators cannot be determined from histologic analysis, without the use of specialized staining techniques. Many of these molecular mediators tend to reduce the pain threshold, either directly by acting on peripheral nerve cells or by promoting the inflammatory process. Thus, a number of these mediators were shown to be elevated in human pulp diagnosed with painful pulpitis. These mediators include prostaglandins,[51,223] the vasoactive amine bradykinin,[150] tumor necrosis factor alpha,[139] neuropeptides such as substance P,[30] CGRP and neurokinin A,[13] and catecholamines.[185] In fact, it was even shown that when patients have painful pulpitis, the crevicular fluid related to the affected teeth has significantly increased neuropeptides compared to the levels in contralateral teeth.[13] In another study, trained volunteers stimulated an incisor with a constant current three fold the threshold value for 90 seconds.[12] This resulted in a significant increase in crevicular matrix metalloproteinase 8 (MMP-8), one of the collagenases involved in tissue destruction.

It has also been determined that peripheral opioid receptors are present in the dental pulp,[119] and these could play a role in why many cases with irreversible pulpitis are asymptomatic. As noted before, carious teeth are frequently not associated with significant symptoms. However, they still have a significant

amount of inflammation. The pulp in teeth with mild to moderate caries has increased neuropeptide Y,[68] and its Y1 receptor,[69] compared to that in normal teeth. Neuropeptide Y is a neurotransmitter for the sympathetic nervous system and is thought to act as a modulator of neurogenic inflammation. Likewise, the levels of vasoactive intestinal peptide (VIP), although not its receptor VPAC1, seemed to increase in the pulp of moderately carious teeth.[67]

With the advances in molecular biology, efficient detection of hundreds of molecular mediators simultaneously by their gene expression has become a reality. Current research seeks to examine which genes are specifically expressed or upregulated in the pulp, in response to the carious lesion. In this regard, preliminary studies have shown that various cytokines and other inflammatory mediators are upregulated underneath a carious lesion in a manner that correlates with the depth of caries.[164] Several researchers have used gene microarrays to obtain an accurate mapping of candidate genes that show elevated expression in inflamed pulp and the odontoblastic cell layer.[165,194,195] In addition, research has revealed the differential expression of microRNAs (miRNAs) in the healthy and diseased dental pulp.[288] MiRNAs are noncoding RNA molecules that regulate gene expression in complex inflammatory responses and may eventually assist in clinical predictions of pulpal status. Therefore, more accurate chairside diagnostic methods are potentially feasible to develop, especially a method that involves sampling from crevicular fluid, dentinal fluid, or the pulp directly. For this reason, more research is needed to determine the key mediators that would predict survival or degeneration of the dental pulp in difficult diagnostic cases.

DENTIN HYPERSENSITIVITY AND ITS MANAGEMENT

Dentin hypersensitivity is a special situation in which a significant, chronic, pulpal pain arises, which does not seem to be associated with irreversible pulpal pathosis in the majority of cases. Dentin hypersensitivity is characterized by brief sharp pain arising from exposed dentin in response to stimuli, typically thermal, evaporative, tactile, osmotic, or chemical, that cannot be ascribed to any other form of dental defect or pathosis.[104] Facial root surfaces in canines, premolars, and molars are particularly affected, especially in areas of periodontal attachment loss. Dentin hypersensitivity may be related to excessive abrasion during tooth brushing, periodontal disease, or erosion from dietary or gastric acids,[2,3,47] and it may increase following scaling and root planing.[47,274] The dentin is hypersensitive, most likely due to the lack of protection by cementum, loss of smear layer by acidic dietary fluids, and the hydrodynamic movement of fluid in dentinal tubules.[4,33] The degree of inflammation in the pulp in cases of dentin hypersensitivity is not well characterized, because the condition is usually not severe enough to warrant tooth extraction or endodontic therapy. However, patent dentinal tubules are present in areas of hypersensitivity[284] (Fig. 13-10) and may result in increased irritation and localized reversible inflammation of the pulp at the sites involved.

The application of neural modulating agents such as potassium nitrate,[163] or tubule blocking agents such as strontium chloride, oxalates or dentin bonding agents (Fig. 13-11),[4,202] usually alleviates the condition, at least temporarily. However, the placement of passive molecules or crystals may provide only temporary relief, thus there has been the need to provide biocompatible materials that bond to the root surface in order to provide a more lasting solution. One such material was a calcium sodium phosphosilicate bioactive glass,[157] which was developed into a commercial product (SootheRx, NovaMin Technology Inc., Alachua, FL). Another product uses a combination of a calcium oxalate and an acid-etched bonding material to seal the dentinal tubules (BisBlock, Bisco Inc., Schaumberg, IL). A concern has been raised that the acidic pH during etching may cause dissolution of the oxalate crystals, thus interfering with the effectiveness of the material.[279] However, one study found that BisBlock and two other products—Seal&Protect (Dentsply Professional, York, Pennsylvania) and Vivasens (Ivoclar Vivadent AG, Schaan, Liechtenstein)—were effective compared to placebo several weeks after treatment.[198] In the long term, the development of smear layer, such as from tooth brushing, dentin sclerosis, reactionary dentin, and the blockage of tubules with large endogenous macro molecules, is all thought to reduce the problem[203] (see animation from the online edition of this chapter). A practice-based, randomized clinical trial compared the effectiveness of non-desensitizing toothpaste (Colgate Cavity Protection Regular, Colgate-Palmolive, New York, New York), desensitizing toothpaste (Colgate Sensitive Fresh Stripe, Colgate-Palmolive), and a professionally applied desensitizing agent (Seal & Protect).[89] The findings showed a significant reduction of dentin hypersensitivity in the desensitizing therapies compared to the non-desensitizing group that was a much more significant reduction in the professionally applied desensitizing agent over a 6-month period.

PULPAL REACTIONS TO LOCAL ANESTHETICS

An intact pulpal blood flow is critical for maintaining the health of the dental pulp. Because the dental pulp is enclosed in a rigid chamber and is supplied by few arterioles through the apical foramina, it cannot benefit from collateral circulation or volumetric changes that compensate for changes in blood flow in other soft tissues. Furthermore, reduction in blood flow has the compounding effect of reducing the clearance of large molecular weight toxins or waste products,[201] thus causing irreversible pulpal pathosis. Vasoconstrictors are added to local anesthetics to enhance the duration of anesthesia. However, vasoconstrictors in local anesthetics could negatively impact the health of the pulp if they reduce blood flow, particularly if the pulp is inflamed preoperatively. Earlier studies have documented that vasoconstrictors in local anesthetics do reduce pulpal blood flow in experimental animals when administered by infiltration and nerve block[132] (Fig. 13-12), and that this effect was more severe with periodontal ligament injections[131] (Fig. 13-13). More recently, clinical trials were conducted in which subjects were given infiltration of different local anesthetics with or without epinephrine at a concentration of 1:100,000 and the pulpal blood flow was measured by laser Doppler flowmetry. In groups that received the epinephrine, there were consistently significant reductions in pulpal blood flow,[5,49,177] even if the infiltration was palatal to maxillary premolars.[213] Interestingly, in one study the reduction in pulpal blood flow with epinephrine infiltration was more than the reduction in gingival blood flow and did not return to baseline

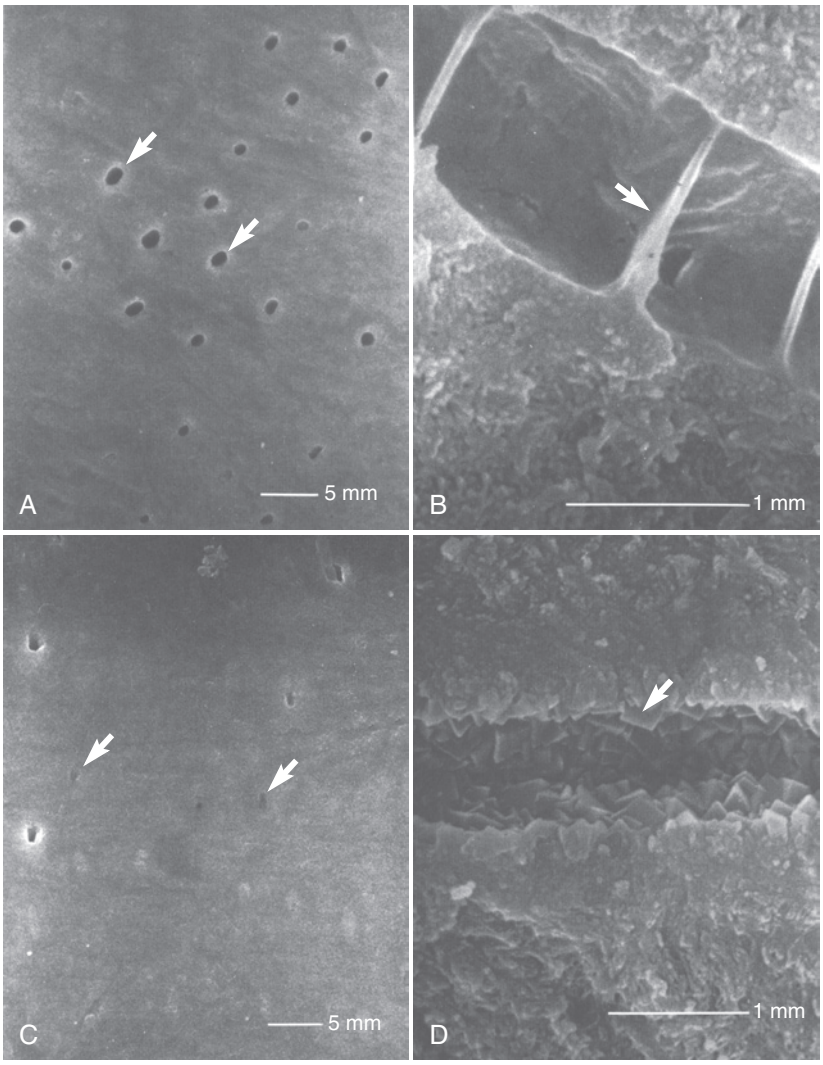

FIG. 13-10 SEM image of an exposed dentin surface of a hypersensitive area. **A,** A large proportion of dentinal tubules *(arrows)* are shown to be open. **B,** SEM image of a fractured dentinal tubule of a hypersensitive area. The lumen of the dentinal tubule is partitioned by membranous structures *(arrow).* **C,** SEM image of exposed dentin surface of a naturally desensitized area. The lumens of dentinal tubules *(arrows)* are mostly occluded, and the surface is extremely smooth. **D,** SEM image of a fractured dentinal tubule of a naturally desensitized area. Rhombohedral platelike crystals of from 0.1 to 0.3 μm *(arrow)* are present. (From Yoshiyama M, Masada J, Uchida A, Ishida H: Scanning electron microscopic characterization of sensitive vs. insensitive human radicular dentin, *J Dent Res* 68:1498, 1989. Reprinted with permission.)

FIG. 13-11 Smear layer treated with 30% dipotassium oxalate for 2 minutes plus 3% monopotassium and monohydrogen oxalate for 2 minutes. Dentin surface is completely covered with calcium oxalate crystals (original magnification ×1900). (From Pashley DH, Galloway SE: The effects of oxalate treatment on the smear layer of ground surfaces of human dentine, *Arch Oral Biol* 30:731, 1985. Reprinted with permission.)

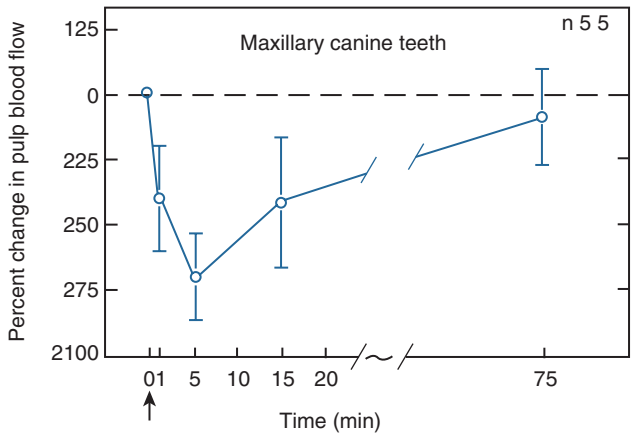

FIG. 13-12 Effects of infiltration anesthesia (i.e., 2% lidocaine with 1:100,000 epinephrine) on pulpal blood flow in the maxillary canine teeth of dogs. There is a drastic decrease in pulpal blood flow soon after the injection. Arrow indicates the time of injection. Bars depict standard deviation. (From Kim S, Edwall L, Trowbridge H, Chien S: Effects of local anesthetics on pulpal blood flow in dogs, *J Dent Res* 63:650, 1984. Reprinted with permission.)

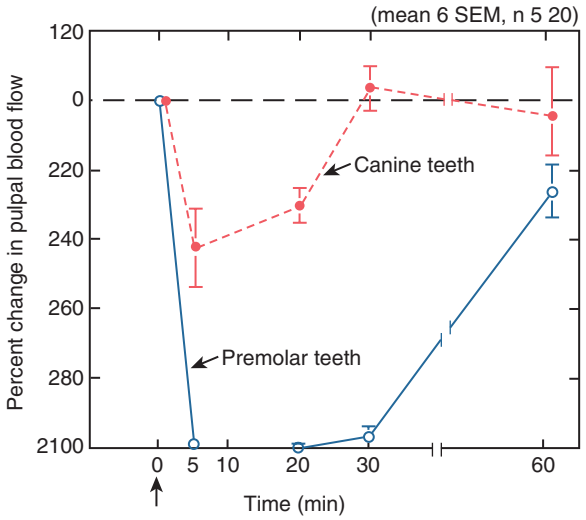

FIG. 13-13 Effects of ligamental injection (i.e., 2% lidocaine with 1:100,000 epinephrine) on pulpal blood flow in the mandibular canine and premolar teeth of dogs. Injection was given in the mesial and distal sulcus of premolar teeth. Injection caused total cessation of pulpal blood flow, which lasted about 30 minutes in the premolar teeth. Arrow indicates time of injection. (From Kim S: Ligamental injection: a physiological explanation of its efficacy, *J Endod* 12:486, 1986. Reprinted with permission.)

values after 1 hour of injection.[5] Similar reductions in pulpal blood flow were reported when inferior alveolar nerve block injections of lidocaine and 1:100,000 or 1:80,000 epinephrine were administered.[187] It is important to note a limitation of studies using laser Doppler flowmetry, which is that a large proportion of the signal measured may be from sources other than the dental pulp.[212,241] Thus, the monitoring of minor changes in pulpal blood flow must be interpreted with caution, particularly if the rubber dam or a similar barrier was not used.[99] Human studies on the effects of periodontal ligament or intraosseous injections on pulpal blood flow are not available, but from animal studies it is probable that these supplemental anesthetic techniques cause a more severe reduction or even transient cessation of pulpal blood flow.[133] It was also shown that intraosseous injection of Depo-Medrol™ (a corticosteroid) in patients with symptomatic irreversible pulpitis causes a significant reduction of prostaglandin E2 in the pulp 1 day after administration, indicating that this route of injection results in significant permeation into the pulpal tissues.[116] Taken together, these findings suggest that local anesthesia with vasoconstrictors may compromise the inflamed pulp's ability to recover from inflammation, particularly if it is severely inflamed, or if the tooth is subjected to extensive restorative procedures, and if the anesthetic is delivered via a periodontal ligament or an intraosseous route. However, it is important to realize that this hypothesis should be supported or refuted by prospective randomized clinical trials.

Intrapulpal anesthesia is often used as a last resort, when pulpal anesthesia is insufficient during root canal therapy. The effect of intrapulpal anesthesia on the pulp in these cases is not considered, as the pulp will be removed. However, occasionally a pulpotomy is performed to maintain pulpal vitality, such as in children where the tooth has an immature apex. One study has shown that intrapulpal anesthesia can be used in these cases, with no clinical differences on follow-up of over 24 weeks between the groups that did or did not receive intrapulpal anesthesia, and when given, in the groups where the anesthetic contained or did not contain epinephrine.[254]

PULPAL REACTIONS TO RESTORATIVE PROCEDURES

A large body of literature exists on the effects of restorative procedures on the dental pulp. This topic, understandably, has been important for practicing dentists for many years. Restorative procedures are performed primarily to treat an infectious disease, dental caries, which itself causes significant irritation of the pulp. They may also be performed to help restore missing teeth; correct developmental anomalies; address fractures, cracks, or failures of previous restorations; or a myriad of other abnormalities. One key requirement of a successful restorative procedure is to cause minimal additional irritation of the pulp so as not to interfere with normal pulpal healing. When pulp vitality is to be maintained during a restorative procedure, then a provisional diagnosis of reversible pulpitis rather than irreversible pulpitis must preexist. Therefore, it would be most desirable to perform a minimally traumatic restorative procedure, which would not potentially convert the diagnosis to irreversible pulpitis. As discussed previously, irreversible pulpitis may present clinically with severe spontaneous postoperative pain, but it may also be asymptomatic, leading to the asymptomatic demise of the pulp. The additive effects of restorative procedures are particularly critical in borderline cases, such as those of moderately symptomatic teeth with deep caries but no pulp exposure. There are still many factors whose influence on the response of the dental pulp to the cumulative effects of caries, microleakage, restorative procedures, and materials is not well understood. It is generally accepted that the effects of pulpal insults, be they from caries, restorative procedures, or trauma, are cumulative—that is, with each succeeding irritation, the pulp has a diminished capacity to remain vital. As a part of informed consent, the clinician is often faced with the task of outlining possible risks of restorative treatment. One study from a hospital in Hong Kong addressed the fate of pulps beneath single-unit metal-ceramic (MC) crowns or MC bridge abutments.[48] Patients who had received either treatment were invited to attend a recall appointment that involved both clinical and radiographic examinations. Researchers examined 122 teeth with preoperatively vital pulps treated with single-unit MC crowns and 77 treated as bridge abutments. The mean observation period was 14 years for the former and 15.6 years for the latter. Pulpal necrosis had occurred in 15.6% of the teeth treated with single-unit crowns, whereas 32.5% of the pulps in the bridge retainer groups had become necrotic. There was a significantly higher percentage of pulpal necrosis in anterior teeth that served as bridge abutments (54.5% of anterior abutment teeth examined). In general, however, the available evidence indicates that the effects of dental procedures on the pulp depend on the following factors.

The Degree of Inflammation of the Pulp Preoperatively

As stated previously, the dental pulp is compromised in its ability to respond to external irritants because it is enclosed in a noncompliant environment and because it lacks collateral circulation. Thus, the more severe the pulp is inflamed, the less

will be its ability to respond to further irritation, such as in the form of restorative procedures.[147]

Most research studies designed to evaluate the effects of restorative procedures (or materials) on the pulp are conducted on human or experimental animal teeth with normal pulp. Furthermore, many of the animal research projects have been performed on anesthetized animals without the use of local anesthesia, which as stated previously, reduces pulpal blood flow. Therefore, the results of these studies may not reveal the true effects of these procedures when the carious lesion already causes inflammation of the pulp, and pulpal blood flow is reduced by local anesthetic with vasoconstrictors. A study that evaluated the response of the pulp to capping procedures as a function of duration of exposure showed that the pulp responds favorably to exposures for up to 24 hours after exposure, but not as favorably after longer periods of exposure to the oral environment.[58] It may be that the longer exposure periods lead to the formation of a bacterial biofilm, which is difficult for the pulpal immune responses to eliminate, or the extension of the infection so deep into the pulp as to preclude healing. This is relevant in cases of aseptic mechanical exposures or teeth where the pulp is exposed by traumatic injuries for a brief duration. In these cases, the pulp usually responds favorably to vital pulp therapy procedures.[6] Models of standardized pulpal inflammation with chronic caries are not commonly used in determining the effects of dental procedures. Older clinical studies show an unfavorable long-term outcome of capping cases with carious pulp exposures[17,108]; however, newer studies in which mineral trioxide aggregate (MTA) was used show more favorable results in these cases.[26,166,287a]

In the absence of severe spontaneous symptoms or pulp exposure, as indicated previously, the clinician currently cannot determine accurately the degree of preoperative pulpal inflammation. Thus, every effort should be made to minimize added irritation during restorative procedures, as it is possible that excessive irritation could convert the inflammatory status of the pulp from a reversible to an irreversible condition. In addition, the patient should always be advised of the possibility of pulpal degeneration and the importance of follow-up.

The Amount of Physical Irritation Caused by the Procedure

Physical irritation during restorative procedures such as from heat, desiccation, or vibration may adversely affect the dental pulp.

Heat

Restorative procedures such as cavity or crown preparation, or curing of resins during direct fabrication of provisional restorations,[255] may cause significant increases in pulpal temperatures. It has been shown using primate models that an intrapulpal temperature rise of 10° C causes irreversible pulp pathosis in 15%, and a 20° C rise caused pulp abscess formation in 60% of teeth evaluated.[285] A number of other older studies documented burns or severe inflammation in the pulp when cavity or crown preparations were performed without coolants (Figs. 13-14 to 13-16). However, a more recent study, in which gradual controlled heat application over a large area of the intact occlusal surface of human unanesthetized teeth was employed, failed to corroborate these earlier findings.[14]

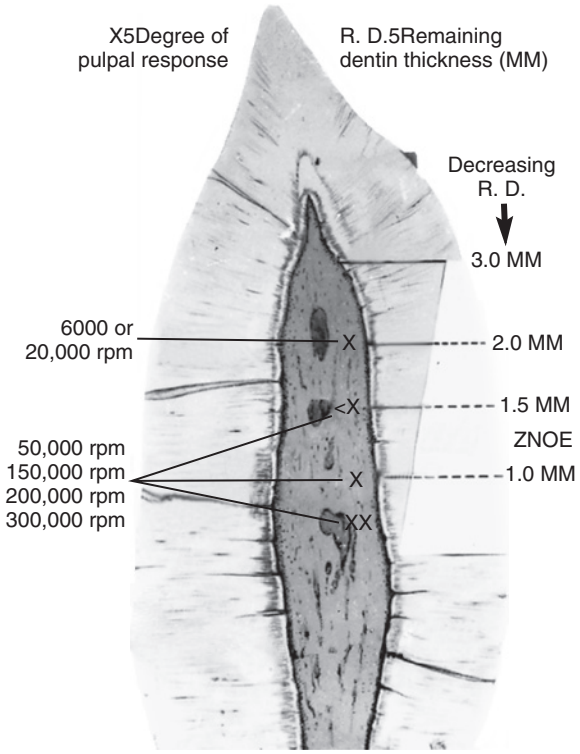

FIG. 13-14 With adequate water and air spray coolant, the same cutting tools, and a comparable remaining dentin thickness, the intensity of the pulpal response with high-speed techniques (i.e., decreasing force) is considerably less traumatic than with lower speed techniques (i.e., increasing force). (From Stanley HR, Swerdlow H: An approach to biologic variation in human pulpal studies, *J Prosthet Dent* 14:365, 1964. Reprinted with permission.)

In this study, an increase of intrapulpal temperature of about 11°C followed by a 2- to 3-month evaluation did not show any clinical or histologic changes in the pulp of any of the teeth evaluated. Heat increase in rat pulp tissue to 42° C in vitro raised heat shock protein-70, which is known to be tissue protective, and caused changes in alkaline phosphatase and gap junction proteins that were reversed to normalcy a few hours later.[8] By contrast in another study, heat applied in deep cavity preparations, prepared atraumatically in human teeth, caused histologic changes that were dependent on the proximity of the heat source to the pulp.[186] It was common in that study to see a loss of odontoblasts or their aspiration into the dentinal tubules. In cases where the cavity floor was less than 0.5 mm from the pulp, areas of coagulation necrosis could be seen, although the patients remained asymptomatic for the 1-month duration of the study. The measurement of heat in the tooth being prepared, in areas other than the site of tooth preparation, occasionally shows reduction in temperature,[91] presumably because of the poor conductive properties of dentin and the cooling effect of compressed air of the high-speed hand piece. Furthermore, cavity and crown preparations include a number of other irritating stimuli such as desiccation, severance of odontoblastic processes, vibration, and smearing of bacterial irritants onto the surface of dentin. Therefore, taken together, these findings suggest that the transient increase in temperature to levels relevant to modern dental procedures on its own may not be the culprit in inducing pulpal changes. Rather, the synergistic application of

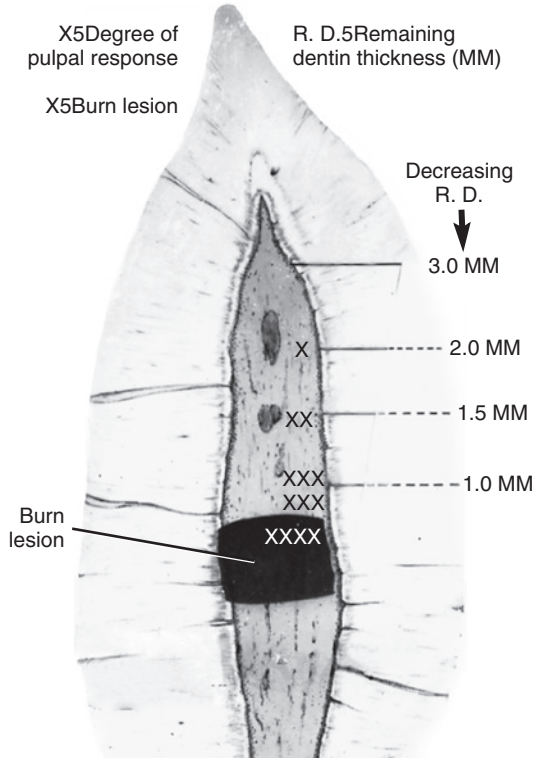

FIG. 13-15 Without adequate water coolant, larger cutting tools (e.g., no. 37 diamond point) create typical burn lesions within the pulp when the remaining dentin thickness becomes less than 1.5 mm. (From Stanley HR, Swerdlow H: An approach to biologic variation in human pulpal studies, *J Prosthet Dent* 14:365, 1964. Reprinted with permission.)

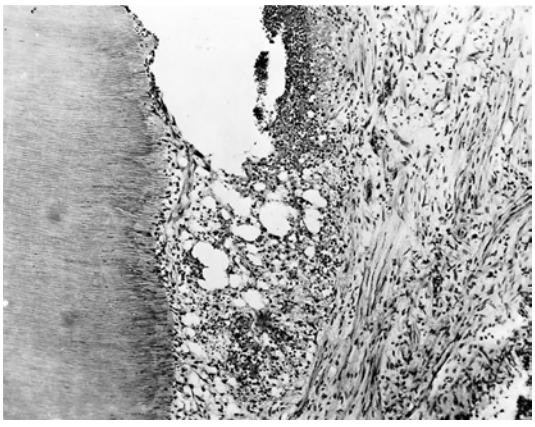

FIG. 13-16 Burn lesion with necrosis and expanding abscess formation in a 10-day specimen. Cavity preparation dry at 20,000 rpm with remaining dentin thickness is 0.23 mm. (From Swerdlow H, Stanley HR Jr: Reaction of the human dental pulp to cavity preparation. I. Effect of water spray at 20,000 rpm, *J Am Dent Assoc* 56:317, 1958. Reprinted with permission.)

excessive heat with other irritation factors and its proximity to the pulp may induce pathologic changes.

Desiccation

Desiccation during cavity and crown preparation has long been known to cause aspiration of odontoblastic nuclei into dentinal tubules and pulpal inflammation.[31] One study showed that as little as 30 seconds of continuous air drying of class V cavities in human molars with uninflamed pulp caused significant displacement of odontoblastic nuclei, pulp inflammation, and even areas of necrosis related to the areas that were dried.[57] However, another study showed that the effects of desiccation are transient in that within 7 to 30 days there is autolysis of the aspirated cells and formation of reactionary dentin.[32] The pulp in cases with aspirated odontoblasts, following desiccation for 1 minute, was not sensitive to clinical scraping with an explorer. The sensitivity was restored with rehydration of the cavities and was increased in other cases where pulp inflammation was induced by microbial contamination.[154] In this study, despite the lack of sensitivity in desiccated cavities, neural elements were seen histologically to be pushed into the tubules like the odontoblastic nuclei. The disruption of the odontoblastic layer and peripheral neural elements in the pulp with desiccation was also observed in a rat model using axonal transport of radioactive protein.[37]

Biologic and Chemical irritation

Dental caries is clearly an infectious disease in which microorganisms and their virulence determinants constantly irritate the pulp, even at the early stages, long before pulp exposure.[34] However, despite the elimination of visible caries during cavity preparation, the cavity floor is undoubtedly left with some contamination by caries bacteria. Although the rubber dam should be used with any cavity preparation to prevent cavity contamination with salivary microorganisms, the use of water coolants allows the cavity to be contaminated with bacteria from water lines. Concerns about residual cavity contamination prompted some to use cavity disinfection with caustic chemicals. Chemicals such as hydrogen peroxide, sodium hypochlorite, or calcium hydroxide solutions have been proposed for this purpose, although they may exert a toxic effect.[54] An earlier study showed that the amounts of residual bacteria following adequate restoration are not significant.[170] Once dentin is exposed, there is a constant outward flow of dentinal fluid that minimizes the inward flow of any noxious agents.[275] This may aid in the reduction of irritation from residual microbial factors in dentinal tubules.

In contemporary practice, most chemical irritation during restorative procedures results from the application of etching agents, especially strong acids, in the form of total dentin etch, particularly if capping of exposed pulp is performed.[93,197] Etching is performed to remove the smear layer, promote physical adhesion of bonding agents to dentin by forming resin tags in the dentinal tubules, and permeate the newer unfilled resin primers into the unmineralized surface layer of collagen to form the so-called hybrid layer.

If the cavity is relatively superficial and is adequately sealed with a restorative resin, then etching of dentin is probably not detrimental to the pulp because of the narrow diameter of dentinal tubules and their low density in peripheral dentin.[28] In fact, one study documented that histologic evidence of bacteria in human cavities restored with composite was significantly less if the cavity had been etched with phosphoric acid than if it were etched with 17% EDTA or nonetched.[176] Pulpal inflammation in this study was not correlated with the etching treatments but with bacterial presence; thus, in cases of etching with phosphoric acid, if bacteria were also present, severe pulpal inflammation and necrosis could be seen.

Self-etching formulations have become popular because they eliminate the separate etching step involved in total-etch

procedures. Some have speculated that the bonding of self-etching systems may be poorer than total-etch systems because of the weaker acidity of the acidic primers of self-etching systems when compared to that of total-etch systems.[28] However, studies have shown no significant differences between the two adhesive systems in postoperative sensitivity,[208] long-term in vivo degradation,[141] or long-term in vitro bond strength.[10] One clinical study showed no differences between the two systems with respect to bacterial leakage and the inflammatory response in the pulp.[181] The most important variable that affected the pulp in this study was the amount of bacterial leakage with either system.

Other factors that may contribute to pulpal irritation during resin placement from chemical/biologic irritants include unpolymerized monomer and polymerization shrinkage. Higher concentrations of monomeric resin components were shown to exert an inhibitory effect on T lymphocytes and spleen cells,[124] and monocytes/macrophages[149,216,217,231] in vitro. These components may leach directly into the pulp in deep cavities and cause chemical irritation.[59,110] Shrinkage during polymerization of composites may induce internal stresses on dentin and create voids that allow microleakage. Shrinkage of resins is estimated to range from 0.6% to 1.4%, and should be minimized during placement by incremental curing and possibly starting the restoration with flowable resins.[28]

In summary, the available evidence indicates that chemicals involved in modern restorative procedures may irritate the pulp if placed directly on an exposure, or if there is microbial leakage along the tooth/restoration interface.

The Proximity of the Restorative Procedures to the Dental Pulp and the Surface Area of Dentin Exposed

It has been known for several decades that as the carious lesion progresses toward the pulp, particularly when the remaining dentin thickness (RDT) is less than 0.5 mm, there is an increasingly severe pulpal reaction, with a greater likelihood of the pulp undergoing irreversible pathosis.[220] The diameter and density of dentinal tubules increase closer to the pulp (Fig. 13-17). Based on the dentinal tubule density at the DEJ (about 65,000/mm^2) and the pulp (about 15,000/mm^2),[84,88] it was estimated that the area occupied by tubule lumina at the DEJ was 1% of the total surface area at the DEJ and 22% at the pulp.[203] Thus, it is not surprising that several studies have shown that pulpal inflammation in response to restorative procedures increases with the reduction in RDT.[159,175] One study examined the differential effects on the rat pulp of the preparation method, remaining dentin thickness, coolant, drill speed, conditioning with EDTA, and filling materials.[174] Subsequent to the cavity preparations a tooth slice was obtained and maintained ex vivo as an organ culture for up to 2 weeks. The results showed that the remaining dentin thickness was the most important factor in pulpal injury.

With the passage of time following cavity preparation, there is reduction in the permeability of RDT.[204] This may be due to rapid deposition of reactionary dentin, the migration of large proteins into the tubules, or the diminution of tubule diameter as dentin becomes more sclerotic. Using a primate model, it was shown that the basic rate of secondary dentin deposition was about 0.8 µm/day and that this rate increased to an average of 2.9 µm/day following restorative procedures. Interestingly, in this study dentin deposition was also more rapid next to shallow cavities than deep cavities[277]; however, another study showed that total reactionary dentin deposited was thicker in deeper and wider cavities.[173]

Clinically, it has been observed that postoperative sensitivity is common with many restorative procedures. Following resin composite restorations on patients, it was shown that postoperative sensitivity was related to the depth of the cavity, but not to the presence or absence of liners or bases.[268]

In addition to the depth or the width of a large cavity preparation, a crown preparation exposes more dentinal tubules to microbial or chemical irritation. During crown fabrication, there are added irritation factors such as length of time of the preparation, impression techniques, and the imperfect adaptation of temporary restorations, causing microleakage during the temporization period. Because of the precise engineering requirements of some restorations, some providers may be inclined to reduce the coolant during crown preparation steps such as finalizing the finishing lines. However, crown preparations without coolants have been shown to dramatically reduce pulpal blood flow in an animal model (Fig. 13-18).[133] There are few studies available on the direct effects of modern crown and bridge techniques on the

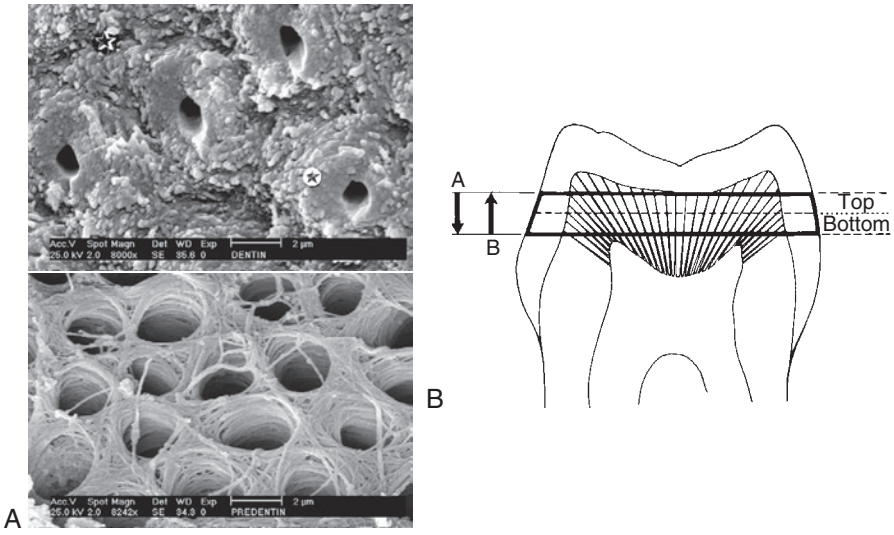

FIG. 13-17 Schematic of convergence of tubules toward the pulp. **A**, Periphery of the dentin. Most surface area is occupied by intertubular dentin (☆), with a few tubules surrounded by hypermineralized peritubular dentin (✪). **B**, Near the pulp, the increase in tubule diameter has occurred largely at the expense of the peritubular dentin. This substrate has high protein content. As the remaining dentin is made thinner (from **A** to **B**), the permeability of the dentin increases, because both the diameter and the density of dentinal tubules are increased. (From Bouillaguet S: Biological risks of resin-based materials to the dentin-pulp complex, *Crit Rev Oral Biol Med* 15:47, 2004. Reprinted with permission.)

pulp. However, some long-term outcome studies have documented that the incidence of pulp necrosis following crown placement ranges from 10% to 50%.[48,79,269]

The Permeability of Dentin and the Odontoblastic Layer Between the Area Being Restored and the Pulp

The permeability of dentin plays an important role in the ingress of potential irritants to the pulp. It is clear from research done since the 1980s that dentin is not uniformly permeable and that permeability depends on factors such as the location within the same tooth, the age of the patient, and the presence of pathologic conditions such as dental caries. Fundamentally, the permeability of dentin depends on the collective sum of the permeability of individual tubules at a particular site in the tooth. The tubular diameter increases from about 0.6 to 0.8 μm close to the DEJ to about 3 μm at the pulp.[200] Given that bacterial cells are about 0.5 to 1 μm in diameter, it is evident that in deep cavity preparations, particularly when total-etch procedures are employed, bacteria can migrate through the remaining dentin into the pulp.

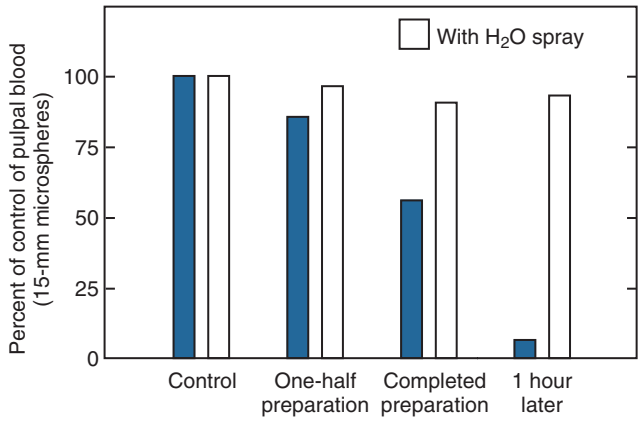

FIG. 13-18 Effects of crown preparation in dogs, with and without water and air spray (at 350,000 rpm), on pulpal blood flow. Tooth preparation without water and air spray caused a substantial decrease in pulpal blood flow, whereas that with water and air spray caused insignificant changes in the flow.

With age the width of peritubular dentin increases, causing a reduction in tubular lumen or sclerosis. Caries causes demineralization in superficial dentin, which is associated with remineralization and the formation of caries crystals within the tubules of inner undemineralized dentin (Fig. 13-19). This causes a decrease in permeability in dentin subjacent to the carious lesion[202] and could be considered a protective mechanism, as it may delay the progress of the carious lesion.

It was shown that irritation from cavity preparation increased the permeability of the odontoblastic cell layer only at the site of the cavity preparation.[117,263] In addition to the physical barrier to permeability and the production of reactionary or reparative dentin, the odontoblastic layer may, in fact, contribute to the host response of the dental pulp by expressing important inflammatory mediators[152,196] or recognizing bacteria through Toll-like receptors.[22,66,74,123]

The Age of the Patient (see also Chapter 26)

Resting pulpal blood flow (PBF), as well as the changes in PBF in response to cold application, will decrease with age.[112] Age

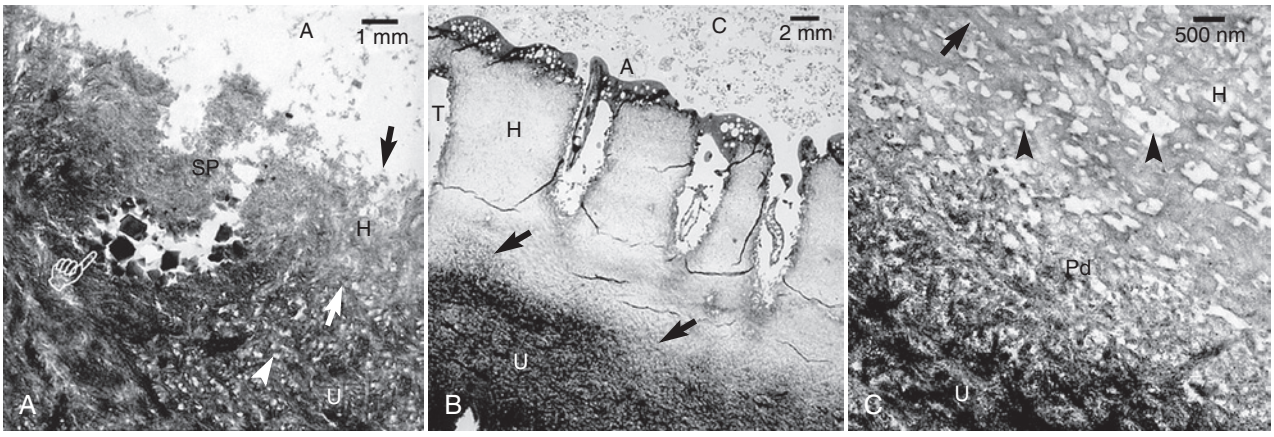

FIG. 13-19 TEM of undemineralized specimens of resin-bonded, caries-affected dentin. **A,** Stained TEM of undemineralized specimens following the application of the self-etch ABF system to caries-affected dentin. The hybrid layer *(H; between arrows)* was about 3 μm thick, and the underlying undemineralized dentin *(U)* was highly porous *(arrowhead)*. The dentinal tubule was covered with a smear plug *(SP)* and was partially obliterated with large caries crystals *(pointer)*. *A,* filled adhesive. **B,** Stained section of the total-etch single bond adhesive bonded to caries-affected dentin. A hybrid layer *(H)* between 15 and 19 μm thick could be seen, with a partially demineralized zone *(open arrows)* above the undemineralized caries affected dentin *(U)*. *T,* dentinal tubule. C, composite. **C,** Higher magnification of the basal part of the unusually thick hybrid layer (H), shown in **(A)**. Banded collagen fibrils *(open arrow)* were separated by unusually wide and porous interfibrillar spaces *(open arrowheads)*. A partially demineralized zone *(Pd)* was present along the demineralization front. This zone was not seen in phosphoric-acid-etched sound dentin **(B)**. *U,* undemineralized caries-affected dentin. (From Yoshiyama M, Tay FR, Doi J, et al: Bonding of self-etch and total-etch adhesives to carious dentin, *J Dent Res* 81:556, 2002. Reprinted with permission.)

may also be associated with reduction in pulpal neuropeptides.[85] However, studies show no differences between young and old pulp in the regenerative capacity of odontoblast-like cells and in the presence of cells positive for class II major histocompatibility complex, heat shock protein 25, or nestin, when subjected to cavity preparation.[128] An examination of young versus old normal human pulp showed that young pulp had increased expression of biologic factors related to cell differentiation, proliferation, and the immune response, whereas older pulp had increased factors related to apoptosis.[259] These analyses cannot translate to the pulp's ability to deal with irritation and its sequelae. Thus, the net result of the ability of the pulp to cope with external stimulation or irritation in humans with advancing age is not clear.

PULPAL REACTIONS TO RESTORATIVE MATERIALS

The effects of restorative materials on the dental pulp have been investigated and seem to relate directly to the permeability of the associated dentin. The degree of dentin permeability, however, is often variable and is governed by several factors including age and caries status.[251] The most important variable in dentin permeability to restorative materials is the thickness of dentin between the floor of the cavity preparation and the pulp.[1]

Given the importance of dentin permeability, there are direct pulpal effects of any given restorative material that are governed by the composition of the material and associated eluted or degraded products. Unbound components of resin materials and preparative agents such as acid etchants can affect the subjacent pulp by inducing an inflammatory response.[87,93,215] The indirect effects of desiccation or demineralization of dentin as well as the direct effects of the material itself when in contact with pulpal tissue mediate this inflammatory response. Studies have shown that the certain cytotoxic components of resin monomers (e.g., triethylene glycol dimethacrylate and 2-hydroxyethyl methacrylate) readily penetrate dentin.[81] Similarly, eugenol and components of Ledermix (triamcinolone and demeclocycline) have been shown to pass through dentin into the subjacent pulp.[109,111] In vivo data show that these chemicals have an effect on the pulp; however, the effect seems to be short lived and, in the absence of bacteria, it is reversible.[21]

The mechanisms whereby restorative materials exert an injurious effect on the dental pulp vary. Evidence exists that supports direct and, in some instances, prolonged cytotoxicity, stimulation of hypersensitivity reactions, or impairment of the host immune response to bacteria.[229] Some of the components of resin restorations are released at cytotoxic levels after polymerization is completed, leading to chronic stimulation and a resultant prolonged inflammatory response.[80] Furthermore, even subtoxic concentrations of certain agents are capable of eliciting allergic reactions in humans.[110] Primates hyperimmunized with BSA showed significant pulpal damage with repeated antigenic challenge in class V cavity preparations, suggesting a role for antigen-antibody complex–mediated hypersensitivity in tissue destruction.[24] In a separate study, exposure to dentin primers elicited a delayed-type hypersensitivity reaction in guinea pigs.[127] These studies taken together present a compelling argument for immune-mediated pulpal tissue damage subsequent to exposure to restorative materials. Foreign body reactions have also been described in pulps containing extruded globules of resin material.[114,115] Histologic examination of these pulps shows macrophages and giant cells surrounding the resin particles. Lastly, resin monomers have been shown to decrease the activity of immunocompetent cells in a dose-dependent manner in in vitro functional assays.[124] Although all of these effects are documented, their extent and therefore morbidity on the dental pulp is speculative and doubtless does not act solely to effect pulpal demise. As previously noted, most restorative materials are placed adjacent to pulps that are previously compromised by bacterial insult, and disease, debridement, and restoration of the tooth have cumulative effects on the dental pulp.

Although pulpal irritation is largely considered to be a negative sequela, the irritant potential of certain restorative materials is central to their usefulness in restorative dentistry. Calcium hydroxide is one of the oldest and most widely used medicaments for stimulation of dentinal bridge formation subsequent to microscopic or gross pulpal exposure. The low-grade pulpal irritation that it induces is important for dentinal bridge formation in exposures.[61,230] The degree of inflammation is dependent on the preparation of calcium hydroxide used. Aqueous suspensions of calcium hydroxide applied to exposed pulps cause superficial necrosis of pulpal tissue followed by low-grade inflammatory changes. Within 30 days the tissue subjacent to the necrotic zone has reorganized and resumed normal architecture. Hard-setting calcium hydroxide preparations as well as mineral trioxide aggregates are effective in eliciting dentinal bridge formation with a much smaller to nonexistent necrotic zone.[102] This is preferable in vital pulp therapies such as the Cvek pulpotomy, where maintenance of the maximum amount of vital pulp tissue is desirable and the extent of pulpal inflammation is minimal[60] (Fig. 13-20). The irritation potential of calcium hydroxide across intact dentin is dependent on factors such as the remaining dentin thickness and permeability. Application of calcium hydroxide to intact dentin appears to induce sclerosis by promoting crystal precipitation within the tubules accompanied by reductions in permeability.[172]

Although the irritation potential of calcium hydroxide plays a role in its effectiveness, the high pH of this material can liberate bioactive molecules from dentin. Numerous authors have demonstrated that dentin matrix proteins like TGF-β1 and adrenomedullin are liberated by both calcium hydroxide and mineral trioxide aggregate.[92,257] Once liberated they are able to facilitate hard tissue formation yet again. This offers another explanation for the ability of these materials to induce hard tissue formation in vivo. Acid etching or the use of chelating agents such as EDTA represent a common step in the placement of bonded restorations. The etching process removes mineral and exposes collagen fibrils. As with a high pH, sequestered bioactive molecules are released and can act on pulpal cells. These include growth factors and metalloproteinases, which can be inductive for healing but can also degrade the bond of the restoration.[205] EDTA has been shown to be even more effective at extracting noncollagenous proteins from dentin than either calcium hydroxide or MTA but less so than citric acid.[92,257]

Investigations into the pulpal response to glass-ionomer materials have shown that both the lowered pH of the material and the release of high concentrations of fluoride can inflict damage to pulp tissues.[126] In direct contact with pulpal cells, glass ionomers are toxic.[143] Direct pulp capping studies have

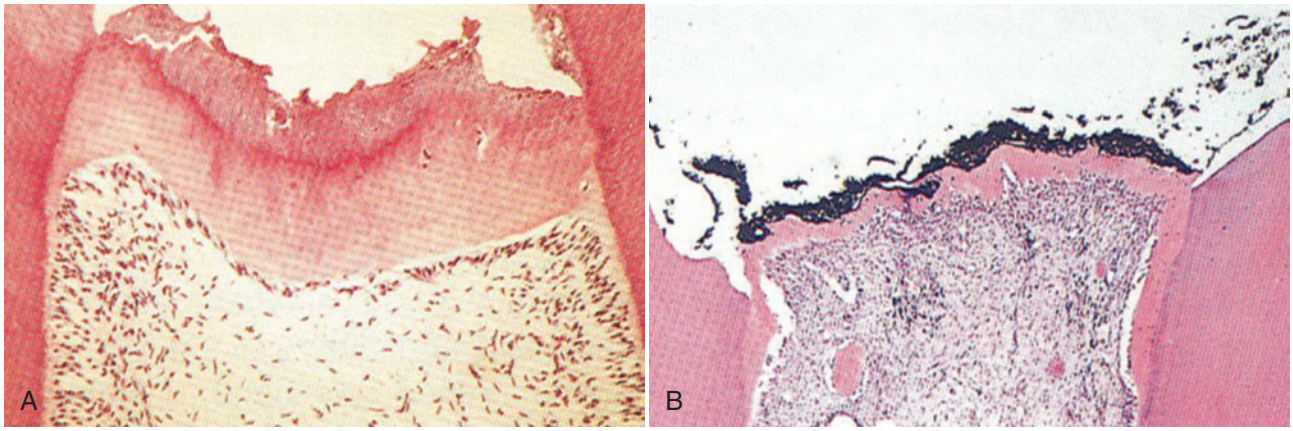

FIG. 13-20 Calcium hydroxide produces an inflammatory response that stimulates dentinal bridge formation. The dentin bridge forms lower in the tooth with calcium hydroxide paste (A) versus hard-setting calcium hydroxide (B).

shown it to be inferior to calcium hydroxide.[63,105] When applied to intact dentin and in the absence of bacteria, there is a transient inflammatory response and reparative dentin is formed.[55]

In addition to the direct chemical effects of restorative materials, there are indirect factors that contribute to pulpal irritation. The technique sensitivity of certain materials predisposes them to faulty bonds to tooth structure that can translate to dentin hypersensitivity, recurrent disease, and pulpal inflammation or necrosis. Much attention has been given to the interface created between resin-bonded materials and dentin. During the etching process, the more highly mineralized peritubular dentin is preferentially dissolved, leaving free collagen fibrils and opening lateral tubular branches.[93,94,171] Applied resin infiltrates the exposed collagen mesh, creating a layer 5 to 10 μm thick referred to as the hybrid layer.[178] This layer, along with the resin permeating exposed tubules, forms the bond between the resin and dentin. If the preparation is too dry, the collagen fibrils collapse and the resin cannot effectively permeate the mesh, which results in a defective bond. As the optimal degree of hydration of the preparation surface can vary from material to material, resin restoration placement is technique sensitive. This same principle is applicable to the practice of bonding fractured tooth fragments where the segment has become dehydrated while outside of the mouth. Current protocols recommend rehydration of the segment prior to bonding, thus increasing the mechanical and presumably the microbial seal.[78] This is particularly important with a complicated crown fracture where the pulpal protection by intact dentin is absent.

Some restorative materials rely on their medicinal properties as well as their ability to seal a cavity preparation. Materials containing zinc oxide and eugenol (ZOE) fall into this category. ZOE is used for a variety of purposes in dentistry largely because of its anesthetic and antiseptic properties. It has been shown to block transmission of action potentials in nerve fibers and to suppress nerve excitability in the pulp when applied to deep excavations.[261] In addition, ZOE has good adaptation to dentin and inhibits bacterial growth on cavity walls. These properties have made this a favored material for temporary fillings but not for long-term restorations, as ZOE temporaries have been shown to leak after only a few weeks in situ.[286]

DIRECT PULP CAPPING WITH MINERAL TRIOXIDE AGGREGATE

Direct capping of pulp exposures is indicated in pulps that were previously healthy and exposed by trauma or dental restorative procedures.[250] Although calcium hydroxide has historically been the preferred dressing agent on mechanically exposed pulps, the use of mineral trioxide aggregate (MTA) has been proposed, even on carious pulp exposures.[16,26,166] Prospective animal studies and human case reports have evaluated the ability of MTA to allow for the formation of a reparative dentin bridge and maintain continued pulp vitality.[73,83,137] Although the results are generally favorable, one concern is of tooth discoloration if the gray MTA formulation is used on anterior teeth.

In one clinical study, MTA was used as a pulp capping material for carious pulp exposures.[26] Forty patients, ages 7 to 45, who were diagnosed with reversible pulpitis had caries removed using a caries detection dye and sodium hypochlorite for hemostasis. The treatment was performed in two visits to allow the MTA to set up and to confirm pulp sensibility to pulp tests in the second visit. Success was determined radiographically, with subjective symptoms, pulp testing with cold, and continued root formation on immature teeth. Outcomes were measured over a period of up to 9 years postoperatively and showed an overall success of 97%, with all the teeth in the immature root group showing success. In another clinical study in which dentists and students participated in the treatment, pulp capping of carious pulp exposures was compared between MTA and calcium hydroxide.[166] At follow-up, 122 cases of patients with a mean age of 40 years were available. The success with MTA was 78% and that with calcium hydroxide was 60%, a difference that was statistically significant. More recently, the same group reported an update of their findings with a sample size of 229 teeth, which were evaluated from 24 to 123 months following treatment. The overall success rates were 80.5% of teeth in the MTA group (137/170) and 59% of teeth in the CH group (35/59), which was a statistically significant difference. The outcome was also significantly better in any group if the tooth was promptly restored. Within the parameters of these studies, it seems that white MTA is a suitable capping agent for pulp exposures of healthy or

reversibly inflamed pulps, including cases with asymptomatic carious exposures. However, given that the prognosis of root canal therapy in this last category has been shown to be predictably high in many studies, the patient needs to be educated carefully of the options, and consent must be documented.

Other calcium silicate–based materials are gaining popularity as pulp capping agents and appear to have properties and effects similar to MTA.[11,184] Biodentine deserves special reference because in addition to the biocompatibility, it has physical properties that are similar to dentin.[39,142]

THE USE OF HEMOSTATIC AGENTS AND DISINFECTANTS ON DIRECT PULP EXPOSURES

There is still debate as to whether the outcome of pulp capping for exposure depends on the toxicity of medicaments and materials placed on vital pulp tissues, the ability of these materials to induce mineralization, or their ability to seal the cavity from further bacterial ingress. It is likely a combination of these factors, as was shown in the clinical trial with MTA mentioned before.[26] Another factor in the prognosis of direct pulp caps is the ability to control hemorrhage at the exposure site.[243] Given the difficulty in creating a bacteria-free operating environment during tooth preparation, the ideal hemostatic agent also would have the ability to kill bacteria.

One study compared the effects of two hemostatic/disinfectant agents on the healing of experimental pulp exposures created in human third molar teeth and capped with calcium hydroxide.[234] Pulp exposures were made in 45 maxillary wisdom teeth scheduled for extraction for orthodontic reasons. Teeth were randomly assigned to receive hard-setting calcium hydroxide pulp caps after a 30-second surface treatment with one of three agents: 0.9% saline, 2% chlorhexidine, or 5.25% sodium hypochlorite. Although the 7-day saline specimens showed slightly less inflammatory response, there were no statistically significant differences between the groups with respect to all dependent measures over the course of the study. Complete healing was seen in 88% of all specimens at 90 days.

The pulps in these teeth were previously uninjured, and the exposures were made in a clean environment. Therefore, it seems that a *mechanical* exposure of a healthy pulp created during cavity preparation could be disinfected with either 2% chlorhexidine or 5.25% sodium hypochlorite, capped with a hard-setting calcium hydroxide formulation, and expected to have a favorable prognosis for healing.

PULPAL REACTIONS TO LASER PROCEDURES

Numerous studies have been published on the effect of using lasers on enamel, dentin, and pulp (see also Chapter 8). Laser use on hard tissue has been a popular area of research because of the potential benefits of efficiency, reduced sensitivity, disinfection, and precision. Several types of laser technologies are available that depend on the wavelength, active medium, emission mode, delivery system, power output, and duration of application. The main types available in dentistry today are shown in Fig. 13-21. The *CO_2 laser* is historically the oldest type used on soft tissues and thus has been the most studied.

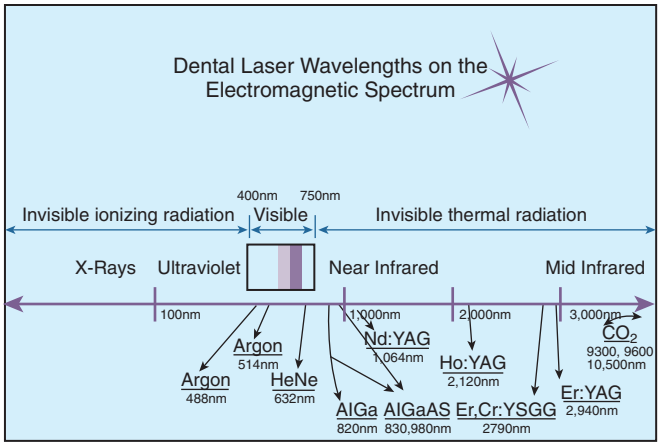

FIG. 13-21 Currently available dental wavelengths on the electromagnetic spectrum. Note all of the wavelengths are nonionizing. (From Coluzzi DJ: An overview of laser wavelengths used in dentistry, *Dent Clin North Am* 44:753, 2000. Reprinted with permission.)

It has the longest wavelength (10,600 nm). It cannot be delivered in an optic fiber, thus it must be used in a hollow-tube-like wave guide in continuous gated-pulse mode. This means that the operator does not feel a solid resistance when using this laser. *Er:YAG, Nd:YAG or Ho:YAG lasers* all have an active medium of a solid crystal of yttrium-aluminum-garnet, which is impregnated in erbium, neodymium, or holmium, respectively. Er:YAG has a wavelength of 2940 nm and is delivered using a solid optic fiber. It has a high affinity for water and hydroxyapatite, thus it can be used to remove caries and cut dentin with coolant. It can also be used on soft tissue. Ho:YAG laser has a wavelength of 2120 nm and has high affinity to water but not to tooth structure, thus it is used primarily for soft tissue surgery. Nd:YAG laser is also delivered fiberoptically, has a wavelength of 1064 nm, and has been used extensively in dentistry because it has a high affinity for water and pigmented tissues and offers good hemostasis; thus is used extensively in surgery.[52] In addition, there are some low-power output lasers such as *HeNe* (helium-neon) (632 nm), and *GaAlAs* (gallium-aluminum-arsenide) (diode; semiconductor) (720-904 nm) lasers, which have been used in laser Doppler flowmetry and in treating dentin hypersensitivity.[134]

To summarize the available data, a discussion of the application of lasers for two specific purposes follows.

Lasers in the Prevention, Diagnosis, and Treatment of Caries

Laser irradiation of deep susceptible pits and fissures may reduce the incidence of dental caries. Once caries develops, some lasers may be effective in removing the carious lesion and sparing undemineralized dentin, because of their differential absorption by water and hydroxyapatite. Furthermore, if caries exposes the pulp in young teeth, particularly those with immature apices, lasers may be able to effectively excise coronal infected pulp in pulpotomy, because of their hemostatic and antibacterial properties. All these potential uses prompted a large number of investigations on the effectiveness of lasers in these applications.

Some clinicians have proposed using lasers to enhance adhesion of pit and fissure sealants; however, this was shown to not

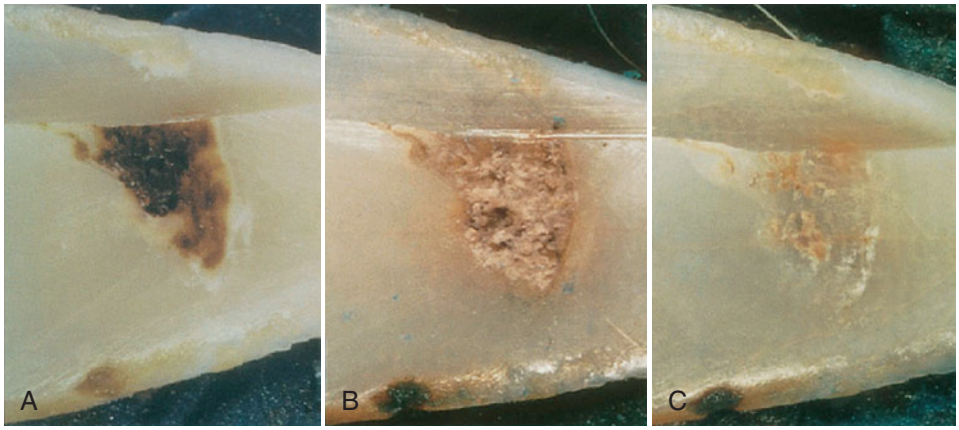

FIG. 13-22 A, Dental caries. B, Pulsed Nd:YAG ablation by-products (160 mJ, 10 Hz). C, Debris removed with acid etch and polishing. Enamel surface was faceted for reflection spectroscopy. (From Harris DM, White JM, Goodis H, et al: Selective ablation of surface enamel caries with a pulsed Nd:YAG dental laser, *Lasers Surg Med* 30:342, 2002. Reprinted with permission.)

add any advantage following acid etching—a necessity for adhesion.[160] Laser fluorescence is used by the DIAGNOdent and DIAGNOdent pen, which are laser devices that have been introduced for the diagnosis of noncavitated caries. Although these devices initially showed some promising results,[161] more recent work shows that they are best used as adjunctive devices to radiography and visual examination.[7,53,144]

Laser ablation of superficial carious lesions may be more conservative than bur preparation. A recent controlled clinical trial supports this tenet, when a free-running pulsed Nd:YAG laser was used to ablate superficial pit and fissure caries in third molars scheduled for extraction[98] (Fig. 13-22). In this study there were no histologic differences in the pulp response between the two groups.

From the perspective of the effects on the pulp, most laser applications that are employed in cutting or modifying cavities in dentin or acting directly on the pulp tissue are important. Earlier studies showed reduced permeability of dentin in vitro with a XeCl excimer laser (a laser with a relatively short wavelength of 308 nm in the ultraviolet range).[242] The apparent fusion of tubules in superficial layers of dentin was shown to occur with CO_2, Nd:YAG, and Er:YAG lasers in vitro.[278] The pulpal responses to Nd:YAG and CO_2 lasers were not favorable. It was shown that Nd:YAG and to a lesser degree CO_2 lasers may be associated with charring and significant inflammation in the pulp compared with the Er:YAG laser[256,278] (Fig. 13-23). More recently it was reported that water cooling was necessary for laser ablation as it is for high-speed bur preparations.[43] Studies have shown that the Er:YAG laser appears to induce similar responses in the pulp to those seen with high-speed bur preparations, at the level of light microscopy analysis.[72,252,253,278] However, the findings with electron microscopy were different: it was reported that while shallow cavities ablated in rat molars using Er:YAG lasers did not show changes from base-line using light microscopy, transmission electron microscopy showed disruption and degeneration of pulpal peripheral nerve endings and of myelin sheath in the immediate postoperative period (Fig. 13-24).[113] This may explain the reduced sensitivity that accompanies laser cavity preparations. Thus, in summary, it does not appear that the use of lasers provides predictable advantages

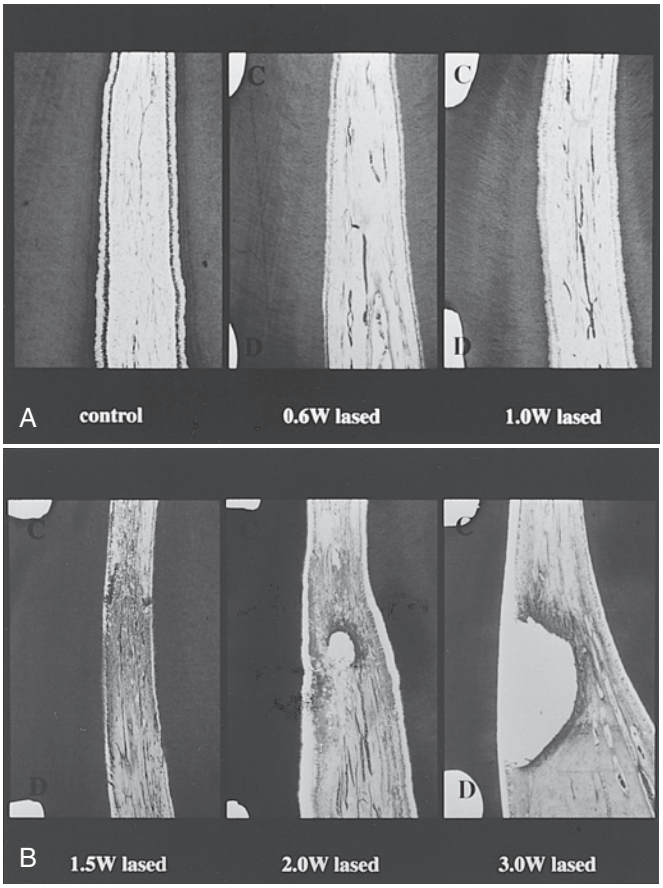

FIG. 13-23 A, B, Histopathologic picture of Nd:YAG laser specimen with an increasing power. There is direct relationship between the degree of pathologic changes and the increasing power of the laser. In fact, 1.5W and the greater power cause permanent damage to the pulp.

in cavity preparation compared with traditional methods at this time.

For pulpotomy procedures, such as in primary teeth, permanent teeth with immature apices, or pulps that are exposed due to fracture and treated promptly, the lasers, particularly

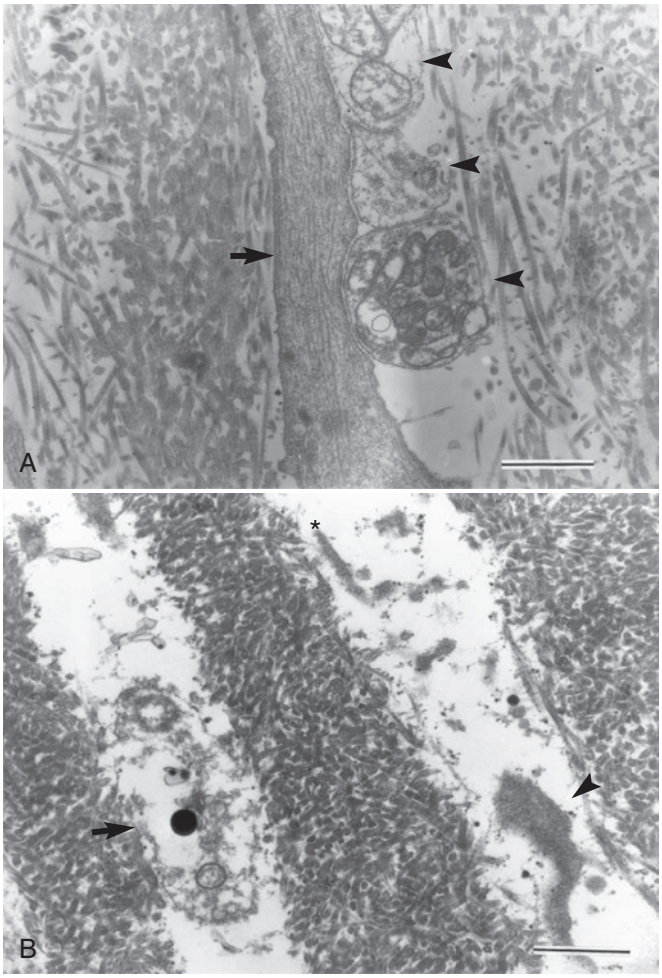

FIG. 13-24 A, Control; normal odontoblastic process *(arrow)* and a few nerve terminals *(arrowheads)* are seen in a dental tubule of rat upper first molar. B, Six hours after Er:YAG laser irradiation; disrupted cell membrane of a nerve terminal that contains some granular vesicles *(arrow)* and shrinkage of an odontoblastic process *(arrowhead)* are noted in dentinal tubules just under the ablated area. An asterisk indicates the irradiated side (TEM, ×13,700, bar = 1 μm). (From Inoue H, Izumi T, Ishikawa H, Watanabe K: Short-term histomorphological effects of Er:YAG laser irradiation to rat coronal dentin-pulp complex, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:246, 2004. Reprinted with permission.)

CO_2 lasers, may be useful in achieving precise surgical excision of coronal pulp and immediate hemostasis. A controlled clinical study showed that CO_2 laser pulpotomy was comparable to traditional methods in experimental pulpotomies of primary teeth scheduled for extraction for orthodontic reasons.[70] However, an animal study in which CO_2 and Nd:YAG lasers were compared revealed poor response with both lasers compared with calcium hydroxide.[125] An animal study using the Er:YAG laser showed that the results are dependent on the power settings, in that lower energy delivered with this laser produced favorable results.[135]

Lasers in the Treatment of Dentin Hypersensitivity

Earlier studies have shown effectiveness ranging from 5% to 100% of low-output lasers on dentin hypersensitivity.[134] One author reported a reduction of hypersensitivity in 73% of mild cases, 19% of moderate cases, and 14% of severe cases after 4 months using a GaAlAs laser.[134] Low-output lasers do not have any effects on the morphology of enamel or dentin, but they are thought to cause a transient reduction in action potential mediated by pulpal C-fibers, but not Aδ-fibers,[276] although this finding was not consistent.[192] Nd:YAG lasers have also been used in dentin hypersensitivity. Because of the higher power output, these lasers cause superficial occlusion of dentinal tubules of up to 4 μm,[158] in addition to action potential blockage within the pulp in vitro or in experimental animals.[191,192] However, a placebo-controlled clinical trial has shown that both Nd:YAG and placebo caused a significant reduction in dentin hypersensitivity for up to 4 months postoperatively but were not different from each other.[153] More recent clinical trials show that although lasers are useful for managing hypersensitivity, they do not appear to be better than less costly and more readily available alternatives.[82,267]

Use of Lasers as a Protective Measure for Dentin, Under Traditional Cavity Preparation

One clinical study has proposed that if lasers are used to prepare cavities or used on prepared dentin after traditional cavity preparation with burs, this would protect the dentin, as its permeability and bacterial content may decrease. In this study, two patients were scheduled to have six teeth extracted during orthodontic treatment.[90] In the teeth that had laser irradiation (GaA1As laser, lambda = 660 nm, power of 30 mW and energy dose of 2 J/cm^2) and were examined with transmission electron microscopy (TEM) after 28 days, the odontoblastic process had increased contact with the extracellular matrix, and the collagen fibrils appeared more organized than those of the control group (traditional bur preparation only). The study concluded that laser irradiation accelerates the recovery of the dental tissues in the predentin region.

PULPAL REACTIONS TO VITAL BLEACHING TECHNIQUES

Vital bleaching techniques employ strong oxidizing agents, namely 10% carbamide peroxide and hydrogen peroxide, to bleach enamel of teeth with vital pulp. There have been concerns about the potential for pulpal irritation during these procedures because of the long duration that the chemicals are in contact with the teeth, particularly if dentin with open tubules or cracks is present. Histologic or histochemical analysis of the pulp following bleaching for up to 2 weeks showed minor inflammatory changes in the pulp of the bleached teeth that were reversible.[9,86] One clinical report documented that if 16% carbamide peroxide was used, gingival irritation was evident; however, no changes in pulp vitality or in symptoms were noted. Even in patients who develop symptoms postoperatively, these tend to be reversible and could be prevented by treating the teeth with fluorides and by correcting restorative deficiencies preoperatively.[180] Clinical symptoms are likely to be due to increases in neuropeptides, such as substance P, in the pulp.[44] An earlier clinical trial showed that vital bleaching using 10% carbamide peroxide in a custom

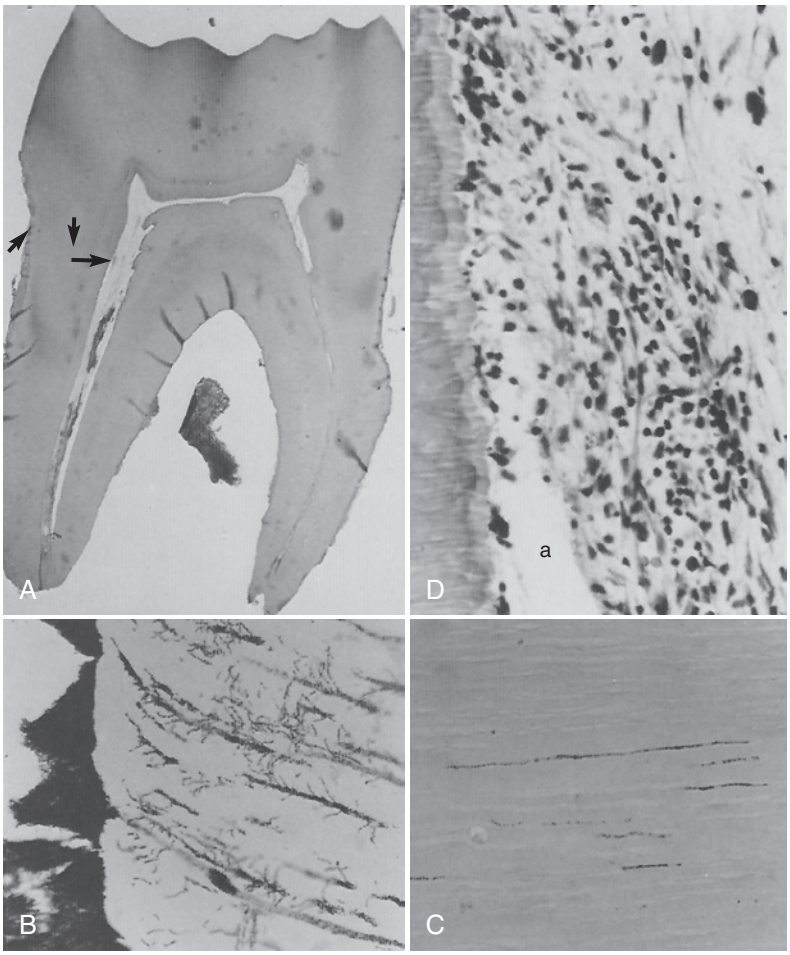

FIG. 13-25 Mandibular first molar of 49-year-old female, no pain. Clinically caries free. Calculus, periodontal disease, and bone loss from two thirds to three fourths of root. **A,** Note narrowness of pulp chamber seen throughout all serial sections (original magnification ×8.5). **B,** Bacterial plaque and adjacent dentinal tubules with bacteria (*oblique arrow* in **A**) (original magnification ×400). **C,** Farthest penetration of bacteria in dentinal tubules (*vertical arrow* in **A**) (original magnification ×400). **D,** Where dentinal tubules invaded by bacteria terminate in the pulp (*horizontal arrow* in A), a small but dense accumulation of lymphocytes and macrophages appears (*a,* artifact; pulp tissue torn away from dentin during processing) (original magnification ×400). (From Langeland K: Tissue response to dental caries, *Endod Dent Traumatol* 3:149, 1987. Reprinted with permission.)

tray for 6 weeks was safe for the pulp health for up to 10 years postoperatively, although the bleaching effectiveness may decline with time.[222] In one clinical study, premolars scheduled for orthodontic extraction had 38% H_2O_2 bleaching gel with and without a halogen light source applied. There were no histologic effects on the dental pulp at 2 to 15 days after bleaching.[136] When the study was performed on incisors with the same treatment for 45 minutes, areas of coagulation necrosis could be seen in the pulp.[56] Therefore, caution should be exercised when this caustic agent is used for extended bleaching.

PULPAL REACTIONS TO PERIODONTAL PROCEDURES

In an intact nontraumatized tooth, the dental pulp maintains its healthy existence throughout life because it is insulated from microbial irritation in the oral cavity (see also Chapter 17). Periodontal disease causes attachment loss, which exposes the root surface to the oral cavity. Occasionally, pulpal inflammation secondary to severe periodontitis is observed.[148] Some reports have described bacterial infiltration through dentinal tubules of exposed root surface causing mild inflammatory changes in the pulp (Fig. 13-25).[146] However, in a case report of 25 teeth that were extracted in a patient with extensive periodontal disease, none of the pulp tissue had significant inflammatory changes.[258] It is much more common for pulp necrosis, or for failure of healing of periapical lesions, to present clinically with signs of periodontal disease than for periodontal disease to cause pulpal pathosis. Primary endodontic, secondary periodontal pathosis is particularly evident if a perforation occurs in the pulp chamber or coronal third of the root during endodontic treatment and is not promptly treated in cases of cracked teeth/vertical root fractures or in cases of congenital tooth defects such as palatal groove defects. Thus, it is more likely for microbial irritants to move outward from a necrotic pulp to cause periodontal breakdown than for them to move inward from a periodontal pocket to cause

irreversible pathosis in the pulp. The reasons for this observation are not fully understood. However, if the assumption is that bacteria may migrate through patent dentinal tubules in these situations, as shown in Fig. 13-25, then it may be that the outward dentinal fluid flow in teeth with vital pulp contributes to the resistance to ingress of bacteria in sufficient amounts to cause a clinically significant disease process. Once the pulp degenerates, dentinal fluid flow no longer exists. Thus, microbial irritants from the pulp may promote pocket formation and periodontal bone loss,[121] and the prognosis of endodontic and periodontal treatment may be related,[122] as the microbial factors may pass across dentin more readily.

Periodontal scaling and root planing may result in removal of cementum and exposure of dentin to the oral cavity. Frequently, this treatment results in dentin hypersensitivity, as discussed previously. In theory, periodontal disease and its treatment should be associated with an increased incidence of pulpal pathosis. In an older study, investigators induced periodontal disease in a primate model using ligatures, and they compared the effects on the pulp of periodontal disease with or without scaling.[22] In teeth with periodontal disease, mild chronic inflammatory changes were observed in 29% of the teeth and could be seen in areas of the pulp related to bone loss. One of 40 teeth developed pulp necrosis. In teeth that received scaling, a similar percentage of 32% developed the same mild inflammation and none developed pulp necrosis. In a later clinical study,[23] 52 periodontitis patients who had 672 teeth with vital pulp were followed up and maintained every 3 to 6 months for 4 to 13 years (mean 8.7 years). Of those teeth, 255 were bridge abutments. The results showed a significantly higher chance of pulpal complications in teeth that were bridge abutments than in teeth that were not abutments (15% versus 3%; $P < 0.01$). Considering that both types of teeth had similar degrees of periodontal disease, the authors concluded that prosthodontic treatment is associated with pulpal involvement more frequently than periodontal disease and its treatment. Another histologic analysis of 46 teeth with varying degrees of periodontal disease and coronal restorations reached a similar conclusion.[62] Furthermore, two comprehensive reviews of the topic concluded that although the potential exists for periodontal disease and its treatment to cause pulpal pathosis, particularly if large lateral or accessory canals are exposed, this occurrence is rare.[97,226]

MECHANICAL IRRITANTS: ORTHODONTIC MOVEMENT

The most conspicuous pulpal change observed in response to orthodontic forces is hemodynamic. Both human and animal studies have confirmed that both lateral and intrusive forces increase pulpal blood flow.[145,182,190] Furthermore, blood flow alterations are not confined to the tooth in active movement. Observed increases in blood flow are seen in teeth adjacent to the focus of movement forces, implying that directed forces on one tooth could shunt blood to proximal vessels supplying other oral structures, including teeth. If orthodontic forces are extreme, circulatory interruptions can occur, resulting in pulpal necrosis.[35]

Biochemical, biologic, and histologic studies of the effects of orthodontic movement have confirmed that metabolic as well as inflammatory changes can result. The dental pulp tissue respiration rate is depressed after short-term application of orthodontic force.[95] Biochemical and molecular markers confirm that apoptosis and necrosis of pulpal cells is also increased subsequent to movement.[209] However, it was shown that the inflammatory mediators IL-1 alpha and TNF-alpha show a minor increase in the pulp during orthodontic movement, compared to the increase in periodontal tissues.[25] Histologic examination of pulps in teeth subjected to intrusive forces showed vascular congestion and dilatation as well as vacuolization of the odontoblastic layer.[245-247] Most if not all of these effects are due to circulatory changes, and the consensus is that they are transient provided that the movement forces are not excessive.[140] It was shown, however, that pulp necrosis of teeth that were undergoing orthodontic treatment, and had also been traumatized prior to that, is significantly increased compared to teeth that had either of these conditions but not the other, particularly in lateral incisors, and if the traumatized tooth had pulp canal obliteration.[18,19] As noted previously, substance P appears to increase in the dental pulp as a result of acutely induced occlusal trauma.[45] Also, CGRP increases under the influence of orthodontic forces.[46]

PULPAL REACTIONS TO ORTHODONTIC/ ORTHOGNATHIC SURGERY

It has been known for decades that osteotomies in the maxilla or mandible may cause disruption in the blood supply to teeth in the area of the surgery, with resultant inflammation or necrosis.[15,138,179,207] Occasionally, the teeth affected show postoperative manifestations common with traumatic injuries, such as pulp canal obliteration.[271] Animal studies have shown that if a safe distance of 5 to 10 mm is maintained between the site of the surgery and the teeth, minimal disruption occurs.[65,283] A number of studies have documented, using laser Doppler flowmetry, actual reduction in pulpal blood flow (PBF) immediately following maxillary Le Fort I osteotomy,[193,218,228] particularly if segmental osteotomy is performed.[71] In most cases, the blood flow is regained within months of the surgery. A modification of the Le Fort I osteotomy technique has been described in which the Le Fort I sectioning is combined with a horseshoe palatal osteotomy to spare any disruption to the descending palatine artery.[96] An examination of PBF of maxillary teeth using laser Doppler flowmetry as well as the responsiveness to electric pulp testing showed significant differences between the two surgical techniques in the postoperative recovery values (Fig. 13-26). In cases where the surgery did not disrupt the palatine artery, the PBF in the anterior teeth consistently increased without disruption in the postoperative period.

It should also be noted that, occasionally, teeth are traumatized during endotracheal intubation for surgery that requires general anesthesia, when the surgery itself is not related to the jaws or teeth.[235]

BIOMECHANICAL IRRITATION: PARAFUNCTIONAL HABITS

Occlusal loading of teeth affects deformation to varying degrees.[167] Whereas enamel is largely resistant to flexure, the underlying dentin demonstrates considerable elastic and viscoelastic characteristics. As a result, defects in enamel secondary to cracks, decay, or restorative preparation allow cuspal flexure with subsequent pulpal responses, presumably due to dentinal fluid flow secondary to compression and

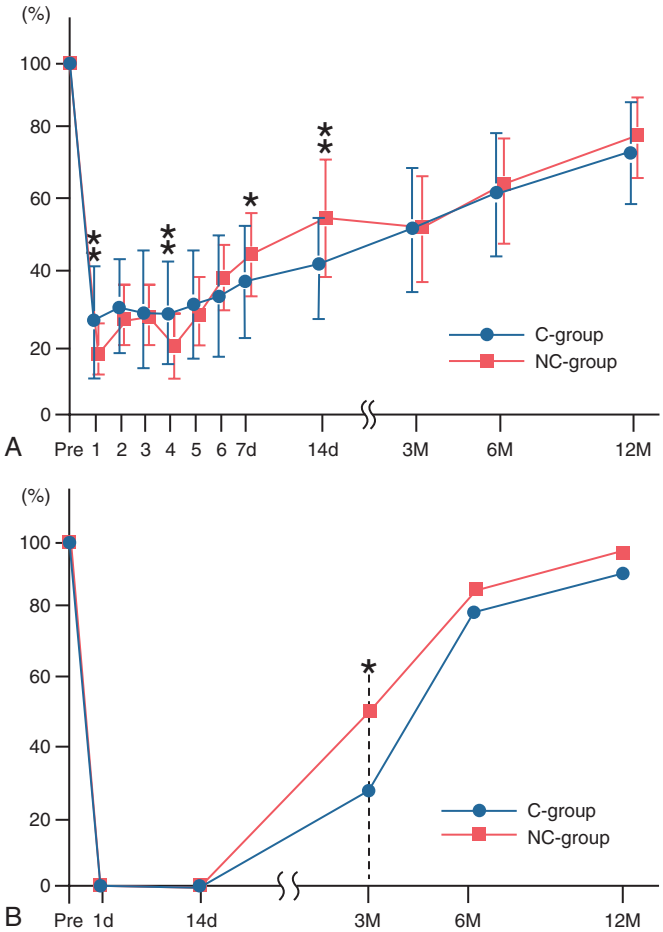

FIG. 13-26 A, The postoperative change of the mean pulpal blood flow in the upper incisors of the two groups (*Pre*, before the operation; *d*, day[s] after the operation; *M*, months after the operation; bars, SD; *, $P < .05$; **, $P < .01$). B, The postoperative change in the percentages of teeth (upper incisors) with positive pulpal sensitivity in the two groups (*Pre*, before the operation; *d*, day[s] after the operation; *M*, months after the operation; *, $P < .05$). (From Harada K, Sato M, Omura K: Blood-flow and neurosensory changes in the maxillary dental pulp after differing Le Fort I osteotomies, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:12, 2004. Reprinted with permission.)

microleakage.[103] The magnitude of the pulpal response is dictated by the degree and chronicity of dentinal deformation.

Multiple factors influence the degree of tooth deformation during occlusal loading. Investigators have noted that preparation geometry has a direct impact on cuspal flexure. The width of the occlusal isthmus relative to the faciolingual dimension of the tooth as well as the ablation of marginal ridges directly impact on the degree of cuspal flexure.[199,219,270] MOD preparations have been shown to effect a 50% reduction in cuspal stiffness and resistance to fracture. Physical properties of the restorative material can also play a part in cuspal flexure. Studies have shown that polymerization shrinkage of certain resin composites can induce an inward deflection of cusps with resultant stresses on tooth structure.[272,273]

Symptomatology from cuspal flexure can result from two primary sources. It has been theorized that cuspal flexure results in dentin deformation, thus promoting the dentinal fluid flow that activates nerve endings in the odontoblast layer of the tooth. This is supported in part by an in vitro study that found that dentinal fluid flow could be induced by occlusal loading of restored teeth.[103] A second source of pulpal pain is bacterial microleakage created by a gap at the restoration/dentin interface that is repeatedly opened during cycles of occlusal loading. If repeated cuspal flexure gives rise to a crack, dentinal exposure to bacteria and their by-products is even greater. It is likely that in vivo both dentinal fluid flow and bacterial access to dentinal surfaces work together to produce inflammation that is often manifested by thermal as well as biting sensitivity for the patient. In the instance of parafunctional habits, this is often combined with concussive periodontal forces to the periodontium that induce acute periradicular periodontitis, mobility, and radiographic changes.

On the cellular level, studies have shown increased levels of substance P after experimentally induced occlusal trauma.[45] Substance P is a modulator of both sensory and immune function in the dental pulp. Alterations in physiologic levels of this mediator could result in disruptions in the pain response as well as secondary stimulation of prostaglandin E2. Furthermore, the immunostimulatory ability of neuropeptides could initiate and maintain a chronic pulpal inflammation, possibly leading to necrosis. Although animal studies have suggested that pulp necrosis is a possible sequela to chronic occlusal trauma, there are no controlled clinical studies that confirm this.

Dentinal cracks expose tubules unoccluded by a smear layer and therefore offer a direct portal to the subjacent pulp. When dentinal tubules are freely exposed, there is an outward flow of dentinal fluid driven by relatively high pulpal tissue pressures. Dentinal fluid is composed of proteins such as fibrinogen and serum albumin, which can coagulate and effectively block the tubule lumen thereby limiting fluid egress and resultant dentin hypersensitivity. This phenomenon can occur within two days. As this serves as a short-term protective mechanism for the pulp, dentinal sclerosis and tertiary dentin formation ultimately can provide greater protection for the pulp and reduction of symptoms. Clinical interventions include the application of materials that occlude the tubules and extracoronal restorations to seal and prevent the propagation of cracks.

PULPAL REACTIONS TO IMPLANT PLACEMENT AND FUNCTION

Osseointegrated implants are now a common option for the replacement of missing teeth. The placement of implants requires multifaceted preoperative radiographic techniques, including intraoral, tomographic, cephalometric, and panoramic imaging.[264] This assures that implant placement fully rests in bone and does not compromise neighboring structures, including teeth. The lack of attention to the three-dimensional anatomy of the site of implant placement and the orientation of neighboring teeth may lead to the implant perforating the root and devitalizing the pulp.[162,249]

It is usually recommended that implants not be placed directly at a site where a periradicular lesion exists, particularly one with signs of purulence, as microbial irritants may interfere with osseointegration.[214] However, some data suggest that immediate implant placement in sites that have been adequately debrided is successful.[40] A systematic review generally corroborates this finding, provided that adequate preoperative, intraoperative, and postoperative antimicrobial measures are utilized.[50]

Case reports have also claimed that teeth with periradicular lesions may reduce the success of neighboring implants even if adequate endodontic treatment is performed.[248] To address this issue, a study was reported in which implants were placed to replace premolars in dogs; periradicular lesions were then induced, and some were treated nonsurgically or with surgical and nonsurgical treatments.[233] The results showed that the presence of treated or untreated periradicular lesions did not affect the long-term osseointegration of implants that were already osseointegrated. Clinical cases with complete resolution of periapical lesions that also involve neighboring implants, following adequate endodontic treatment, have been published.[155,244]

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CHAPTER 14

Microbiology of Endodontic Infections

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CHAPTER OUTLINE

Apical Periodontitis as an Infectious Disease
Routes of Root Canal Infection
Mechanisms of Microbial Pathogenicity and Virulence Factors
Spatial Distribution of the Microbiota: Anatomy of Infection
Biofilm and Community-Based Microbial Pathogenesis
Biofilm and Bacterial Interactions
Biofilm Community Lifestyle
Resistance to Antimicrobial Agents
Apical Periodontitis as a Biofilm-Related Disease
Methods for Microbial Identification
The Five Generations of Endodontic Microbiology Studies
Impact of Molecular Methods in Endodontic Microbiology
Types of Endodontic Infections
Diversity of the Endodontic Microbiota
Primary Intraradicular Infection
Microbial Composition and Diversity
Symptomatic Infections
Geographic Influence
Microbial Ecology and the Root Canal Ecosystem
Other Microorganisms in Endodontic Infections
Persistent/Secondary Endodontic Infections
Persistent/Secondary Infections and Treatment Failure
Bacteria at the Root Obturation Stage
Microbiota in Endodontically Treated Teeth
Extraradicular Infections

Apical periodontitis is essentially an inflammatory disease of microbial etiology primarily caused by infection of the root canal system.[196] Although chemical and physical factors can induce periradicular inflammation, a large body of scientific evidence indicates that endodontic infection is essential to the progression and perpetuation of the different forms of apical periodontitis.[16,86,120,250] Endodontic infection develops in root canals devoid of host defenses, as a consequence of either pulp necrosis (as a sequel to caries, trauma, periodontal disease, or invasive operative procedures) or pulp removal for treatment.

Although fungi and most recently archaea and viruses have been found in association with endodontic infections,[57,177,232,238,268] bacteria are the major microorganisms implicated in the pathogenesis of apical periodontitis. In advanced stages of the endodontic infectious process, bacterial organizations resembling biofilms can be observed adhered to the canal walls.[121,126,152,223] Consequently, apical periodontitis has been included in the roll of biofilm-related oral diseases.[92,228] Bacteria colonizing the root canal system enter in contact with the periradicular tissues via apical/lateral foramina or root perforations. As a consequence of the encounter between bacteria and host defenses, inflammatory changes take place in the periradicular tissues and give rise to the development of apical periodontitis. Depending on several bacterial and host-related factors, endodontic infections can lead to acute (symptomatic) or chronic (asymptomatic) apical periodontitis.

The ultimate goal of endodontic treatment is either to prevent the development of apical periodontitis or, in cases where the disease is already present, to create adequate conditions for periradicular tissue healing. The intent is to repair and preserve the tooth and associated periradicular bone. Because apical periodontitis is an infectious disease, the rationale for endodontic treatment is to eradicate the occurring infection or prevent microorganisms from infecting or reinfecting the root canal or the periradicular tissues. The cardinal principle of any health care profession is the thorough understanding of disease etiology and pathogenesis, which provides a framework for effective treatment. In this context, understanding the microbiologic aspects of apical periodontitis is the basis for endodontic practice and should be managed with an evidence-based approach. This chapter focuses on diverse aspects of endodontic microbiology, including pathogenetic, taxonomic, morphologic, and ecologic issues.

APICAL PERIODONTITIS AS AN INFECTIOUS DISEASE

The first recorded observation of bacteria in the root canal dates back to the 17th century and the Dutch amateur microscope builder Antony van Leeuwenhoek (1632-1723). He reported that the root canals of a decayed tooth "were stuffed with a soft matter" and that "the whole stuff" seemed to be

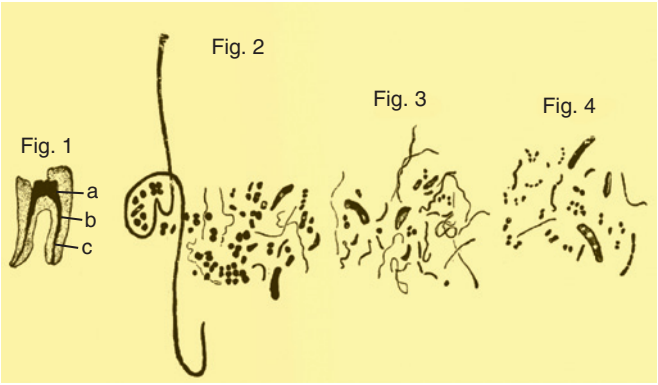

FIG. 14-1 Drawings from Miller's classic paper showing different bacterial forms in a root canal sample observed by microscopy.

alive.[46] At that time, the role of Leeuwenhoek's "animalcules" in disease causation was unsuspected. It took almost 200 years until his observation was confirmed and a cause-and-effect relationship between bacteria and apical periodontitis was suggested. This occurred specifically in 1894, when Willoughby Dayton Miller, an American dentist working at the laboratory of Robert Koch in Berlin, Germany, published a milestone study reporting the association between bacteria and apical periodontitis after an analysis of samples collected from root canals.[115] By means of bacterioscopy of the canal samples, he found bacterial cells in the three basic morphologies known at the time: cocci, bacilli, and spirilla (or spirochetes) (Fig. 14-1). Morphologically, the endodontic microbiota was clearly different in the coronal, middle, and apical parts of the root canal. Spirochetes were found in high frequencies in abscessed cases, and a pathogenic role was suspected for these bacteria. Most of the bacteria Miller observed under light microscopy could not be cultivated using the technology available at that time. Those bacteria were conceivably anaerobic bacteria, which were only successfully cultivated about 50 to 100 years later with the advent of anaerobic culture techniques. However, it is now widely recognized that a large number of bacterial species living in diverse environments still remain to be cultivated by current technology,[6,147] and the root canal is no exception (discussed later in this chapter). Based on his findings, Miller raised the hypothesis that bacteria were the causative agents of apical periodontitis.

Approximately 70 years after Miller's classic study, his assumptions were confirmed by an elegant study from Kakehashi and colleagues.[86] These authors investigated the response of exposed dental pulps to the oral cavity in conventional and germ-free rats. Histologic evaluation was performed and revealed that pulp necrosis and apical periodontitis lesions developed in all conventional rats; however, the exposed pulps of germ-free rats not only remained vital but also repaired themselves with hard-tissue formation. Dentin-like tissue sealed the exposure area and isolated the pulps again from the oral cavity.

The important role of bacteria in the etiology of apical periodontitis was further confirmed by Sundqvist's classic study.[250] This author applied advanced anaerobic culturing techniques to the evaluation of bacteria occurring in the root canals of teeth whose pulps became necrotic after trauma. Bacteria were found only in the root canals of teeth exhibiting radiographic evidence of apical periodontitis, confirming the infectious cause of this disease. Anaerobic bacteria accounted for more than 90% of the isolates. Findings from Sundqvist's study also demonstrated that in the absence of infection, the necrotic pulp tissue itself and stagnant tissue fluid in the root canal cannot induce or perpetuate apical periodontitis lesions.

Möller and colleagues[120] also provided strong evidence about the microbial causation of apical periodontitis. Their study using monkeys' teeth demonstrated that only devitalized pulps that were infected induced apical periodontitis lesions, whereas devitalized and noninfected pulps showed an absence of significant pathologic changes in the periradicular tissues. In addition to corroborating the importance of microorganisms for the development of apical periodontitis, this study also confirmed that necrotic pulp tissue per se is unable to induce and maintain an apical periodontitis lesion.

Microorganisms causing apical periodontitis are primarily organized in biofilms colonizing the root canal system. Nair[126] was possibly the first to observe intracanal bacterial organizations adhered to the root canal walls, resembling biofilm structures. Several other morphologic studies found similar structures,[121,190,223] but it was not until the study of Ricucci and Siqueira[152] that the high prevalence of bacterial biofilms were consistently revealed in association with primary and post-treatment apical periodontitis (see the section Spatial Distribution of the Microbiota: Anatomy of Infection for a further discussion of biofilms in endodontic infections).

ROUTES OF ROOT CANAL INFECTION

Under normal conditions, the pulpodentin complex is sterile and isolated from oral microbiota by overlying enamel, dentin, and cementum. In the event that the integrity of these natural layers is breached (e.g., as a result of caries, trauma-induced fractures and cracks, restorative procedures, scaling and root planning, attrition, abrasion) or naturally absent (e.g., because of gaps in the cemental coating at the cervical root surface), the pulpodentin complex is exposed to the oral environment. The pulpodentin complex is then challenged by microorganisms present in caries lesions, saliva bathing the exposed area, or dental plaque formed on the exposed area. Microorganisms from subgingival biofilms associated with periodontal disease may also have access to the pulp via dentinal tubules at the cervical region of the tooth and lateral or apical foramina (see also Chapter 25). Microorganisms may also have access to the root canal any time during or after endodontic intervention, emphasizing the need for effective (fluid tight) use of the rubber dam.

Whenever dentin is exposed, the pulp is put at risk of infection as a consequence of the permeability of normal dentin dictated by its tubular structure[135] (Fig. 14-2). Dentinal tubules traverse the entire width of the dentin and have a conical conformation, with the largest diameter located near the pulp (mean, 2.5 μm) and the smallest diameter in the periphery, near the enamel or cementum (mean, 0.9 μm).[62] The smallest tubule diameter is entirely compatible with the cell diameter of most oral bacterial species, which usually ranges from 0.2 to 0.7 μm. One might assume that once exposed, dentin offers an unimpeded pathway for bacteria to reach the pulp via these tubules. However, it has been demonstrated that bacterial invasion of dentinal tubules occurs more rapidly with a nonvital pulp than with a vital pulp.[124] With a vital pulp, outward movement of dentinal fluid and the tubular contents (including

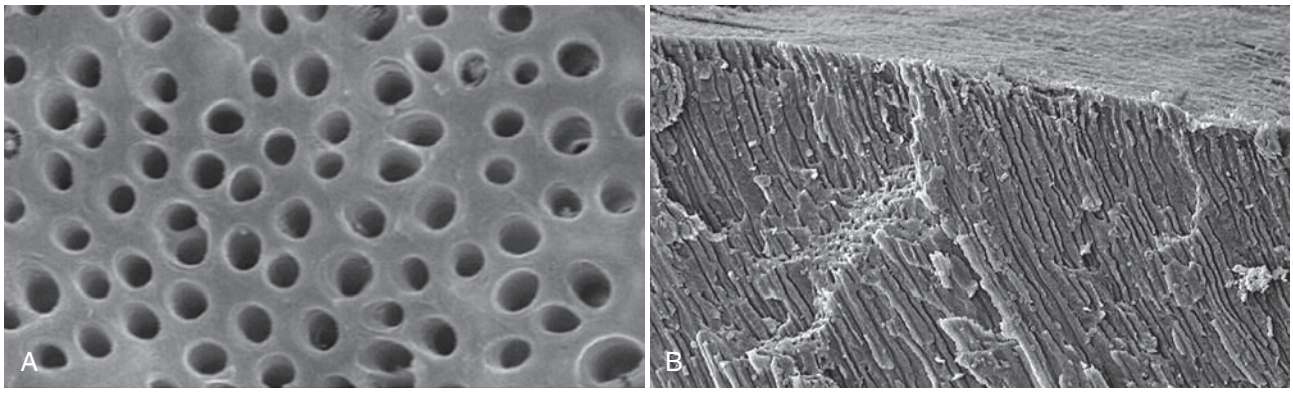

FIG. 14-2 A, Scanning electron micrographs of dentin showing tubules in cross-sectional view (magnification ×850). B, Longitudinal views (magnification ×130).

odontoblast processes, collagen fibrils, and the sheathlike lamina limitans that lines the tubules) influence dentinal permeability and can conceivably delay intratubular invasion by bacteria. Because of the presence of tubular contents, the functional or physiologic diameter of the tubules is only 5% to 10% of the anatomic diameter seen by microscopy.[113] Other factors such as dentinal sclerosis beneath a carious lesion, tertiary dentin, smear layer, and intratubular deposition of fibrinogen also reduce dentin permeability and thereby limit or even impede bacterial progression to the pulp via dentinal tubules.[137] Host defense molecules, such as antibodies and components of the complement system, may also be present in the dentinal fluid of vital teeth and can assist in the protection against deep bacterial invasion of dentin.[3,130,131] As long as the pulp is vital, dentinal exposure does not represent a significant route of pulpal infection, except when dentin thickness is considerably reduced or when the dentin permeability is significantly increased.

Most of the bacteria in the carious process are nonmotile; they invade dentin by repeated cell division, which pushes cells into tubules. Bacterial cells may also be forced into tubules by hydrostatic pressures developed on dentin during mastication.[114] Bacteria inside tubules under a deep carious lesion can reach the pulp even before frank pulpal exposure.[81] As mentioned, it has been assumed that the pulp will not be infected if it is still vital. The few bacteria that reach the pulp may not be significant, because the vital pulp can eliminate such a transient infection and rapidly clear or remove bacterial products. This efficient clearance mechanism tends to prevent injurious agents from reaching a high enough concentration to induce significant inflammatory reactions.[136] On the other hand, if the vitality of the pulp is compromised and the defense mechanisms are impaired, even a small amount of bacteria may initiate infection.

Direct exposure of the dental pulp to the oral cavity is the most obvious route of endodontic infection. Caries is the most common cause of pulp exposure, but bacteria may also reach the pulp via direct pulp exposure as a result of iatrogenic restorative procedures or trauma. The exposed pulp tissue comes in direct contact with oral bacteria from carious lesions, saliva, or plaque accumulated onto the exposed surface. Almost invariably, exposed pulps will undergo inflammation and necrosis and become infected. The time elapsed between pulp exposure and infection of the entire canal is unpredictable, but it is usually a slow process.[38]

The egress of microorganisms and their products from infected root canals through apical, lateral, or furcation foramina, dentinal tubules, and iatrogenic root perforations can directly affect the surrounding periodontal tissues and give rise to pathologic changes in these tissues. However, there is no consensus as to whether the opposite is true—that is, whether subgingival biofilms associated with periodontal disease can directly cause pulpal disease. Conceptually, microorganisms in subgingival plaque biofilms associated with periodontal disease could reach the pulp by the same pathways intracanal microorganisms reach the periodontium and could thereby exert harmful effects on the pulp. However, it has been demonstrated that although degenerative and inflammatory changes of different degrees may occur in the pulp of teeth with associated marginal periodontitis, pulpal necrosis as a consequence of periodontal disease only develops if the periodontal pocket reaches the apical foramen, leading to irreversible damage to the main blood vessels that penetrate through this foramen[98] (Fig. 14-3). After the pulp becomes necrotic, periodontal bacteria can reach the root canal system via exposed dentinal tubules at the cervical area of the root or via lateral and apical foramina to establish an endodontic infectious process.

It has been claimed that microorganisms can reach the pulp by *anachoresis*.[73] Theoretically, microorganisms can be transported in the blood or lymph to an area of tissue damage, where they leave the vessel, enter the damaged tissue, and establish an infection.[64,156] However, there is no clear evidence showing that this process can represent a route for root canal infection. It has been revealed that bacteria could not be recovered from unfilled root canals when the bloodstream was experimentally infected, unless the root canals were overinstrumented during the period of bacteremia, with resulting injury to periodontal blood vessels and blood seepage into the canal.[42] Another argument against anachoresis as a route for pulpal infection comes from the study by Möller and colleagues,[120] who induced pulpal necrosis in monkeys' teeth and reported that all cases of aseptic necrosis remained bacteria-free after 6 to 7 months of observation.

Bacteria have been isolated from traumatized teeth with necrotic pulps with apparently intact crowns.[250,278] Although anachoresis has been suggested to be the mechanism through which these traumatized teeth become infected,[73] current evidence indicates that the main pathway of pulpal infection in these cases is dentinal exposure due to enamel cracks.[106,107] Macro- and microcracks in enamel can be present in most teeth

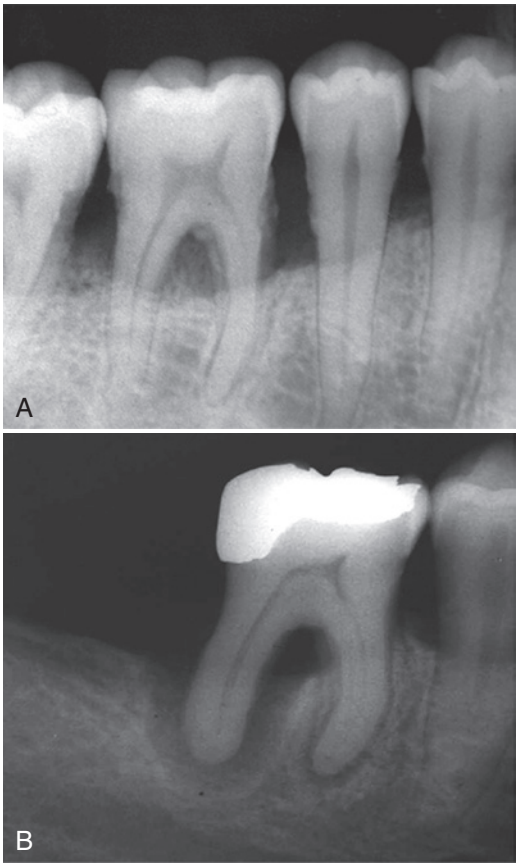

FIG. 14-3 Periodontal disease (A) mostly affects the pulp vitality when the subgingival biofilm reaches the apical foramen (B).

(not only traumatized teeth) and do not necessarily end at the enamel-dentin junction; they can extend deep into the dentin.[107] A large number of dentinal tubules can be exposed to the oral environment by a single crack. These cracks can be clogged with dental plaque and provide portals of entry for bacteria. If the pulp remains vital after trauma, bacterial penetration into tubules is counteracted by the dentinal fluid and tubular contents, as discussed earlier, and pulpal health is not usually jeopardized. But if the pulp becomes necrotic as a consequence of trauma, it loses the ability to protect itself against bacterial invasion, and regardless of dentin thickness, dentinal tubules then will become true avenues through which bacteria can reach and colonize the necrotic pulp.

Whatever the route of bacterial access to the root canal, necrosis of pulp tissue is a prerequisite for the establishment of primary endodontic infections. To reiterate: if the pulp is vital, it can protect itself against bacterial invasion and colonization. If the pulp becomes necrotic due to caries, trauma, operative procedures, or periodontal disease, then it can be easily infected. This is because host defenses do not function in the necrotic pulp tissue, and those in the periradicular tissues do not reach deep into the root canal space.

Another situation in which the root canal system is devoid of host defenses relates to cases in which the pulp was removed for treatment. Microbial penetration in the canal can occur during treatment, between appointments, or even after root canal obturation. The main causes of microbial introduction into the canal *during treatment* include remnants of dental biofilm, calculus, or caries on the tooth crown; leaking rubber dam; contamination of endodontic instruments (e.g., after touching with the fingers); and contamination of irrigant solutions or other solutions of intracanal use (e.g., saline solution, distilled water, citric acid). Microorganisms can also enter the root canal system *between appointments* by leakage through the temporary restorative material; breakdown, fracture, or loss of the temporary restoration; fracture of the tooth structure; and in teeth left open for drainage. Microorganisms can penetrate the root canal system even *after completion of the root canal obturation* by leakage through the temporary or permanent restorative material; breakdown, fracture, or loss of the temporary/permanent restoration; fracture of the tooth structure; recurrent decay contaminating the root canal obturation; or delay in the placement of permanent restorations.[205]

MECHANISMS OF MICROBIAL PATHOGENICITY AND VIRULENCE FACTORS

The ability of a microorganism to cause disease is regarded as its *pathogenicity*. *Virulence* denotes the degree of pathogenicity of a microorganism, and *virulence factors* are the microbial products, structural cellular components, or strategies that contribute to pathogenicity. One example of bacterial strategy that contributes to pathogenicity includes the ability to coaggregate and form biofilms, which confers protection against microbial competitors, host defenses, and antimicrobial agents. Some microorganisms routinely cause disease in a given host and are called *primary pathogens*. Other microorganisms cause disease only when host defenses are impaired and are called *opportunistic pathogens*. Bacteria that make up the normal microbiota are usually present as harmless commensals and live in balance with the host. One of the greatest beneficial effects of human microbiota is probably the tendency to protect the host from exogenous infections by excluding other microorganisms. Nevertheless, in certain situations, the balance may be disturbed by a decrease in the normal level of resistance, and then the commensal bacteria are usually the first to take advantage. Most bacteria involved with endodontic infections are normal inhabitants of the oral microbiota that exploit changes in the balance of the host–bacteria relationship, becoming opportunistic pathogens.

Bacteria involved with the pathogenesis of primary apical periodontitis may have participated in the early stages of pulp inflammation and necrosis, or they may have gained entry into the root canal space any time after pulpal necrosis. In the former situation, involved bacteria are usually those present in the advanced front of caries lesions and from saliva bathing the affected area. Bacteria in caries lesions form authentic biofilms adhered to dentin (Fig. 14-4). Diffusion of bacterial products through dentinal tubules induces pulpal inflammation long before the tissue is exposed. After pulp exposure, the surface of the tissue can also be colonized and covered by bacteria present in the caries biofilm. The exposed pulp tissue is in direct contact with bacteria and their products and responds with severe inflammation. Some tissue invasion by bacteria may also occur. Bacteria in the battlefront have to survive the attack from the host defenses and at the same time acquire nutrients to keep themselves alive. In this bacteria–pulp clash, the latter invariably is "defeated" and becomes necrotic, so

bacteria move forward and "occupy the territory"—that is, they colonize the necrotic tissue. These events advance through tissue compartments, coalesce, and move toward the apical part of the canal until virtually the entire root canal is necrotic and infected (Fig. 14-5). At this stage, involved bacteria can be regarded as the early root canal colonizers or pioneer species.

Early colonizers play an important role in the initiation of the apical periodontitis disease process. Moreover, they may significantly modify the environment, making it conducive to the establishment of other bacterial groups. These new species may have access to the canal via coronal exposure or exposed dentinal tubules, establish themselves, and contribute to a shift in the microbiota. Rearrangement in the proportions of the pioneer species and latecomers occurs, and as the environment changes, some early colonizers are expected to no longer participate in the consortium of advanced disease. With the passage of time, the endodontic microbiota becomes more and more structurally and spatially organized.

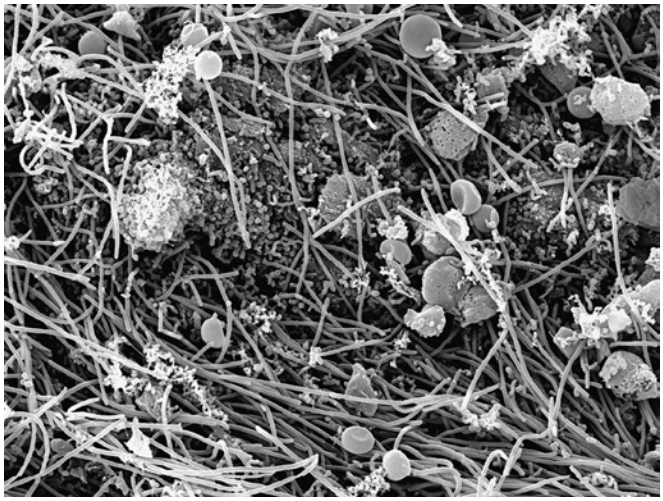

FIG. 14-4 Scanning electron micrograph showing a bacterial biofilm covering dentin in a deep carious lesion. Note the presence of different bacterial morphotypes (magnification ×3500). (From Torabinejad M, Walton RE: *Endodontics: principles and practice,* ed 4, St. Louis, 2009, Saunders/Elsevier.)

Some virulence attributes required for pathogens to thrive in other sites may be of no value for bacteria that reach the root canal after necrosis—for instance, the ability to evade host defenses. This is because latecomers face no significant opposition from host defenses, which are no longer active in the canal after necrosis. Although colonization may appear an easy task for late colonizers, other environmental factors (e.g., interaction with pioneer species, oxygen tension, nutrient availability) will determine whether new species entering the canal will succeed in establishing themselves and join the early colonizers to make up a dynamic mixed community in the root canal. Ultimately, the root canals of teeth with radiographically detectable apical periodontitis lesions harbor both early colonizers that managed to stay in the canals and late colonizers that managed to adapt to the new but propitious environmental conditions.[214]

Bacteria colonizing the necrotic root canal induce damage to the periradicular tissues and give rise to inflammatory changes. In fact, periradicular inflammation can be observed even before the frontline of infection reaches the apical foramen.[8,121,242,280] Bacteria exert their pathogenicity by wreaking havoc on the host tissues through direct or indirect mechanisms. Bacterial virulence factors that cause direct tissue harm include those that damage host cells or the intercellular matrix of the connective tissue. These factors usually involve secreted products, including enzymes, exotoxins, heat-shock proteins, and metabolic end products.[214] Furthermore, bacterial structural components, including lipopolysaccharide (LPS), peptidoglycan, lipoteichoic acid, fimbriae, flagella, outer membrane proteins and vesicles, lipoproteins, DNA, and exopolysaccharides, can act as modulins by stimulating the development of

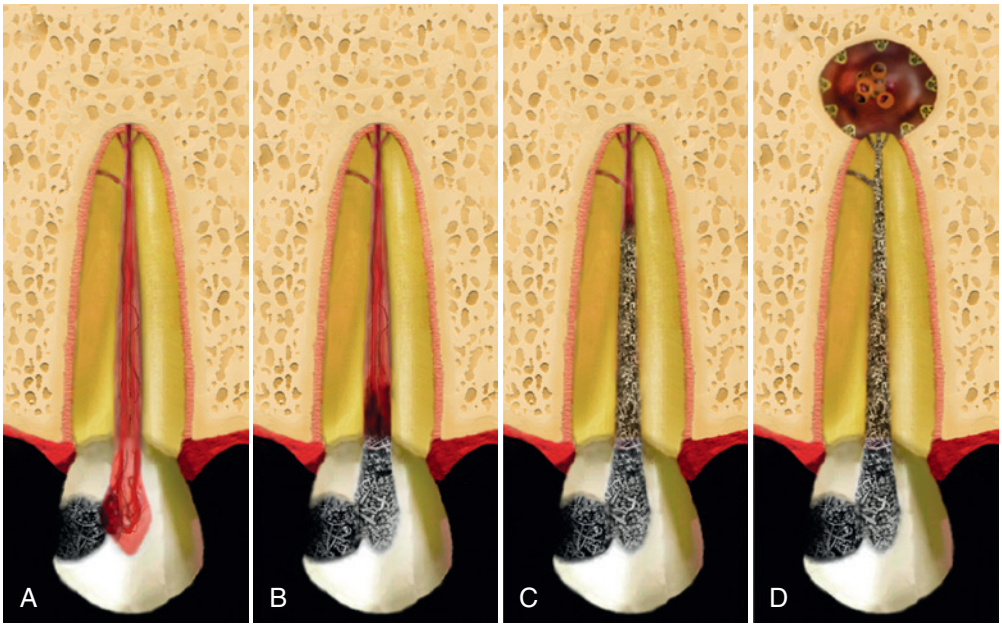

FIG. 14-5 Dynamics of pulp response from caries exposure (**A**) to pulp inflammation (**B**) to pulp necrosis (**C**) to apical periodontitis formation (**D**).

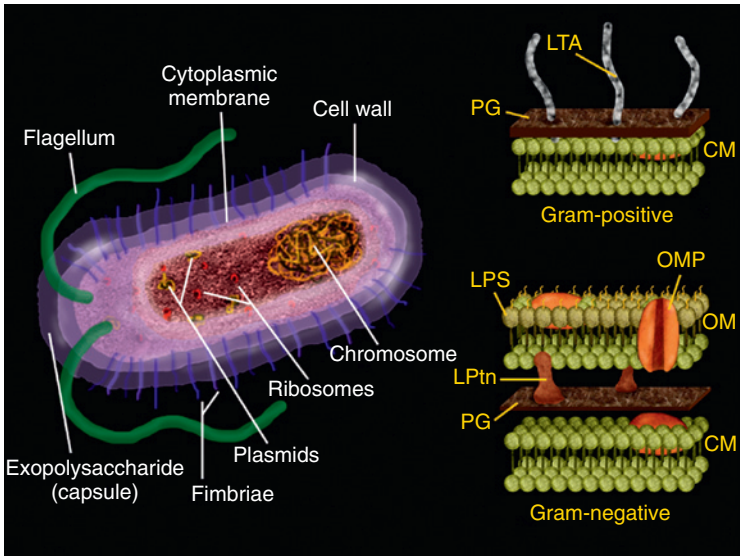

FIG. 14-6 Bacterial cell and its structural components that can act as virulence factors. *Right:* A detailed scheme of the bacterial cell walls from gram-positive and gram-negative bacteria. CM, cytoplasmic membrane; LPS, lipopolysaccharide (endotoxin); LPtn, lipoproteins; LTA, lipoteichoic acid; OM, outer membrane; OMP, outer membrane protein; PG, peptidoglycan.

host immune reactions capable not only of defending the host against infection but also of causing severe tissue destruction[80,214,265] (Fig. 14-6). For instance, inflammatory and noninflammatory host cells can be stimulated by bacterial components to release chemical mediators such as cytokines and prostaglandins, which are involved in the induction of bone resorption characteristically observed in asymptomatic (chronic) apical periodontitis lesions.[184] Another example of indirect damage caused by bacteria is the formation of purulent exudate in acute apical abscesses. Host defense mechanisms against bacteria emanating from the root canal appear to be the most important factor involved in the formation of purulent exudate associated with abscesses. Formation of oxygen-derived free radicals, such as superoxide and hydrogen peroxide, alongside the release of lysosomal enzymes by polymorphonuclear leukocytes, gives rise to destruction of the connective extracellular matrix, leading to pus formation.[264] Although direct damage caused by bacterial products may certainly be involved in the pathogenesis of apical periodontitis, bacterial indirect destructive effects seem to be more significant in this regard.[197]

Apical periodontitis is a multifactorial disease that is resultant of the interplay of many host and bacterial factors. Few if any of the putative endodontic pathogens are individually capable of inducing all of the events involved in the pathogenesis of the different forms of apical periodontitis. Probably, the process requires an integrated and orchestrated interaction of the selected members of the mixed endodontic microbiota and their respective virulence attributes. Although LPS is undoubtedly the most studied and quoted virulence factor, it sounds simplistic to ascribe to this molecule all responsibility for apical periodontitis causation. This statement is further reinforced by the fact that some cases of primary infections and many cases of secondary/persistent infections harbor exclusively gram-positive bacteria. Therefore, the involvement of other factors must not be overlooked. In fact, the pathogenesis of different forms of apical periodontitis and even the same form in different individuals is unlikely to follow a stereotyped course with regard to the bacterial mediators involved.[197]

SPATIAL DISTRIBUTION OF THE MICROBIOTA: ANATOMY OF INFECTION

Mounting evidence indicates that apical periodontitis, like caries and periodontal diseases, is also a biofilm-related disease. Morphologic studies have shown that the root canal microbiota in primary infections is dominated by bacterial morphotypes that include cocci, rods, filaments, and spirilla (spirochetes) (Fig. 14-7). Fungal cells are sporadically found[190,223] (Fig. 14-8). Although planktonic bacterial cells suspended in a fluid phase and enmeshed in necrotic pulp tissue can be observed in the main root canal, most bacteria colonizing the root canal system usually grow in sessile multispecies biofilms adhered to the dentinal walls[121,126,223] (Fig. 14-9). Lateral canals, apical ramifications, and isthmuses connecting main canals may also be clogged with bacterial biofilms[125,151] (Figs. 14-10 and 14-11).

Bacterial cells from endodontic biofilms are often seen penetrating the dentinal tubules (Fig. 14-12). Dentinal tubule infection can occur in about 70% to 80% of the teeth evincing apical periodontitis lesions.[112,143] A shallow penetration is more common, but bacterial cells can be observed reaching approximately 300 µm in some teeth[223] (Fig. 14-13). Dividing cells are frequently observed within tubules during in situ investigations[223] (see Fig. 14-13), indicating that bacteria can derive nutrients within tubules, probably from degrading odontoblastic processes, denatured collagen, bacterial cells that die during the course of infection, and intracanal fluids that enter the tubules by capillarity.

Several putative endodontic pathogens have been shown to be capable of penetrating dentinal tubules in vitro, including *Porphyromonas endodontalis, Porphyromonas gingivalis, Fusobacterium nucleatum, Actinomyces israelii, Propionibacterium*

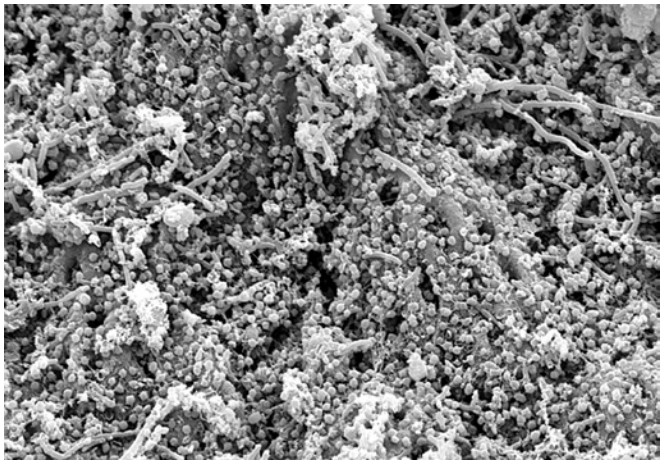

FIG. 14-7 Mixed bacterial population colonizing the root canal wall. Cocci are the predominant forms, but rods, filaments, and spirochetes are also observed. In some areas, coccoid cells are relatively apart from each other (magnification ×2200). (From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:174, 2002.)

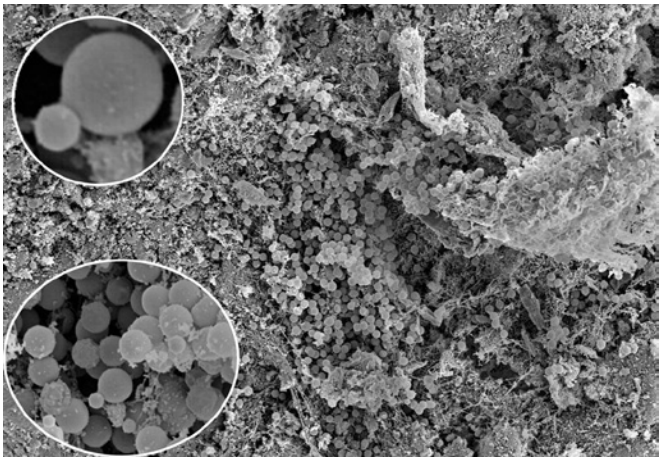

FIG. 14-8 Heavy colonization of yeast cells in the root canal of an extracted tooth with primary infection associated with apical periodontitis (magnification ×300). Note that some cells are in the stage of budding. A daughter cell is growing on the surface of the mother cell (insets: magnification ×2700 [*bottom*], magnification ×3500[*top*]). (From Siqueira JF Jr, Sen BH: Fungi in endodontic infections, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:632, 2004.)

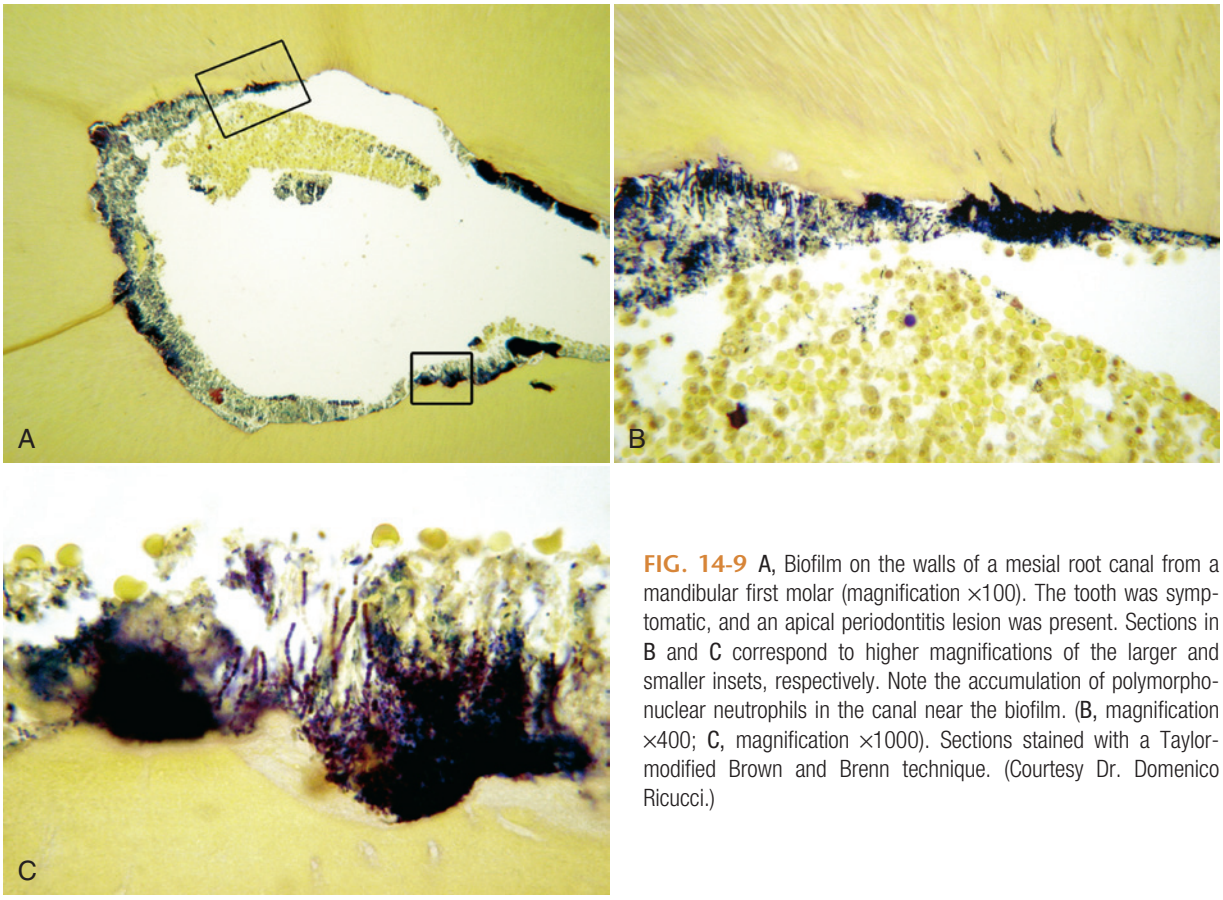

FIG. 14-9 **A**, Biofilm on the walls of a mesial root canal from a mandibular first molar (magnification ×100). The tooth was symptomatic, and an apical periodontitis lesion was present. Sections in **B** and **C** correspond to higher magnifications of the larger and smaller insets, respectively. Note the accumulation of polymorphonuclear neutrophils in the canal near the biofilm. (**B**, magnification ×400; **C**, magnification ×1000). Sections stained with a Taylor-modified Brown and Brenn technique. (Courtesy Dr. Domenico Ricucci.)

acnes, Enterococcus faecalis, Candida albicans, and streptococci.[107,141,201,224,273] In their clinical study, Peters and colleagues[143] isolated and identified bacteria present in root dentin at different depths, and the most common isolates belonged to the genera *Prevotella, Porphyromonas, Fusobacterium, Veillonella, Peptostreptococcus, Eubacterium, Actinomyces,* lactobacilli, and streptococci. Using immunohistologic analysis, Matsuo and associates[112] observed the occurrence of *F. nucleatum, Pseudoramibacter alactolyticus, Eubacterium nodatum, Lactobacillus casei,* and *Parvimonas micra* inside dentinal tubules from the canal walls of extracted infected teeth with apical periodontitis.

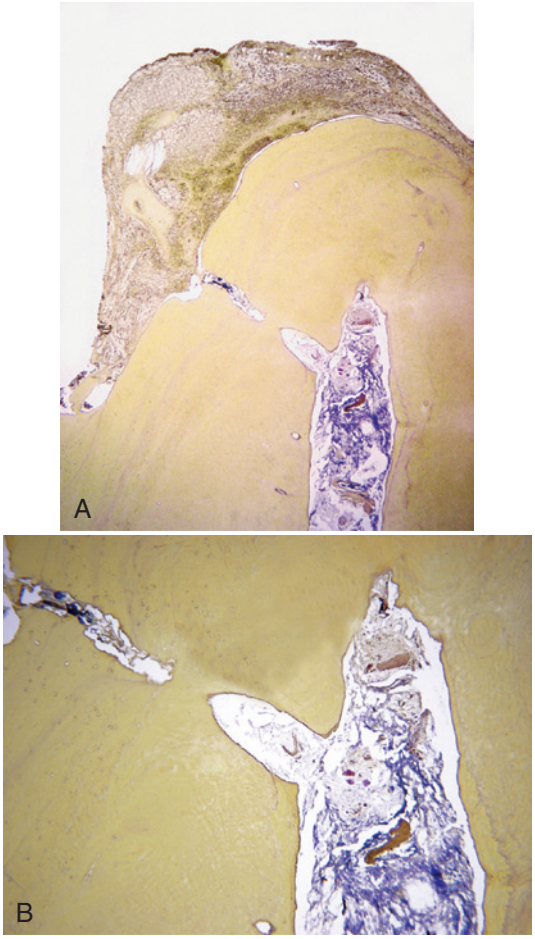

FIG. 14-10 A, Bacterial biofilm in the necrotic canal and in an apical ramification contiguous to inflamed periradicular tissues (magnification ×25). B, Higher magnification of A (magnification ×100). Sections stained with a Taylor-modified Brown and Brenn technique. (Courtesy Dr. Domenico Ricucci.)

Whereas bacteria present as planktonic cells in the main root canal may be easily accessed and eliminated by instruments and substances used during endodontic treatment, those organized in biofilms attached to the canal walls or located into isthmuses, lateral canals, and dentinal tubules are definitely more difficult to reach[125,267] and may require special therapeutic strategies to be eradicated.

BIOFILM AND COMMUNITY-BASED MICROBIAL PATHOGENESIS

Individual microorganisms proliferating in a habitat give rise to *populations*. Such populations often occur as microcolonies in the environment. Populations interact with one another to

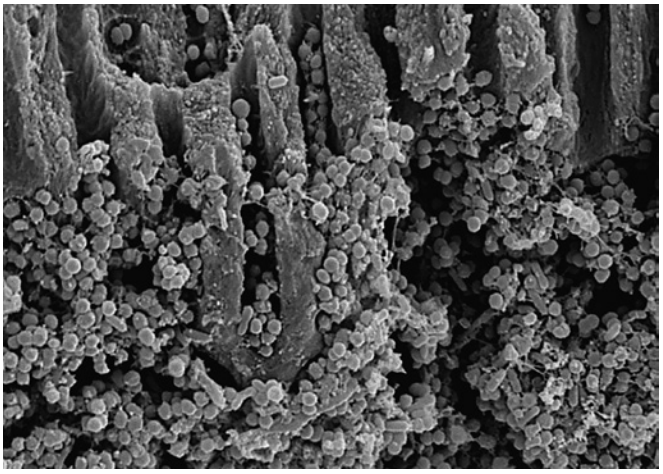

FIG. 14-12 Heavy infection of the root canal walls, mainly by cocci, but some small rods are also seen. Cocci are penetrating into dentinal tubules (magnification ×3500). (From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:174, 2002.)

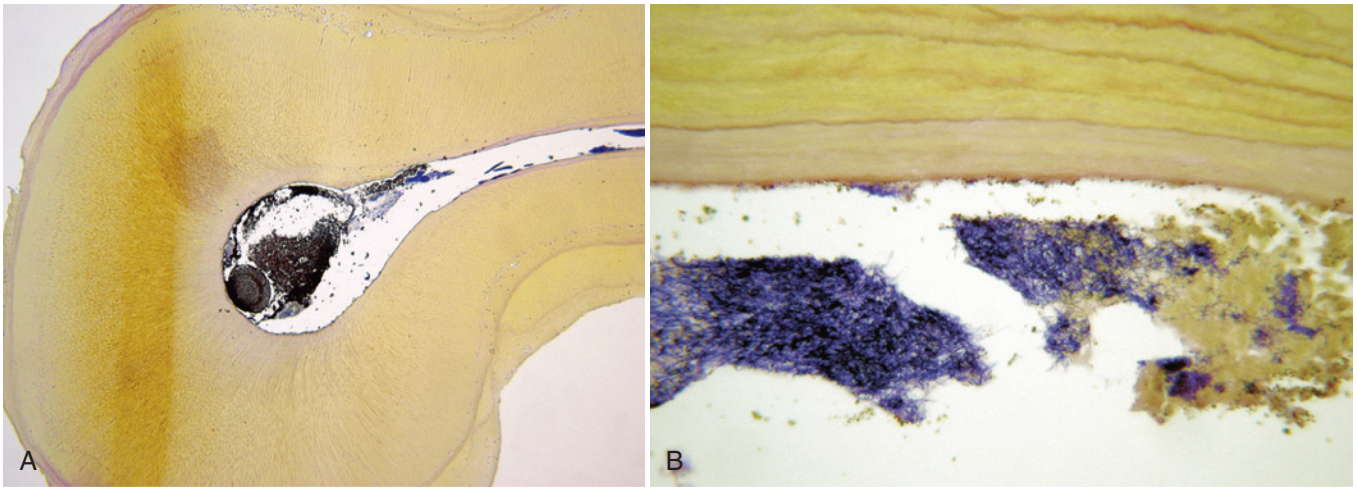

FIG. 14-11 Transverse sections of a maxillary second molar with fused mesial and palatal roots. A, Heavy bacterial infection of the canal, spreading to an isthmus (magnification ×25). B, Higher magnification of the isthmus clogged with bacteria (magnification ×400). Sections stained with a Taylor-modified Brown and Brenn technique. (Courtesy Dr. Domenico Ricucci.)

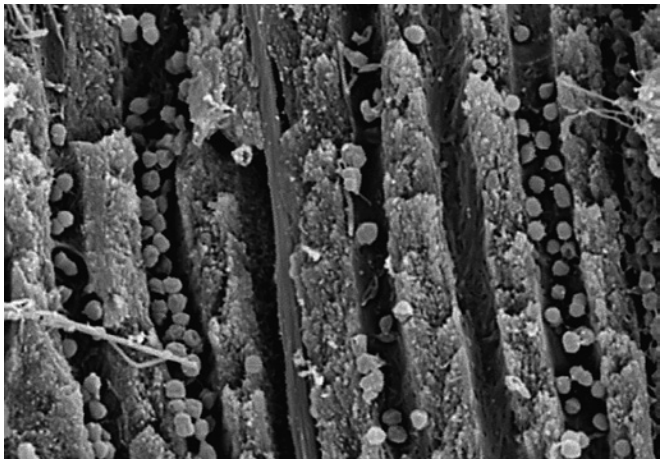

FIG. 14-13 Cocci in dentinal tubules approximately 300 μm from the main root canal (magnification ×5000). (From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:174, 2002.)

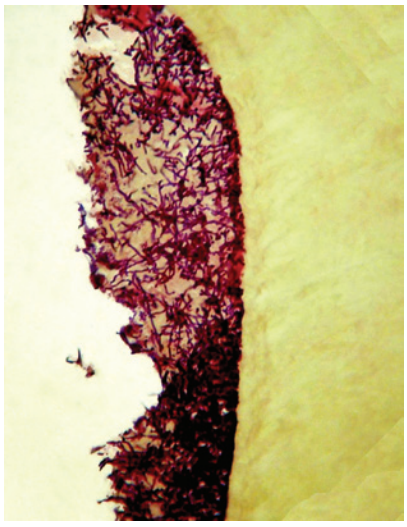

FIG. 14-14 Mixed bacterial biofilm adhered to the tooth surface (Brown and Brenn staining, magnification ×1000).

form a community. Thus, *community* refers to a unified assemblage of populations that coexist and interact at a given habitat. The community and habitat are part of a larger system called an *ecosystem,* which can be defined as a functional self-supporting system that includes the microbial community and its environment. In summary, the following hierarchy becomes apparent: ecosystem, community, population, and the single individual (cell).

Populations perform functions that contribute to the overall community and maintain the ecologic balance of the ecosystem. Each population occupies a functional role *(niche)* within the community. There are a limited number of niches within the community for which populations must compete. More competent populations occupy the niches and displace those less competent. As discussed later, highly structured and spatially organized microbial communities may exhibit properties that are greater than the sum of the component populations. In reality, complex microbial communities have been shown to be endowed with the ability to confront and withstand the challenges imposed by the environment by creating a mosaic of microenvironments that enable the survival and growth of the community members.

Historically, microbiologists dealing with infectious diseases have faced periods of "reductionism" and "holism."[95] *Reductionism* is based on the idea that the whole can be understood by examining smaller and smaller pieces of it, that is, all complex systems can be completely understood in terms of their individual components. Through reductionist approaches, individual species are isolated from complex mixed communities and metabolically and genetically studied so as to allow understanding of the community by examining every single constituent. However, it has become quite apparent for the microbiota associated with many human infectious diseases that the whole is very often greater than the simple sum of its parts. This concept has prompted microbiologists to adopt a holistic approach to understand the community behavior associated with pathogenesis of many infectious diseases known to have a polymicrobial etiology. *Holism* holds that any component cannot be thoroughly understood except in their relation to the whole. The holistic theory has been largely employed in ecology: the interplay of the different parts composing the ecosystem will ultimately determine its properties.

It has been recognized that the biofilm (dental plaque) associated with caries and periodontal diseases represents a sophisticated community that exert functions essential for the biofilm architecture and physiology, with consequent pathogenetic implications. Recent evidence indicates that apical periodontitis can also develop as a result of collaborative activities of a biofilm community established in the root canal system.

Community profiling studies revealed that bacterial composition of the endodontic microbiota differs consistently between individuals suffering from the same disease.[32,108,168,229] This indicates that apical periodontitis has a heterogeneous etiology, where multiple bacterial combinations can play a role in disease causation. Interindividual variability is even more pronounced when different geographic locations are studied.[108,222] Moreover, community structure differs significantly between different disease forms (e.g., asymptomatic apical periodontitis versus acute apical abscess),[179,183,229] suggesting existence of a pattern associated with each form.

Biofilm and Bacterial Interactions

The community-forming ability can be regarded as essential for microbial survival in virtually all environments. Indeed, the majority of microorganisms in nature invariably grow and function as members of metabolically integrated communities, or biofilms.[35,110] *Biofilm* can be defined as a sessile multicellular microbial community characterized by cells that are firmly attached to a surface and enmeshed in a self-produced matrix of extracellular polymeric substance (EPS), usually a polysaccharide[35,47] (Fig. 14-14). The ability to form biofilms has been regarded as a virulence factor,[76] and biofilm infections account for an estimated 65% to 80% of bacterial infections that affect humans in the developed world.[34] Given its importance in varied aspects, there has been a high level of interest in the study of biofilm properties, not only in medical microbiology but also in different sectors of industrial and environmental microbiology.

Bacterial cells in biofilms form microcolonies (±15% by volume) that are embedded and nonrandomly distributed in

the EPS matrix (±85% by volume) and separated by water channels.[37,47,240,244] Microcolonies are usually shaped as "towers" or "mushrooms." Dental biofilms can reach up to 300 or more cell layers in thickness.[240] Individual microcolonies may consist of a single bacterial species but more frequently are composed of several different species in a mixed community.

As the biofilm matures on the surface, extracellular polysaccharides are continually synthesized to form an extracellular matrix that eventually may constitute as much as 85% of the volume of the biofilm.[34] Although the matrix is primarily composed of polysaccharides, it can also contain proteins and nucleic acids.[76] The matrix is not only important physically as part of the scaffold that determines the biofilm structure, but it is also biologically active and can retain nutrients, water, and essential enzymes within the biofilm.[5] The matrix can also protect the biofilm community from exogenous threats and may participate in adherence to the surface.

Community members form distinct populations or microcolonies separated by open water channels that traverse the biofilm matrix and create primitive circulatory systems.[36] Fluid in these channels carries substrate, end products of bacterial metabolism, and signal molecules involved in bacterial interactions.[20] Thus, vital nutrients and communication molecules can diffuse, and wastes can be washed out through these channels.

Microcolonies that form in the biofilm arise from surface colonization by planktonic (unattached) bacterial cells. During the early stages of biofilm formation, bacteria bind to many host proteins and coaggregate with other bacteria. These interactions lead to changes in growth rate, gene expression, and protein production. It has been demonstrated by proteomic techniques or DNA arrays that genes expressed by cells in biofilms differed by 20% to 70% from those expressed by the same cells growing in planktonic culture.[15,132,185] Thus, bacteria in biofilms adopt a radically different phenotype compared with their planktonic counterparts. Within biofilms, some bacteria also use sophisticated systems of cell-cell communication (quorum sensing) to coordinate gene expression. Phenotypic heterogeneity in biofilms is also observed as a result of exposure of microcolonies to a variety of gradients (e.g., oxygen tension, pH, osmolarity, type and amounts of nutrients, cell density), which contribute to form diverse microenvironments throughout the biofilm structure.

Biofilm Community Lifestyle

Many naturally occurring biofilms have a highly diverse microbiota. These multispecies biofilms are not merely passive bacterial assemblages that are stuck to surfaces; they are complex biologic systems formed by populations (microcolonies) that are not randomly distributed but are spatially and functionally organized throughout the community. Indeed, populations are strategically positioned for optimal metabolic interaction, and the resultant architecture favors the ecologic role of the community in the ecosystem. The properties displayed by a multispecies biofilm community are mostly dictated by the interactions between populations, which create novel physiologic functions that cannot be usually observed with individual components. As a result, biofilm communities have a collective physiology, responding in concert to environmental challenges.

The biofilm community lifestyle affords a number of advantages to colonizing bacteria, including establishment of a broad habitat range for growth, increased metabolic diversity and efficiency, enhanced possibilities for genetic exchanges and bacterial intercommunications (quorum-sensing systems), and protection from external threats (competing microorganisms, host defenses, antimicrobial agents, and environmental stress).[111]

Biofilm organizations can also result in enhanced pathogenicity. To cause disease, bacteria must adhere to host surfaces, obtain nutrients from the host and multiply, invade tissues, overcome or evade the host defenses, and induce tissue damage. A diverse range of virulence traits are required for these particular stages of the disease process, and it is highly probable that each will require the concerted action of bacteria in a community. Similarly, it is possible that certain species can have more than one role in disease, and different species can perform similar functions. This helps explain why communities with different bacterial composition can be found in different individuals with similar disease. In multispecies communities, a broad spectrum of relationships may arise between the component species, ranging from no effect or reduced pathogenicity to additive or synergistic pathogenic effects. Endodontic abscesses are examples of polymicrobial infections whereby bacterial species that individually have low virulence and are unable to cause disease can do so when in association with others as part of a mixed consortium (pathogenic synergism).[21,39]

Resistance to Antimicrobial Agents

From a clinical standpoint, biofilm increased resistance to antimicrobial agents is of special concern. Bacteria arranged in biofilms are considered more resistant to antibiotics than the same cells grown in planktonic state. The antibiotic concentration required to kill bacteria in the biofilm is about 100 to 1000 times higher than that needed to kill the same species in planktonic state.[109] Several possible mechanisms are involved with biofilm resistance to antimicrobials.

Biofilm Structure May Restrict Penetration of Antimicrobial Agents

The agent may adsorb to and even inhibit the bacteria at the biofilm surface, but cells deeply located in the biofilm may remain relatively unaffected. The matrix in biofilms can also bind and retain neutralizing enzymes at concentrations that could inactivate the antimicrobial agent.[243]

Altered Growth Rate of Biofilm Bacteria

Many antibiotics can freely penetrate the biofilm matrix, but cells are often still protected. The occurrence of starved bacteria entering the stationary phase in biofilms seems to be a significant factor in the resistance of biofilm populations to antimicrobials. Bacteria grow slowly under conditions of low availability of nutrients in an established biofilm and as a consequence are much less susceptible than faster-dividing cells. Most antibiotics require at least some degree of cellular activity to be effective. Therefore, bacterial cells in stationary phase might represent a general mechanism of antibiotic resistance in the biofilm.[76]

Presence of "Persister" Bacteria

Increased tolerance of some biofilms to antibiotics may be largely due to the presence of a subpopulation of specialized survivor cells known as *persisters*.[89] It remains unclear whether

these bacteria actually represent a distinct phenotype or are simply the most resistant cells within a population.[76]

APICAL PERIODONTITIS AS A BIOFILM-RELATED DISEASE

The evidence that apical periodontitis is a disease associated with polymicrobial biofilms comes mostly from in situ morphologic investigations.[25,121,125,126,152,153,154,186,223] These studies have observed that bacteria colonizing the root canal system of teeth with primary or posttreatment apical periodontitis usually formed sessile biofilm communities covering the walls of the main canal, apical ramifications, lateral canals, and isthmuses.

Although the concept of apical periodontitis as a biofilm-related disease has been built upon these observations, the prevalence of biofilms and their association with diverse presentations of apical periodontitis were only recently disclosed by Ricucci and Siqueira.[152] These authors evaluated the prevalence of biofilms in untreated teeth with primary apical periodontitis and treated teeth with posttreatment disease and looked for associations between biofilms and clinical/histopathologic conditions. Some of the most important findings of their study are as follows:

1. Intraradicular biofilms were generally observed in the apical segment of approximately 80% of the root canals of teeth with primary or posttreatment apical periodontitis.
2. Morphology of endodontic biofilms differed consistently from individual to individual (e.g., thickness, morphotypes, bacterial cells/extracellular matrix ratio).
3. Dentinal tubules underneath biofilms were often invaded by bacterial cells from the bottom of the biofilm community.
4. Biofilms were also commonly seen covering the walls of apical ramifications, lateral canals, and isthmuses.
5. Bacterial biofilms were more frequent in root canals of teeth with large apical periodontitis lesions. Because it takes time for apical periodontitis to develop and become radiographically visible, one can surmise that large lesions represent a longstanding pathologic process caused by an even "older" intraradicular infection. In longstanding infectious processes, involved bacteria may have had enough time and conditions to adapt themselves to the environment and set a mature and organized biofilm community. The fact that the apical root canal of teeth with large lesions harbors a large number of bacterial cells and species almost always organized in biofilms may help explain the long-held concept that treatment outcome is influenced by lesion size.[33,128]
6. The prevalences of intraradicular biofilms in teeth associated with apical cysts, abscesses, and granulomas were 95%, 83%, and 69.5%, respectively. Biofilms were significantly associated with epithelialized lesions. Because apical cysts develop as a result of epithelial proliferation in some granulomas,[102] it may be anticipated that the older the apical periodontitis lesion, the greater the probability of it becoming a cyst. Similar to teeth with large lesions, the age of the pathologic process may also help explain the high prevalence of biofilms in association with cysts.
7. Extraradicular biofilms were infrequent; they occurred in only 6% of the cases. Except for one case, they were always associated with intraradicular biofilms. All cases showing an extraradicular biofilm exhibited clinical symptoms. Thus, it seems that extraradicular infections in the form of biofilms or planktonic bacteria are not a common occurrence, are usually dependent on the intraradicular infection, and are more frequent in symptomatic teeth.
8. Bacteria were also seen in the lumen of the main canal, ramifications, and isthmuses as flocs and planktonic cells, either intermixed with necrotic pulp tissue or possibly suspended in a fluid phase. Bacterial flocs are sometimes regarded as "planktonic biofilms" and may originate from growth of cell aggregates/coaggregates in a fluid or they may have detached from biofilms.[77]

Some criteria have been proposed to establish a causal link between biofilms and a given infectious disease[77,134,152]:

1. Infecting bacteria are adhered to or associated with a surface.
2. Direct examination of the infected tissue shows bacteria forming clusters or microcolonies encased in an extracellular matrix.
3. The infection is generally confined to a particular site and although dissemination may occur, it is a secondary event.
4. The infection is difficult or impossible to eradicate with antibiotics in spite of the responsible microorganisms being susceptible to killing in the planktonic cell state.
5. Ineffective host clearance is evident, as suggested by the location of bacterial colonies in areas of the host tissue associated with host inflammatory cells. Accumulation of polymorphonuclear neutrophils and macrophages surrounding bacterial aggregates/coaggregates in situ considerably increases the suspicion of biofilm involvement with disease causation.
6. Elimination or significant disruption of the biofilm structure and ecology leads to remission of the disease process.

Based on findings from the Ricucci and Siqueira's study,[152] apical periodontitis can be considered to fulfill five of the six criteria. Bacterial aggregates/coaggregates are observed adhered to or at least associated with the dentinal root canal walls (criterion 1). Bacterial colonies are often seen encased in an amorphous extracellular matrix (criterion 2). Endodontic biofilms are frequently confined to the root canal system, in only a few cases extending to the external root surface, but dissemination through the lesion never occurred (criterion 3). In the great majority of cases, biofilms are directly faced by an accumulation of inflammatory cells, especially polymorphonuclear neutrophils (criterion 5).

As for criterion 4, it is widely known that intraradicular endodontic infections cannot be effectively treated by systemic antibiotic therapy, even though most endodontic bacteria in the planktonic cell state are susceptible to currently used antibiotics.[13,66,90] The lack of efficacy of systemic antibiotics against intraradicular infections is mainly because the drug does not reach endodontic bacteria that are located in an avascular necrotic space. The recognition of biofilms as the main mode of bacterial establishment in the root canal system further strengthens the explanations for the lack of antibiotic effectiveness against endodontic infections. Finally, there is a clear potential for fulfillment of criterion 6, because biofilms are

frequently observed in canals of treated teeth with posttreatment apical periodontitis,[154] whereas teeth with a successful outcome show no biofilm infection of the root canal.[149]

METHODS FOR MICROBIAL IDENTIFICATION

The endodontic microbiota has been traditionally investigated by microbiologic culture methods. Culture is the process of propagating microorganisms in the laboratory by providing them with the required nutrients and proper physicochemical conditions, including temperature, moisture, atmosphere, salt concentration, and pH.[237] Essentially, culture analyses involve the following steps: sample collection and transport, dispersion, dilution, cultivation, isolation, and identification.[251] Endodontic samples are collected and transported to the laboratory in a viability-preserving, nonsupportive, anaerobic medium. They are then dispersed by sonication or vortex mixing, diluted, distributed onto various types of agar media, and cultivated under aerobic or anaerobic conditions. After a suitable period of incubation, individual colonies are subcultivated and identified on the basis of multiple phenotype-based aspects, including colony and cellular morphology, gram-staining pattern, oxygen tolerance, comprehensive biochemical characterization, and metabolic end-product analysis by gas-liquid chromatography. The outer cellular membrane protein profile as examined by gel electrophoresis, fluorescence under ultraviolet light, and susceptibility tests to selected antibiotics can be needed for identification of some species.[52] Marketed packaged kits that test for preformed enzymes have also been used for rapid identification of several species.

Culture analyses of endodontic infections have provided a substantial body of information about the etiology of apical periodontitis, composition of the endodontic microbiota in different clinical conditions, effects of treatment procedures in microbial elimination, susceptibilities of endodontic microorganisms to antibiotics, and so on. Advantages and limitations of culture methods are listed in Box 14-1; however, some important limitations of culture methods make a comprehensive analysis of the endodontic microbiota difficult to achieve.

The difficulties in culturing or identifying many microbial species are of special concern. Unfortunately, not all microorganisms can be cultivated under artificial conditions, and this is simply because the nutritional and physiologic needs of most microorganisms are still unknown. Investigations of many aquatic and terrestrial environments using culture-independent methods have revealed that the cultivable members of these systems represent less than 1% of the total extant population.[6,276] Furthermore, 50% to 80% of bacterial species composing the microbiota associated with diverse human sites, including the oral cavity, represent unknown and still uncultivated bacteria.[1,2,44,49,94,138,146,245]

That a given species has not been cultivated does not imply that this species will remain indefinitely impossible to cultivate. Myriad obligate anaerobic bacteria were uncultivable in the early 1900s, but further developments in anaerobic culturing techniques have to a large extent helped to solve this problem. It must be assumed that no single method or culture medium is suitable for isolating the vast diversity of microorganisms present in most environments.[70] Because we remain relatively unaware of the requirements for many bacteria to grow, identification methods not based on cultivability are required.

BOX 14-1

Advantages and Limitations of Culture Methods

Advantages	Limitations
Broad-range nature, identification of unexpected species	Impossibility of culturing a large number of extant bacterial species
Allow quantification of all major viable cultivable microorganisms in samples	Not all viable bacteria can be recovered
Allow determination of antimicrobial susceptibilities of isolates	Once isolated, bacteria require identification using a number of techniques
Physiologic studies are possible	Misidentification of strains with ambiguous or aberrant phenotypic behavior
Pathogenicity studies are possible	Low sensitivity
Widely available	Strict dependence on the mode of sample transport
	Samples require immediate processing
	Costly, time consuming, and laborious, as for cultivation of anaerobes
	Specificity is dependent on experience of microbiologist
	Extensive expertise and specialized equipment needed to isolate anaerobes
	Takes several days to weeks to identify most anaerobes

In some situations, even the successful cultivation of a given microorganism does not necessarily mean that this microorganism can be successfully identified. Culture-dependent identification is based on phenotypic traits observed in reference strains, with predictable biochemical and physical properties under optimal growth conditions. However, many phenotype-related factors can lead to difficulties in identification and even to misidentification.[14,18,210,260] As a consequence of all these factors, phenotype-based identification does not always allow an unequivocal identification.

To sidestep the limitations of culturing, tools and procedures based on molecular biology have become available and have substantially improved the ability to achieve a more realistic description of the microbial world without the need for cultivation (Fig. 14-15). Molecular technology has also been applied to reliably identify cultivated bacteria, including strains with ambiguous or aberrant phenotypic behavior, rare isolates, poorly described or uncharacterized bacteria, and newly named species.[19,48,144,211,241,258]

Molecular approaches for microbial identification rely on certain genes that contain revealing information about the microbial identity. Of the several genes that have been chosen as targets for bacterial identification, the 16S rRNA gene (or 16S rDNA) has been the most widely used because it is universally distributed among bacteria, is long enough to be highly informative and short enough to be easily sequenced, possesses conserved and variable regions, and affords reliability for inferring phylogenetic relationships.[279] Similarly, the 18S rRNA gene of fungi and other eukaryotes has also been used extensively to identify these organisms.

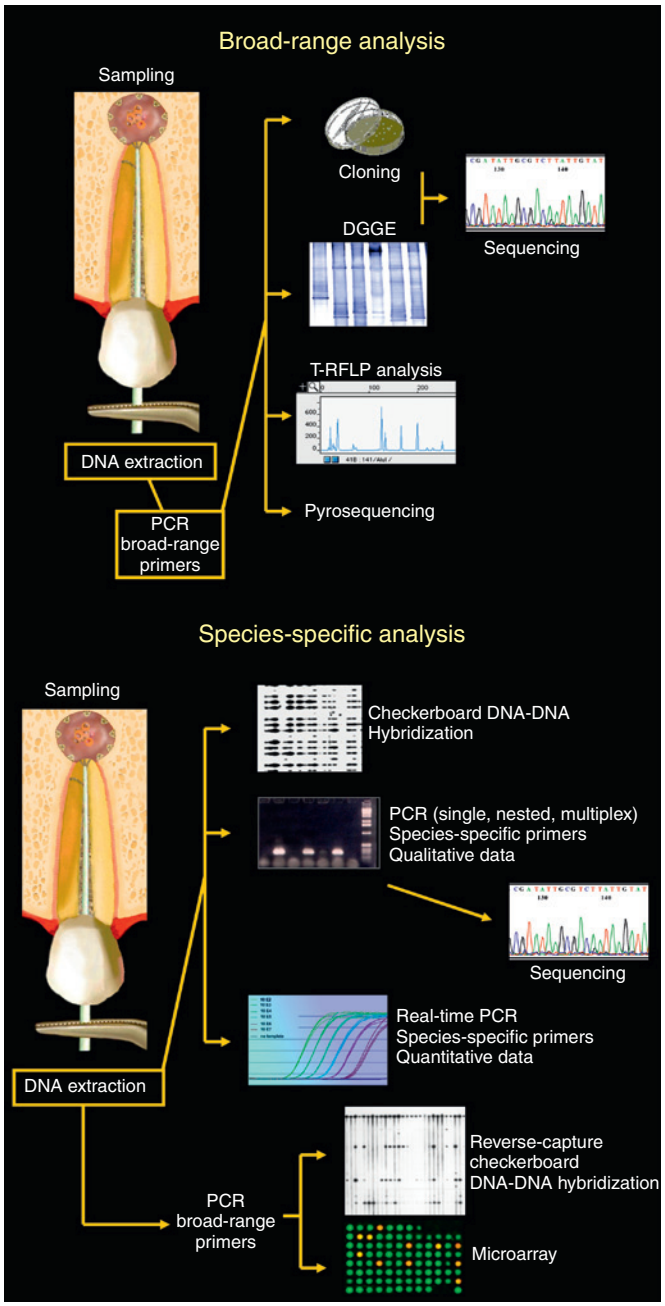

FIG. 14-15 Molecular biology methods used (or with potential to be used) in the study of endodontic infections. The choice for a particular technique will depend on the type of analysis to be performed. Some methods can be used for detection of target microbial species or groups and others for a broader analysis of the microbiota.

Data from 16S rRNA gene sequences can be used for accurate and rapid identification of known and unknown bacterial species, using techniques that do not require cultivation. The 16S RNA gene of virtually all bacterial species in a given environment, including still uncultivated and uncharacterized bacteria, can be amplified by polymerase chain reaction (PCR) using broad-range (or universal) primers that are complementary to conserved regions of this gene. Sequencing of the variable regions flanked by the broad-range primers will provide information for accurate bacterial identification. Primers or probes that are complementary to variable regions can also be designed to detect specific target species directly in clinical samples.

Many molecular methods for the study of microorganisms exist; the choice of a particular approach depends on the questions being addressed.[218] Broad-range PCR followed by cloning and sequencing can be used to unravel the breadth of microbial diversity in a given environment. Bacterial community structures can be analyzed via pyrosequencing technology and by fingerprinting techniques such as denaturing gradient gel electrophoresis (DGGE) and terminal restriction fragment length polymorphism (T-RFLP). Fluorescence in situ hybridization (FISH) can measure the abundance of target species and provide information on their spatial distribution in tissues. Among other applications, DNA-DNA hybridization arrays (checkerboard techniques, DNA microarrays), species-specific single PCR, nested PCR, multiplex PCR, and quantitative real-time PCR can be used to survey large numbers of clinical samples for the presence of target species. Variations in PCR technology can also be used to type microbial strains. As with any other method, molecular methods have their own advantages and limitations (Box 14-2).

BOX 14-2

Advantages and Limitations of Molecular Biology Methods

Advantages	Limitations
Detect both cultivable and as-yet-uncultivated species or strains	Most assays are qualitative or semiquantitative (exceptions: real-time PCR)
High specificity and accurate identification of strains with ambiguous or aberrant phenotypic behavior	Most assays only detect one species or a few different species at a time (exceptions: broad-range PCR, checkerboard, microarray)
Detect species directly in clinical samples	Most assays detect only the target species and fail to detect unexpected species (exception: broad-range PCR)
High sensitivity	Some assays can be laborious and costly (e.g., broad-range PCR)
Rapid; most assays take no more than minutes to a few hours to identify a microbial species	Biases in broad-range PCR introduced by homogenization procedures, preferential DNA amplification, and differential DNA extraction
Do not require carefully controlled anaerobic conditions during sampling and transportation	Hybridization assays using whole genome probes detect only cultivable species
Can be used during antimicrobial treatment	Can be very expensive
Anaerobic handling and expertise not required	
Samples can be stored frozen for later analysis	
DNA can be transported easily between laboratories	
Detect dead microorganisms	

The Five Generations of Endodontic Microbiology Studies

Microbiologic studies for identification of the species participating in endodontic infections can be chronologically

TABLE 14-1

Generations of Studies for Microbiologic Identification in Endodontic Infections

Study Generation	Identification Method	Nature	Description and Findings
First	Culture	Open ended (broad range)	Revealed many cultivable species in association with apical periodontitis
Second	Molecular methods (e.g., PCR and its derivatives, original checkerboard assay)	Closed ended (species specific)	Target cultivable bacteria Confirmed and strengthened data from first generation Allowed inclusion of some culture-difficult species in the set of candidate endodontic pathogens
Third	Molecular methods (e.g., PCR-cloning-sequencing, T-RFLP)	Open ended (broad range)	Allowed a more comprehensive investigation of the bacterial diversity in endodontic infections Not only cultivable species but also as-yet-uncultivated and uncharacterized bacteria were identified
Fourth	Molecular methods (e.g., PCR, microarrays, reverse-capture checkerboard)	Closed ended (species specific)	Target cultivable and as-yet-uncultivated bacteria Large-scale clinical studies to investigate prevalence and association of species/phylotypes with endodontic infections
Fifth	Molecular methods (e.g., pyrosequencing)	Open ended (broad range)	Permit a deep-coverage and more comprehensive analysis of the diversity of endodontic infections

divided into five generations on the basis of the different strategic approaches used.[73] These generations are detailed in Table 14-1.

Impact of Molecular Methods in Endodontic Microbiology

Culture studies (first generation) identified a set of species thought to play an important role in the pathogenesis of apical periodontitis.[189,253] Further, not only have findings from culture-based methods been confirmed, but they have also been significantly supplemented with those from culture-independent molecular biology techniques, which constitute the other four generations of endodontic microbiology studies.[216] Molecular methods have confirmed and strengthened the association of many cultivable bacterial species with apical periodontitis and have also revealed new suspected endodontic pathogens.[212] The list of candidate pathogens has expanded to include culture-difficult species or as-yet-uncultivated bacteria that had never been found in endodontic infections by culturing approaches. The results from molecular studies impact remarkably on the knowledge of bacterial diversity in endodontic infections. More than 400 different bacterial species have already been detected in different types of endodontic infections.[216] Of these, about 45% were exclusively reported by molecular biology studies, compared with 32% detected by culture studies alone.[216] Twenty-three percent of the total bacterial species richness has been detected by application of both culture and molecular studies (Fig. 14-16). As a consequence, it becomes quite evident that the endodontic microbiota has been refined and redefined by molecular methods.[212]

TYPES OF ENDODONTIC INFECTIONS

Endodontic infections can be classified according to the anatomic location as intraradicular or extraradicular infection. *Intraradicular infection* is caused by microorganisms colonizing the root canal system and can be subdivided into three categories according to the time microorganisms entered the root canal system: *primary infection,* caused by microorganisms that initially invade and colonize the necrotic pulp tissue (initial or "virgin" infection); *secondary infection,* caused by microorganisms not present in the primary infection but introduced in the root canal at some time after professional intervention (i.e., *secondary* to intervention); and *persistent infection,* caused by microorganisms that were members of a primary or secondary infection and in some way resisted intracanal antimicrobial procedures and were able to endure periods of nutrient deprivation in treated canals. *Extraradicular infection* in turn is characterized by microbial invasion of the inflamed periradicular tissues and is a sequel to the intraradicular infection. Extraradicular infections can be dependent on or independent of the intraradicular infection.

DIVERSITY OF THE ENDODONTIC MICROBIOTA

Microbiota is a collective term for microorganisms and should replace terms such as *flora* and *microflora,* which perpetuate an outdated classification of microorganisms as plants.[43] *Diversity* refers to the number of different species present (richness) and their relative abundance (evenness) in a given ecosystem.[83]

The oral cavity harbors one of the highest accumulations of microorganisms in the body. Even though viruses, archaea, fungi, and protozoa can be found as constituents of the oral microbiota, bacteria are by far the most dominant inhabitants of the oral cavity. There are an estimated 10 billion bacterial cells in the oral cavity,[116] and culture-independent studies (microscopy and molecular biology technologies) have shown that over 50% to 60% of the oral microbiota still remains to be cultivated and fully characterized.[2,44,239] A high diversity of bacterial species has been revealed in the oral cavity by culturing approaches,[122] but application of molecular biology methods to the analysis of the bacterial diversity has revealed a still broader and more diverse spectrum of extant oral bacteria.[139] More than 1000 bacterial species/phylotypes have

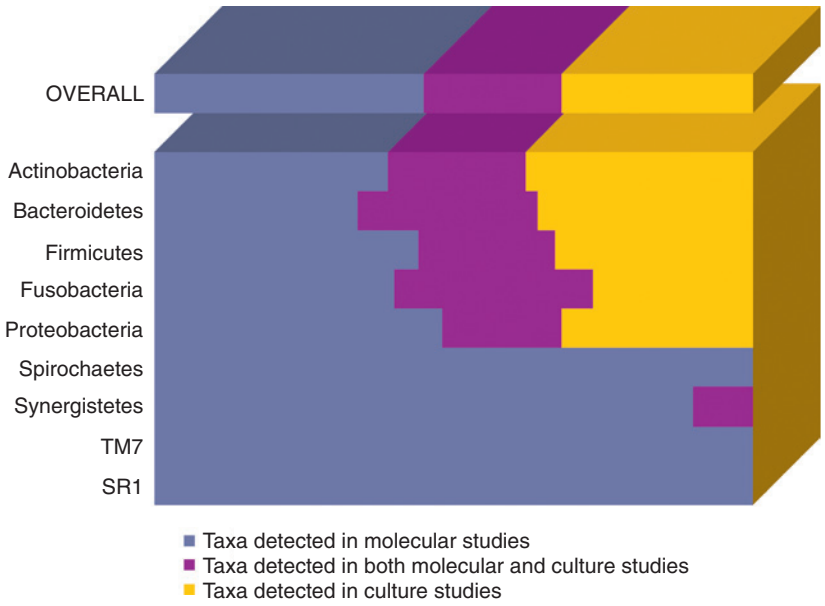

FIG. 14-16 Percentage distribution of bacterial species/phylotypes found in endodontic infections according to the detection method. Data refer to the percentage of species or phylotypes overall or in each of the nine phyla that have endodontic representatives.

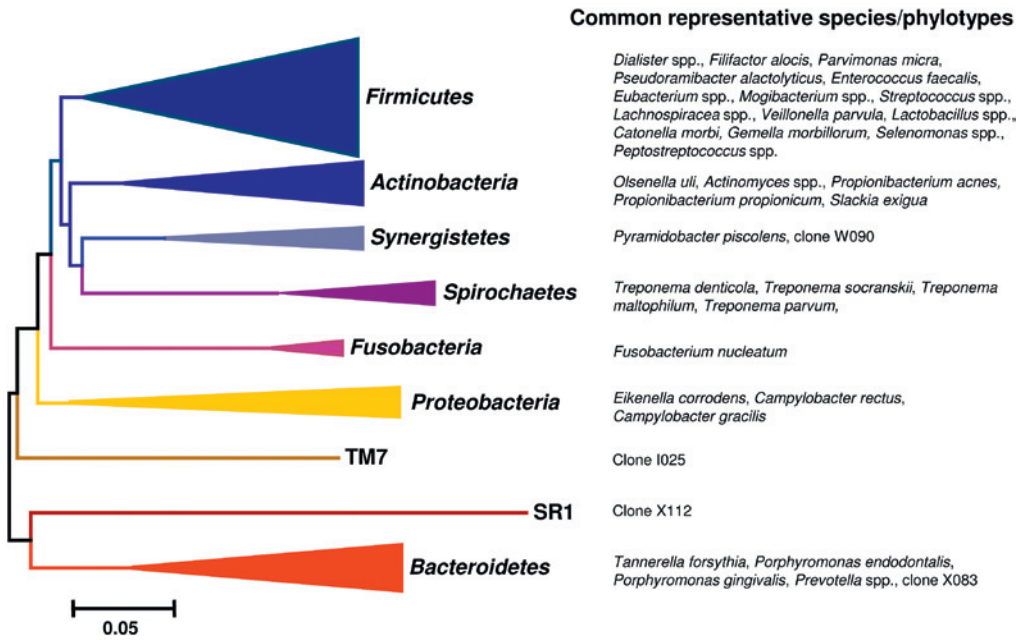

FIG. 14-17 Bacterial phyla with their respective endodontic representatives. *Right:* Example species or phylotypes for each phylum.

been found in the human oral cavity,[44] but advanced DNA pyrosequencing studies have indicated that this number may still have been largely underestimated.[4,72,88] Modern anaerobic culture and sophisticated molecular biology techniques have demonstrated that, at a broader taxonomic level, endodontic bacteria fall into nine phyla—namely, Firmicutes, Bacteroidetes, Spirochaetes, Fusobacteria, Actinobacteria, Proteobacteria, Synergistetes, TM7, and SR1[123,148,162,178-180,213,216] (Fig. 14-17). However, data from studies using pyrosequencing technology reveal that several other phyla may have been overlooked by previous identification techniques.[82,100,183,198,202] Fungi and archaea are types of microorganisms that have been only occasionally found in endodontic infections.

Endodontic infections develop in a previously sterile place that does not contain a normal microbiota. Any species found has the potential to be an endodontic pathogen or at least play a role in the ecology of the endodontic microbial community. Culture and molecular studies reveal only prevalence of species; consequently, only association can be inferred. Causation is usually surmised on the basis of both frequency of detection and potential pathogenicity (in animal models or association with other human diseases), and several species

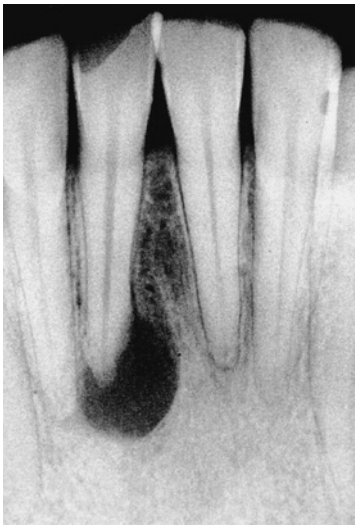

FIG. 14-18 Apical periodontitis due to primary intraradicular infection. Pulp is necrotic and lesion size is usually directly proportional to complexity of the microbiota involved.

have emerged as candidate endodontic pathogens. The following sections discuss specific aspects of each type of endodontic infection.

PRIMARY INTRARADICULAR INFECTION

Microbial Composition and Diversity

Primary intraradicular infection is infection of the necrotic pulp tissue (Fig. 14-18). It occurs in untreated teeth and is the cause of primary apical periodontitis. Participating microorganisms may be involved in earlier stages of pulp invasion (usually via caries) that culminated in inflammation and further necrosis, or they can be latecomers that took advantage of the environmental conditions in the root canal after pulp necrosis.

Primary infections are characterized by a mixed (multispecies) community conspicuously dominated by anaerobic bacteria. The number of bacterial cells may vary from 10^3 to 10^8 per root canal.[17,180,231,251,180,226,250,269] Molecular studies have disclosed a mean of 10 to 20 species/phylotypes per infected canal.[123,148,162,212,229] Canals of teeth with associated sinus tracts may exhibit a number of species close to the top of this range.[162] The size of apical periodontitis lesion has been shown to be proportional to the number of bacterial species and cells in the root canal.[162,227,250] A molecular study[162] demonstrated that the number of taxa per canal was clearly in direct proportion to the lesion size: small lesions (< 5 mm) harbored about 12 taxa, lesions from 5 to less than 10 mm harbored 16 taxa, and lesions over 10 mm harbored about 20 species. Some canals associated with large lesions may harbor even more than 40 taxa.[162] Therefore, the larger the lesion, the higher the bacterial diversity and density in the canal.

The most prevalent named bacterial species detected in primary infections, including abscessed cases, belong to diverse genera of gram-negative bacteria (i.e., *Fusobacterium, Dialister, Porphyromonas, Prevotella, Tannerella, Treponema, Pyramidobacter*, *Campylobacter,* and *Veillonella*) and gram-positive (*Parvimonas, Filifactor, Pseudoramibacter, Streptococcus, Propionibacterium, Olsenella, Actinomyces, Peptostreptococcus,* and *Eubacterium*).[11,60,61,75,90,123,148,162,164,178,179,212,227,230,231,252,250,270] Bacterial prevalence in primary infections may vary from study to study as a function of several factors, such as sensitivity and specificity of the detection and identification methods, sampling technique, geographic location, and accuracy or divergence in clinical diagnosis and disease classification. Even so, the species most frequently detected are expected to be the same in most well-conducted studies. Figs. 14-19 to 14-21 display the most frequently detected species associated with asymptomatic apical periodontitis, symptomatic apical periodontitis, and acute apical abscesses, as revealed by studies from the authors' group using a highly sensitive molecular biology technique.

About 40% to 66% of the endodontic microbiota in primary infections is composed of species still uncultivated.[123,148,179] As for their abundance in these infections, as-yet-uncultivated phylotypes corresponded to about 40% of the clones sequenced.[179] Molecular studies investigating the breadth of bacterial diversity in infected root canals have disclosed the occurrence of uncultivated phylotypes belonging to several genera, including *Dialister, Prevotella, Solobacterium, Olsenella, Fusobacterium, Treponema, Eubacterium, Megasphaera, Veillonella,* and *Selenomonas,* as well as phylotypes related to the family *Lachnospiraceae* or the TM7 and Synergistetes phyla.[123,148,160,161,172,178,179,182,213,221] Some uncultivated phylotypes can even be among the most prevalent bacteria in primary intraradicular infections, and others may be associated with pain.[179] *Bacteroidetes* clone X083 is one of the most prevalent phylotypes found in endodontic infections.[162-164,219] Detection of as-yet-uncultivated phylotypes in samples from endodontic infections suggests that they can be previously unrecognized bacteria that play a role in the pathogenesis of different forms of apical periodontitis. The fact that they have not yet been cultivated and phenotypically characterized does not mean that they are not important.

Symptomatic Infections

Symptomatic apical periodontitis and acute apical abscesses are typical examples of endodontic infections causing severe symptoms. In these cases, the infection is located in the canal, but it has also reached the periradicular tissues and, in abscessed cases, can spread to other anatomic spaces. Acute apical abscesses are caused by bacteria that egress from the infected root canal and invade the periradicular tissues to establish an extraradicular infection and evoke purulent inflammation. Clinically, the disease leads to pain or swelling and has the potential to diffuse to sinuses and other fascial spaces of the head and neck to form a cellulitis or other complications (Fig. 14-22). The microbiota involved in endodontic abscesses is mixed and dominated by anaerobic bacteria[41,90,96,155,179,217,229] (see Fig. 14-19). Direct comparisons using molecular technology reveal an average of 12 to 18 taxa per abscess case, compared with 7 to 12 taxa present in root canals of teeth with asymptomatic lesions.[179,229] Uncultivated phylotypes constitute approximately 40% of the taxa found in abscesses and collectively represent more than 30% of the 16S rRNA gene sequences retrieved in clone libraries.[179]

Whereas microbial causation of apical periodontitis is well established, there is no strong evidence disclosing specific involvement of a single species with any particular sign or symptom of apical periodontitis. Some gram-negative anaerobic bacteria have been suggested to be involved with

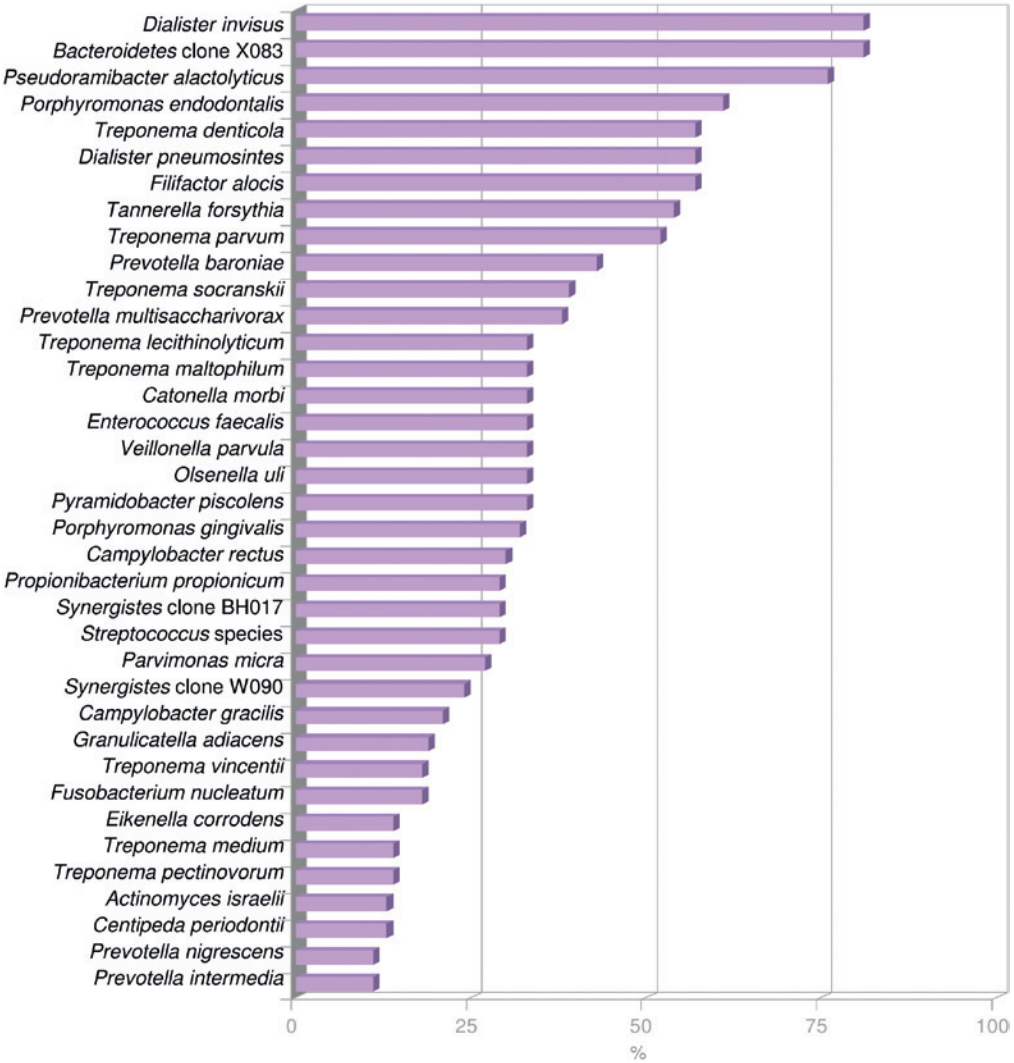

FIG. 14-19 Prevalence of bacteria detected in primary infections of teeth with asymptomatic (chronic) apical periodontitis. Data from the authors' studies using a taxon-specific nested-polymerase chain reaction protocol. (From references 159, 168, 211, and 210.)

symptomatic lesions,[67,71,169,179,250,266,281] but the same species may also be present in somewhat similar frequencies in asymptomatic cases,[11,61,75,85,230,231] so factors other than the mere presence of a given putative pathogenic species may play a role in the etiology of symptomatic endodontic infections.[193,199] These factors include differences in virulence ability among strains of the same species, bacterial interactions resulting in additive or synergistic effects among species in mixed infections, number of bacterial cells (infectious load), environmental cues regulating expression of virulence factors, host resistance; and concomitant herpesvirus infection. Association of some or all of these factors (instead of an isolated event) is likely to determine the occurrence and intensity of symptoms.[193,199]

Molecular studies using DGGE, T-RFLP, or pyrosequencing analysis have revealed that the structure of the endodontic bacterial communities in symptomatic teeth, including abscess cases, is significantly different from that of asymptomatic teeth.[179,183,229] Differences are represented by different dominant species in the communities and larger numbers of species in symptomatic cases. Differences in the type and load of dominant species and the resulting bacterial interactions may affect virulence of the whole bacterial community. Indeed, it has been shown that different species combinations may result in different outcomes because of the network of interactions.[170] The possibility exists that bacterial interactions result in communities that are more or less aggressive and consequently can cause host responses of corresponding intensity. This is consistent with the concept that the entire bacterial community is the unit of pathogenicity.

Geographic Influence

Findings from laboratories in different countries are often quite different regarding the prevalence of the species involved in endodontic infections. Although these differences may be attributed to variations in identification methodologies, a geographic influence in the composition of the root canal microbiota has been suspected. Studies using molecular biology techniques directly compared the endodontic microbiota of patients residing in distinct geographic locations and suggested that significant differences in the prevalence of some important species can actually exist.[10,157,204] In a more holistic approach, analysis of the bacterial community profiles of the microbiota

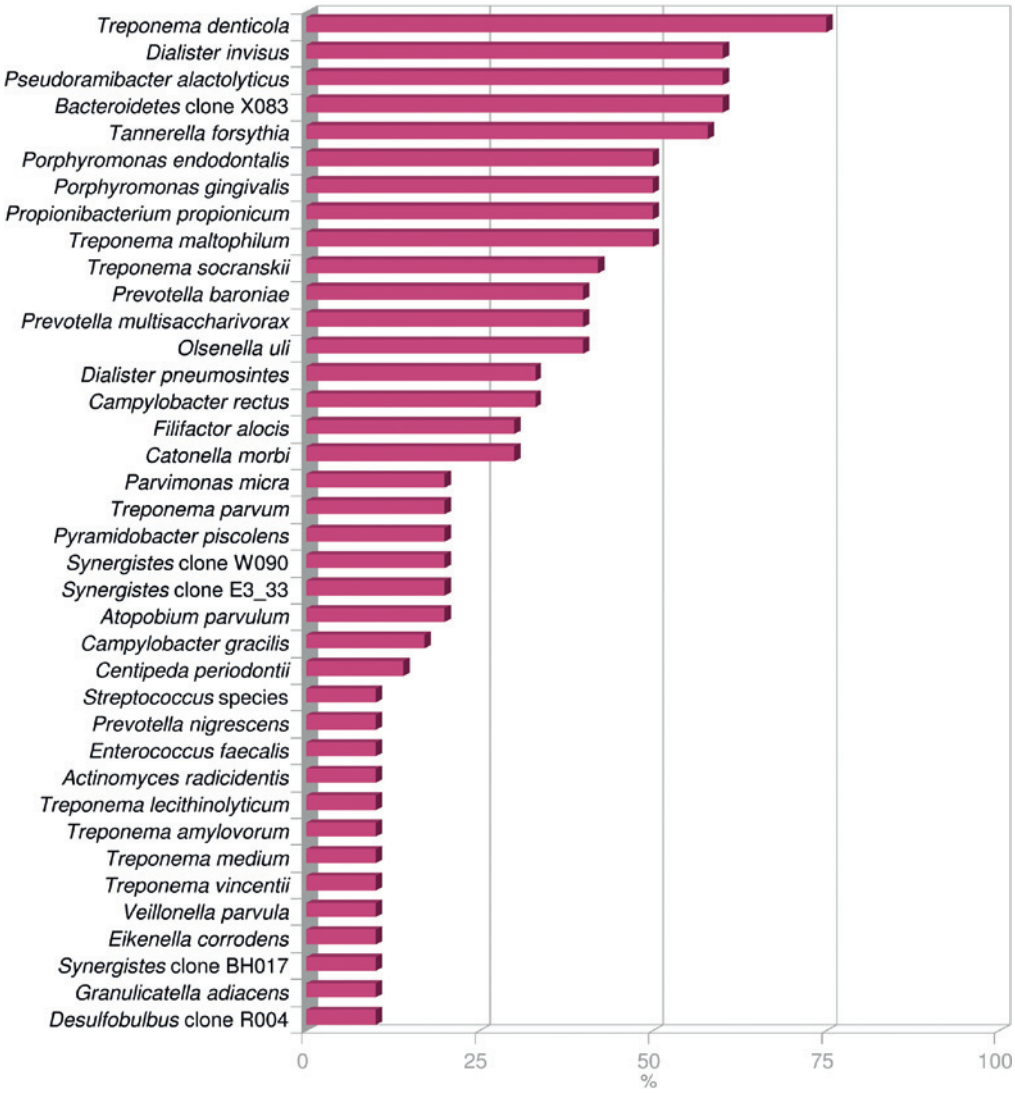

FIG. 14-20 Prevalence of bacteria detected in primary infections of teeth with symptomatic (acute) apical periodontitis. Data from the authors' studies using a taxon-specific nested-polymerase chain reaction protocol. (From references 159, 168, 211, and 210.)

associated with endodontic infections from different countries also revealed a geography-related pattern, with several species being exclusive for each location and others shared by the locations showing great differences in prevalence.[108,222] The factors that can lead to differences in the composition of the endodontic microbiota and the impact of these differences on therapy, particularly in abscessed cases requiring systemic antibiotic therapy, remain elusive.

Microbial Ecology and the Root Canal Ecosystem

A root canal with necrotic pulp provides a space for bacterial colonization and affords bacteria a moist, warm, nutritious, and anaerobic environment, which is by and large protected from the host defenses because of lack of active blood circulation in the necrotic pulp tissue. Also, the root canal walls are nonshedding surfaces conducive to persistent colonization and formation of complex communities. The necrotic root canal might be considered a fertile environment for bacterial growth, with colonization not being a difficult task for virtually all oral bacterial species. Although a large number of bacterial species (about 100 to 200) can be found in the oral cavity of a particular individual,[139] only a limited assortment of these species (about 10 to 20) is consistently selected out for growth and survival within a root canal containing necrotic pulp tissue from the same individual. This indicates that ecologic determinants operate in the necrotic canal and dictate which species will succeed in colonizing this previously sterile environment. The major ecologic factors that determine the composition of the root canal microbiota include oxygen tension, type and amount of available nutrients, and bacterial interactions. Other factors such as temperature, pH, and receptors for adhesins may also be involved.

The root canal infection is a dynamic process, and different bacterial species apparently dominate at different stages.[259] Shifts in the composition of the microbiota are largely due to changes in environmental conditions, particularly in regard to oxygen tension and nutrient availability. In the initial phases of the pulpal infectious process, facultative bacteria

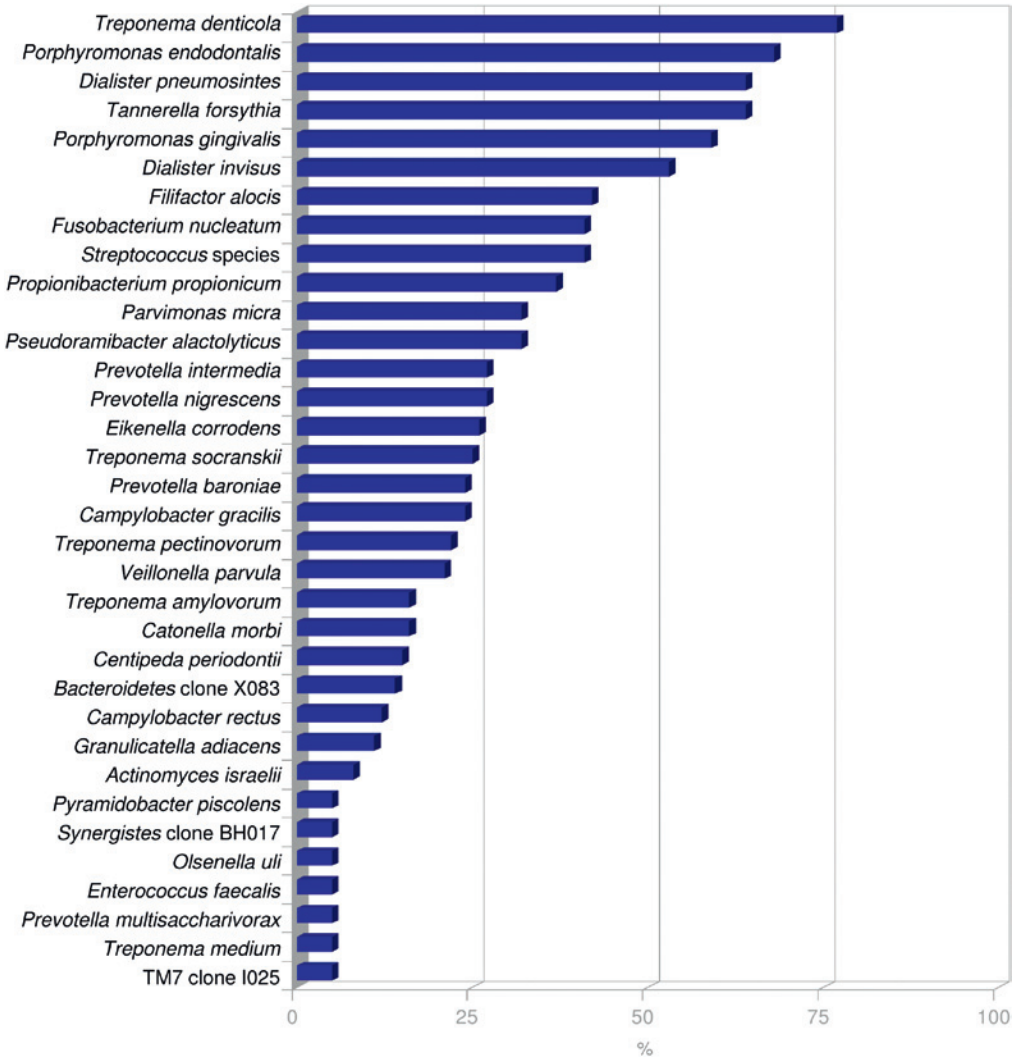

FIG. 14-21 Prevalence of bacteria detected in primary infections of teeth with acute apical abscesses. Data from the authors' studies using a taxon-specific nested-polymerase chain reaction protocol. (From references 159, 168, 211, and 210.)

FIG. 14-22 Acute apical abscess. The infection has spread to other anatomic spaces to form cellulitis. (Courtesy Dr. Henrique Martins.)

predominate.[54] After a few days or weeks, oxygen is depleted within the root canal as a result of pulp necrosis and consumption by facultative bacteria. Further oxygen supply is interrupted with loss of blood circulation in the necrotic pulp. An anaerobic milieu develops and is highly conducive to the survival and growth of obligate anaerobic bacteria. With the passage of time, anaerobic conditions become even more pronounced, particularly in the apical third of the root canal; as a consequence, anaerobes will dominate the microbiota, outnumbering facultative bacteria (Fig. 14-23).

The main sources of nutrients for bacteria colonizing the root canal system include (1) the necrotic pulp tissue, (2) proteins and glycoproteins from tissue fluids and exudate that seep into the root canal system via apical and lateral foramina, (3) components of saliva that may coronally penetrate into the root canal, and (4) products of the metabolism of other bacteria. Because the largest amount of nutrients is available in the main canal, the most voluminous part of the root canal system, most of the infecting microbiota (particularly fastidious anaerobic species) is expected to be located in this region. Bacterial species that can best utilize and

compete for nutrients in the root canal system will succeed in colonization.

In addition to being influenced by variations in oxygen levels, shifts in the composition of the microbiota colonizing the root canal system can also be dependent upon the dynamics of nutrient utilization. Saccharolytic species dominate the very early stages of the infectious process but are soon outnumbered by asaccharolytic species, which will dominate later stages.[254] Even though the necrotic pulp tissue can be regarded as a finite source of nutrients to bacteria (given the small volume of tissue that is progressively degraded), induction of periradicular inflammation guarantees a sustainable source of nutrients, particularly in the form of proteins and glycoproteins present in the exudate that seep into the canal. At this stage of the infectious process, bacteria that have a proteolytic capacity or establish a cooperative interaction with those that can utilize this substrate in the metabolism, start to dominate. Therefore, as the infectious process reaches the stage of induction of periradicular inflammation, proteins become the principal nutrient source, particularly in the apical part of the canal, favoring the establishment of anaerobic species that utilize peptides or amino acids in their metabolism (see Fig. 14-23).

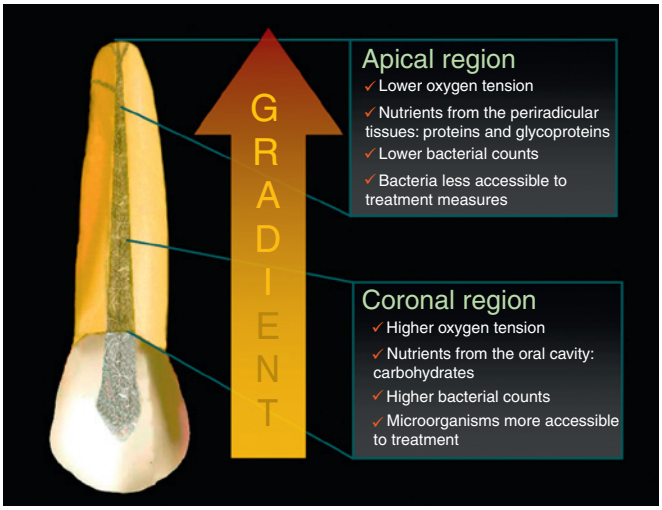

FIG. 14-23 Ecologic conditions in different areas of the root canal. A gradient of oxygen tension and nutrients (type and availability) is formed. Consequently, the microbiota residing in different parts can also differ in diversity, density, and accessibility to treatment procedures.

Because primary endodontic infections are usually characterized by mixed communities, different bacterial species are in close proximity with one another, and interactions become inevitable. Thus, establishment of certain species in the root canal is also influenced by interactions with other species. In this regard, early colonizers play an important role in dictating which species will live along with them in the community. Bacterial interactions can be positive or negative. Positive interactions enhance the survival capacity of the interacting bacteria and enable different species to coexist in habitats where neither could exist alone. For instance, interbacterial nutritional interactions are important ecologic determinants that result in higher metabolic efficiency of the whole community. Nutritional interactions are mainly represented by food webs, including utilization of metabolic end products from one species by another and bacterial cooperation for the breakdown of complex host-derived substrates. Fig. 14-24 displays a complex array of interbacterial nutritional interactions that can take place in infected root canals, where the growth of some species can be dependent on products of the metabolism of other species. Moreover, one species can provide growth conditions favorable to another—for example, by reducing oxygen tension in the environment and favoring the establishment of anaerobes, or by releasing some proteinases that can provide protection from host defenses. Negative interactions in turn act as feedback mechanisms that limit population densities. Examples include competition (for nutrients and space) and amensalism (when one species produces a

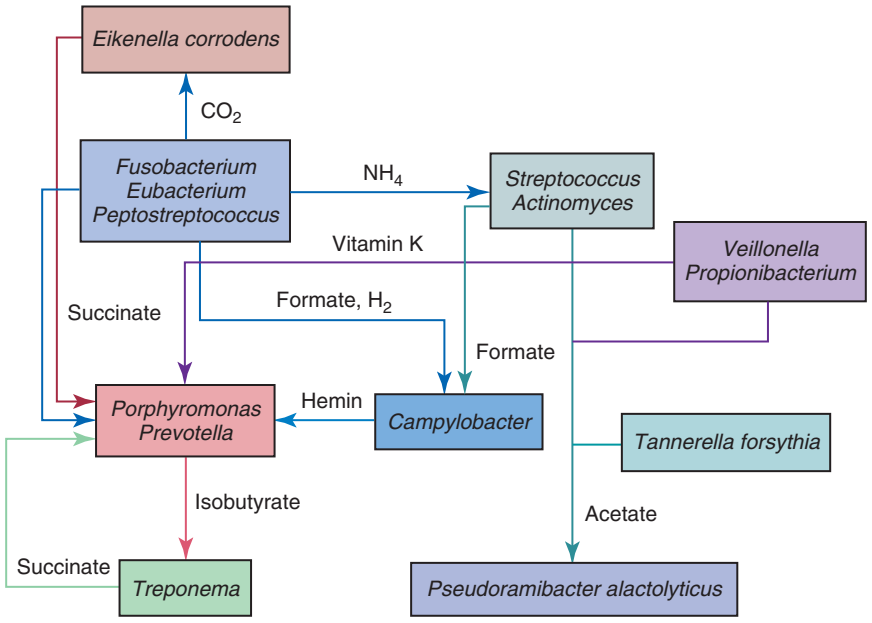

FIG. 14-24 Interbacterial nutritional interactions that can take place in infected root canals where growth of some species can be dependent on products of metabolism of other species.

substance that inhibits another species, including antibiotics such as penicillin).

Many species adhere directly to host surfaces, whereas other species adhere to bacteria already attached to the surface. The latter is called *coaggregation* and is a highly specific phenomenon with regard to the partners involved.[93] A given pair of species can attach to each other by means of specific receptor-adhesin interactions, which are usually lectin-like interactions (attachment of a specific protein on the surface of one species to a specific carbohydrate on the surface of the other). Coaggregation can favor colonization of host surfaces and also facilitate metabolic interactions between the partners. Coaggregation has been demonstrated for several pairs of bacterial taxa found in endodontic infections.[91]

Other Microorganisms in Endodontic Infections

Fungi

Fungi are eukaryotic microorganisms that may colonize the oral cavity, especially *Candida* species, but they have been only occasionally detected in primary intraradicular infections.[51,97,119,225] One molecular study[12] has, however, reported the occurrence of *C. albicans* in 21% of samples from primarily infected canals. Fungi are more frequently detected in canals of teeth with posttreatment disease (discussed later).

Archaea

Archaea consist of a highly diverse group of prokaryotes distinct from bacteria. Members of this domain have been traditionally recognized as extremophiles, but some of these microorganisms have also been found to flourish in nonextreme environments, including the human body.[50] To date, no member of the *Archaea* domain has been described as a human pathogen. Although studies have failed to detect archaea in samples from primary endodontic infections,[165,220] others detected a *Methanobrevibacter oralis*–like phylotype in some primarily infected canals.[268,270] Because these microorganisms have not been consistently detected in infected root canals, their role, if any, in the pathogenesis of apical periodontitis is questionable.

Viruses

Viruses are not cells but inanimate particles composed of a nucleic acid molecule (DNA or RNA) and a protein coat. On their own, they have no metabolism. In order to replicate the viral genome, they need to infect living cells and use the cell's machinery. Because viruses require viable host cells to infect and replicate themselves, they cannot thrive in the root canal with necrotic pulp. Viruses have been reported to occur in root canals only in teeth with vital pulps. For instance, the human immunodeficiency virus (HIV) has been detected in vital pulps of HIV-seropositive patients,[65] and some herpesviruses have been identified in both noninflamed and inflamed vital pulps.[99] Human cytomegalovirus (HCMV) and Epstein-Barr virus (EBV) have been detected in apical periodontitis lesions.[175] It has been hypothesized that HCMV and EBV may be implicated in the pathogenesis of apical periodontitis as a direct result of virus infection and replication or as a result of virally induced impairment of local host defenses, which might give rise to overgrowth of pathogenic bacteria in the very apical part of the root canal.[238] Bacterial challenge emanating from the canals may cause an influx of virus-infected cells into the periradicular tissues. Reactivation of HCMV or EBV by tissue injury induced by bacteria may evoke impairment of host immune response in the periradicular microenvironment, changing the potential of local defense cells to mount an adequate response against infectious agents. In addition, herpesvirus may directly stimulate inflammatory cells to release proinflammatory cytokines.[117,275] Evidence of herpesvirus infection has been observed in symptomatic apical periodontitis lesions,[174,176] abscesses,[29,57] large lesions,[175-176] and lesions from HIV-positive patients.[177] However, the role of herpesviruses in the pathogenesis of apical periodontitis, if any, has still to be elucidated.

PERSISTENT/SECONDARY ENDODONTIC INFECTIONS

As defined earlier in this chapter, persistent intraradicular infections are caused by microorganisms that resisted intracanal antimicrobial procedures and survived in the treated canal. Involved microorganisms are remnants of a primary or secondary infection. The latter, in turn, is caused by microorganisms that at some time entered the root canal system secondary to clinical intervention. The moment can be during treatment, between appointments, or even after root canal filling. In any circumstance, if penetrating microorganisms manage to adapt themselves to the new environment, surviving and flourishing, a secondary infection is established. Species involved can be oral microorganisms or not, depending on the source of secondary infection.

Persistent and secondary infections are for the most part clinically indistinguishable. Exceptions include infectious complications (such as an apical abscess) arising after the treatment of noninfected vital pulps or cases in which apical periodontitis was absent at the time of treatment but present on the follow-up radiograph. Both situations are typical examples of secondary infections. Both persistent and secondary infections can be responsible for several clinical problems, including persistent exudation, persistent symptoms, interappointment flare-ups, and failure of the endodontic treatment, characterized by a posttreatment apical periodontitis lesion.

Persistent/Secondary Infections and Treatment Failure

Although there is some suggestion in the literature that extraradicular infection or nonmicrobial factors may be involved,[127,262] intraradicular infections, either persistent or secondary, can be regarded as the major causes of endodontic treatment failure (Fig. 14-25). This statement is supported by two strong evidence-based arguments. First, it has been demonstrated that there is an increased risk of adverse treatment outcome when bacteria are present in the canal at the time of obturation.[55,234,271] Second, most (if not all) root canal–treated teeth evincing persistent apical periodontitis lesions have been demonstrated to harbor an intraradicular infection.[101,103,145,154,159,181,210,255] Based on these arguments, studies investigating bacteria remaining in the root canals at the obturation stage disclose species that have the potential to influence the treatment outcome *(outcome into perspective)*. On the other hand, studies dealing with the microbiota of root canal–treated teeth with apical periodontitis show the association of species with treatment failure, because the microorganisms detected

FIG. 14-25 Posttreatment apical periodontitis lesions in root canal–treated teeth. In poorly treated canals, the microbiota is similar to primary infections. In cases apparently well treated, fewer species are found. Regardless of treatment quality, persistent or secondary intraradicular infections are the main causative agents of endodontic treatment failure.

are likely to be participating in the etiology of posttreatment disease *(outcome already established)*.

Bacteria at the Root Obturation Stage

Diligent antimicrobial treatment may still fail to completely eliminate bacteria from the infected root canal system. This is because persisting bacteria are either inaccessible or even resistant to treatment procedures. Whatever the cause of persistence, bacterial diversity and density in infected canals are substantially reduced after treatment. Root canal samples positive for bacterial growth after chemomechanical procedures, followed or not by intracanal medication, have been shown to harbor one to five bacterial species per case, and the number of persistent bacterial cells usually varies from 10^2 to 10^5 per sample.[24,180,207,208,234,269]

No single species has been significantly found to persist after treatment procedures. Gram-negative bacteria, which are common members of primary infections, are usually eliminated. Exceptions include some anaerobic rods, such as *F. nucleatum, Prevotella* species, and *Campylobacter rectus,* which are among the species found in postinstrumentation or postmedication samples.[24,68,142,180,226,234] Most studies on this subject have clearly revealed that when bacteria resist treatment procedures, gram-positive bacteria are more frequently present. Gram-positive facultatives or anaerobes often detected in these samples include streptococci, *P. micra, Propionibacterium* species, *P. alactolyticus, Actinomyces* species, lactobacilli, *E. faecalis,* and *Olsenella uli*.[24,26-28,31,68,140,142,164-166,180,203,207,208,234,257] This supports the notion that gram-positive bacteria can be more resistant to antimicrobial treatment measures and have the ability to adapt to the harsh environmental conditions in instrumented and medicated root canals. As-yet-uncultivated bacteria have also been detected in posttreatment samples,[133,180] indicating that they may also resist antimicrobial treatment.

Bacteria persisting in the root canal after chemomechanical procedures or intracanal medication will not always maintain an infectious process. This statement is supported by evidence that some apical periodontitis lesions healed even after bacteria were isolated from the canal at the obturation stage.[55,234] There are some possible explanations[215]:

- Residual bacteria may die after obturation because of toxic effects of the filling material or sealer, access denied to nutrients, or disruption of bacterial ecology.
- Residual bacteria may be present in quantities and virulence subcritical to sustaining periradicular inflammation.
- Residual bacteria remain in locations where access to periradicular tissues is denied.

Bacteria that resist intracanal procedures and are present in the canal at the time of obturation can influence the outcome of the endodontic treatment provided they do the following[215]:

- Have the ability to withstand periods of nutrient scarcity, scavenging for low traces of nutrients or assuming a dormant state or a state of low metabolic activity, to prosper again when the nutrient source is reestablished.
- Resist treatment-induced disturbances in the ecology of the bacterial community, including disruption of quorum-sensing systems, food webs, genetic exchanges, and disorganization of protective biofilm structures.
- Reach a critical population density (load) necessary to inflict damage to the host.
- Have unrestrained access to the periradicular tissues through apical/lateral foramina or iatrogenic root perforations.
- Possess virulence attributes that are expressed in the modified environment and reach concentrations adequate to directly or indirectly induce damage to the periradicular tissues.

In this context, it should not be forgotten that host resistance to infection is also an important and probably decisive counteracting factor.

Microbiota in Endodontically Treated Teeth

The microbiota in root canal–treated teeth with apical periodontitis also exhibits decreased diversity in comparison to primary infections. Canals apparently well treated harbor one to five species, but the number of species in canals with inadequate treatment can reach up to 10 to 20 species, which is similar to untreated canals (see Fig. 14-25).[145,168,181,210,255] The number of bacterial cells in treated teeth with posttreatment disease varies from 10^3 to 10^7 per canal.[17,140,167,187]

Several culture and molecular biology studies have revealed that *E. faecalis* is the most frequent species in root canal–treated teeth, with prevalence values reaching up to 90% of cases[118,145,159,171,167,187,210,255,283] (Fig. 14-26). Root canal–treated teeth are about nine times more likely to harbor *E. faecalis* than cases of primary infections.[171] This suggests that other members of a mixed bacterial consortium commonly present in primary infections can inhibit this species, and that the bleak environmental conditions within obturated root canals do not prevent its survival. The fact that *E. faecalis* has been commonly recovered from teeth treated at multiple visits or left open for drainage[233] suggests that this species is a secondary invader capable of colonizing the canal and resisting treatment. In other words, *E. faecalis* may cause secondary infections that later become

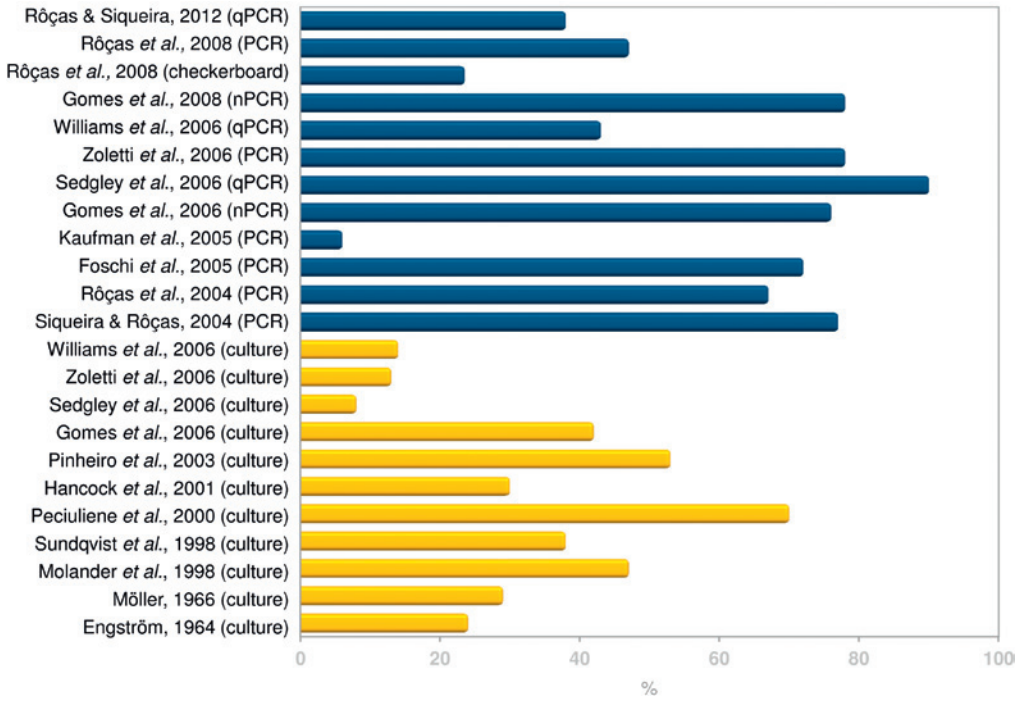

FIG. 14-26 Prevalence of *Enterococcus faecalis* in samples from root canal–treated teeth with apical periodontitis. Data from culture *(yellow bars)* and molecular *(blue bars)* studies follow.

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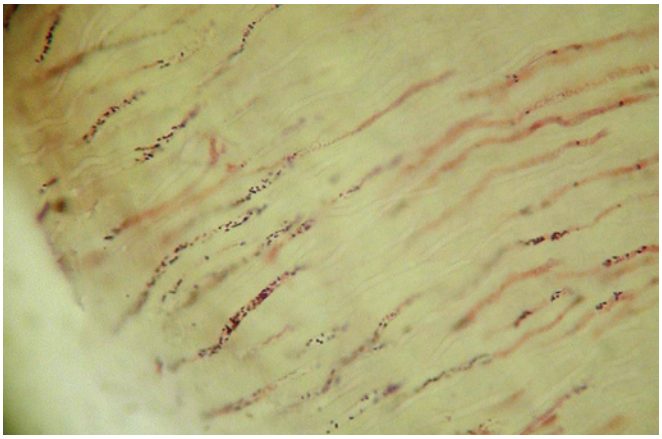

FIG. 14-27 Dentinal tubule infection by *Enterococcus faecalis* in dog's teeth after experimental infection. Notice that cells invaded the entire extent of some tubules up to the cementum (Brown and Brenn staining, ×1000).

persistent. *E. faecalis is* considered a transient species in the oral cavity; its source may be food.[282]

For a given microorganism to survive in endodontically treated teeth, it has to resist intracanal disinfection procedures and adapt to the harsh environmental conditions caused by treatment. The ability of *E. faecalis* to penetrate dentinal tubules, sometimes to a deep extent[74,201] (Fig. 14-27), can enable it to escape the action of endodontic instruments and irrigants used during chemomechanical preparation.[74,200] Moreover, its ability to form biofilms in root canals can be important for its resistance to and persistence after intracanal antimicrobial procedures.[45] *Enterococcus faecalis* is also resistant to calcium hydroxide[22]; such an ability to resist high pH values seems to be related to a functioning proton pump that drives protons into the cell to acidify the cytoplasm.[53] Unlike most putative endodontic pathogens frequently found in primary infections, *E. faecalis* may colonize root canals in single infections,[255] and such relative independence of living without deriving nutrients from other bacteria can be extremely important for its establishment in treated root canals. Finally, environmental cues can regulate gene expression in *E. faecalis,* affording this bacterium the ability to adapt to varying (and adverse) conditions.[84] Indeed, *E. faecalis* can enter a so-called viable but noncultivable (VBNC) state,[104] which is a survival mechanism adopted by several bacteria when exposed to unfavorable environmental conditions.[105] In the VBNC state, bacteria lose the ability to grow in culture media but maintain viability and pathogenicity and sometimes can resume division when optimal environmental conditions are restored. Studies have demonstrated that *E. faecalis* has the ability to survive in environments with scarcity of nutrients and then flourish when the nutrient source is reestablished.[59] It also has the capacity to recover from a prolonged starvation state in obturated canals,[188] suggesting that viable cells of this species, entombed at the time of canal obturation, may provide a long-term nidus for subsequent infection.

Taken together, all these properties help explain the significantly high prevalence of *E. faecalis* in root canal–treated teeth. Although association of this species with posttreatment disease is suggested by epidemiologic studies and supported by the species attributes that allow it to survive under unfavorable environmental conditions, causation is unproved. In fact, the following findings from studies carried out in independent laboratories have questioned the status of *E. faecalis* as the main causative agent of endodontic failures:

- Despite being easily cultivated, *E. faecalis* is not detected in all studies evaluating the microbiota of root canal–treated teeth with posttreatment disease.[30,172]
- Even when present, *E. faecalis* is rarely one of the most dominant species in retreatment cases.[158,167,168,181]
- *E. faecalis* has been found not to be more prevalent in root canal–treated teeth with lesions when compared with treated teeth with no lesions.[87,283]

Other bacteria found in endodontically treated teeth with apical periodontitis include streptococci and some fastidious anaerobic species, such as *P. alactolyticus, Propionibacterium* species*, Filifactor alocis, Dialister pneumosintes, Dialister invisus, Tannerella forsythia, P. micra, Prevotella intermedia,* and *Treponema denticola*[7,69,118,145,158,167,168,181,210,213,255] (Fig. 14-28). Uncultivated phylotypes correspond to 55% of the taxa detected in treated canals, and collectively they can also be in high proportions, corresponding to about half of the 16S rRNA gene sequences retrieved in clone libraries.[181] Detection of uncultivated phylotypes helps explain why culture studies fail to detect bacteria in some treated root canals.

The bacterial community profiles in treated cases vary from individual to individual, indicating that distinct bacterial combinations can play a role in treatment failure.[168,181] All these findings strongly suggest that the microbiota of root canal–treated teeth with apical periodontitis is more complex than previously anticipated by culture studies.

Fungi are only occasionally found in primary infections, but *Candida* species have been detected in root canal–treated teeth in up to 18% of the cases.[30,51,118,119,140,145,210,255] Fungi gain access to root canals via contamination during endodontic therapy (secondary infection) or they overgrow after inefficient intracanal antimicrobial procedures that cause an imbalance in the primary endodontic microbiota.[232] *Candida albicans* is by far the most commonly detected fungal species in root canal–treated teeth. This species has several properties that can be involved in persistence following treatment, including its ability to colonize and invade dentin[191,192,224] and resistance to calcium hydroxide.[272,274]

EXTRARADICULAR INFECTIONS

Apical periodontitis lesions are formed in response to intraradicular infection and by and large constitute an effective barrier against spread of the infection to the alveolar bone and other body sites. In most situations, apical periodontitis inflammatory lesions succeed in preventing microorganisms from invading the periradicular tissues. Nevertheless, in some specific circumstances, microorganisms can overcome this defense barrier and establish an extraradicular infection. The most common form of extraradicular infection is the acute apical abscess, characterized by purulent inflammation in the periradicular tissues in response to a massive egress of virulent bacteria from the root canal. There are, however, other forms of extraradicular infection, which have been discussed as one of the possible etiologies of persistence of apical periodontitis lesions in spite of diligent root canal treatment.[194,263] These conditions entail the establishment of microorganisms in the periradicular tissues either by adherence to the apical external root surface in the form of extraradicular biofilm

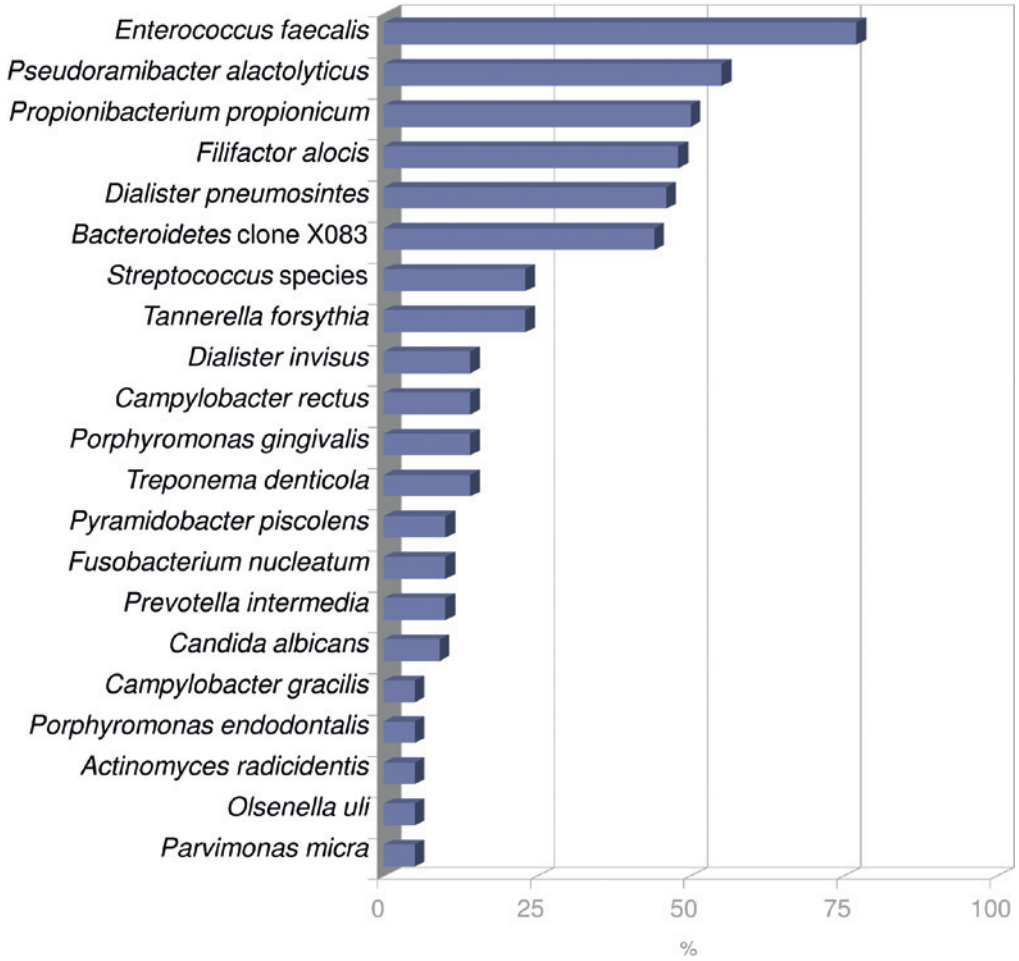

FIG. 14-28 Prevalence of microorganisms detected in root canal–treated teeth with posttreatment disease. Data from the authors' studies using a taxon-specific polymerase chain reaction assay. (From references 218 and 210.)

structures[129,261] or by formation of cohesive actinomycotic colonies within the body of the inflammatory lesion.[79]

Therefore, extraradicular infection can develop as follows[195]:

- It can be a result of direct advance of some bacterial species that overcome host defenses concentrated near or beyond the apical foramen, an extension of the intraradicular infectious process, or bacterial penetration into the lumen of pocket (bay) cysts, which are in direct communication with the apical foramen. The borderline between the infecting endodontic microbiota and the host defenses is often located intraradicularly, short of or at the apical foramen. In some cases, however, microorganisms may reach the periradicular tissues, and the borderline is then situated extraradicularly, beyond the boundaries of the apical foramen (Fig. 14-29). In the latter situation, the whole infectious process would be a continuum composed of an intraradicular and an extraradicular segment, in which the former fosters the latter (see Fig. 14-29). It has been suggested that in some cases, the extraradicular segment may become independent of the intraradicular component of the infectious process, but this has not been confirmed by scientific findings.
- It can result from bacterial persistence in the apical periodontitis lesion after remission of an acute apical abscess. The acute apical abscess is for the most part clearly dependent on the intraradicular infection; once the intraradicular infection is properly controlled by root canal treatment or tooth extraction and drainage of pus is achieved, the extraradicular infection is handled by the host defenses and usually subsides. Nonetheless, it should be appreciated that in some rare cases, bacteria that have participated in acute apical abscesses may persist in the periradicular tissues following resolution of the acute response and establish a persistent extraradicular infection associated with chronic periradicular inflammation, sometimes resulting in an actively draining sinus tract.
- It can be a sequel to apical extrusion of debris during root canal instrumentation (particularly after overinstrumentation). Bacteria embedded in dentinal chips can be physically protected from the host defense cells and therefore can persist in the periradicular tissues and sustain periradicular inflammation. The virulence and quantity of the involved bacteria, as well as the host's ability to deal with infection, will be decisive factors dictating whether an extraradicular infection will develop or not.

Conceivably, the extraradicular infection can be dependent on or independent of the intraradicular infection.[194] Independent extraradicular infections would be those that are no longer fostered by the intraradicular infection and can persist even after successful eradication of the latter. So far, it has been

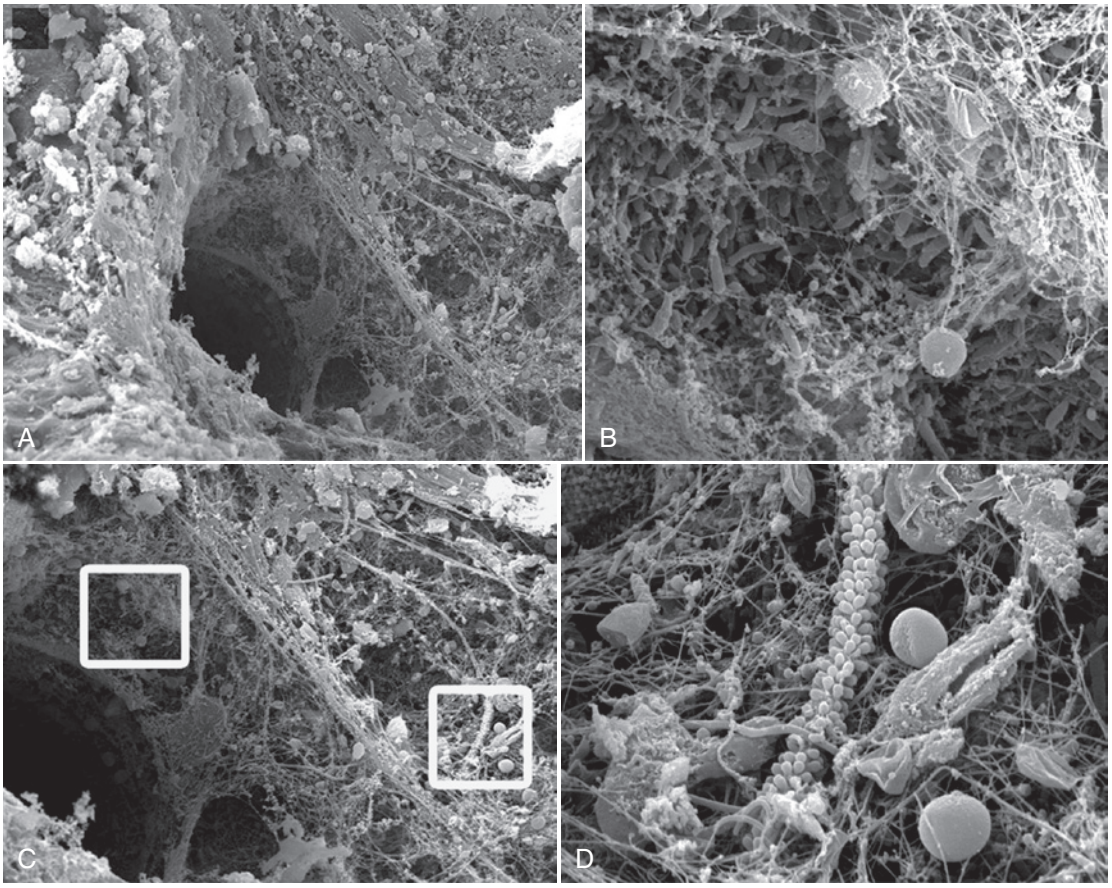

FIG. 14-29 Scanning electron micrograph showing extensive bacterial colonization in the very apical part of the canal, near and at the apical foramen (**A-B**, magnification ×550 and magnification ×850, respectively). **C**, Higher magnification of left inset in **B** showing a bacterial biofilm adhered to the very apical canal walls (magnification ×3700). **D**, Higher magnification of right inset in **B** showing a fully developed "corncob" in tissue meshwork adjacent to the apical foramen (magnification ×4000). (Modified from Siqueira JF Jr, Lopes HP: Bacteria on the apical root surfaces of untreated teeth with periradicular lesions: a scanning electron microscopy study, *Int Endod J* 34:216, 2001.)

suggested that the main bacterial species implicated in independent extraradicular infections are *Actinomyces* species and *Propionibacterium propionicum,* in a pathologic entity named *apical* (or periapical, periradicular) *actinomycosis*[23,79,236,256] (Fig. 14-30). These bacteria form cohesive colonies that may be collectively resistant to phagocytosis.[58] However, existence of apical actinomycosis as a self-sustained pathologic entity, no longer nurtured by the intraradicular infection, and its involvement as an exclusive cause of treatment failure is speculative and remains to be proved.[151,209]

It is still controversial whether chronic apical periodontitis lesions can harbor bacteria for very long beyond initial tissue invasion.[9] Studies using culture-dependent[247,262,277] or culture-independent molecular biology methods, such as checkerboard hybridization,[63,249] fluorescence in situ hybridization (FISH),[248] clone library analysis,[78] and pyrosequencing,[173] have reported the extraradicular occurrence of a complex microbiota associated with apical periodontitis lesions that did not respond favorably to the root canal treatment. Anaerobic bacteria have been reported as the dominant microorganisms in several of those lesions.[247,249] Apart from a discussion about whether contamination can be effectively prevented during surgical sampling of periradicular lesions, these studies did not evaluate the bacteriologic conditions of the apical part of the root canal. This makes it difficult to ascertain whether those extraradicular infections were dependent on or independent of an intraradicular infection. Actually, findings from a study evaluating both the resected root ends and the apical periodontitis lesions from treated teeth suggested that the large majority of cases of extraradicular infections are maintained by a concomitant apical root canal infection.[246]

The incidence of extraradicular infections in untreated teeth is conceivably low.[126,206] Extraradicular biofilms are infrequent, and when present they are virtually always associated with intraradicular biofilms.[152] This is in consonance with the high success rate of nonsurgical root canal treatment.[40,150,235] Even in root canal–treated teeth with recalcitrant lesions in which a higher incidence of extraradicular bacteria has been reported, a high rate of healing following retreatment[56,235] indicates that the major cause of posttreatment disease is located within the root canal system, characterizing a persistent or secondary intraradicular infection. This has been confirmed by culture and by molecular and histobacteriologic studies investigating the microbiologic conditions of root canals associated with posttreatment apical periodontitis.[103,118,145,154,168,210,255]

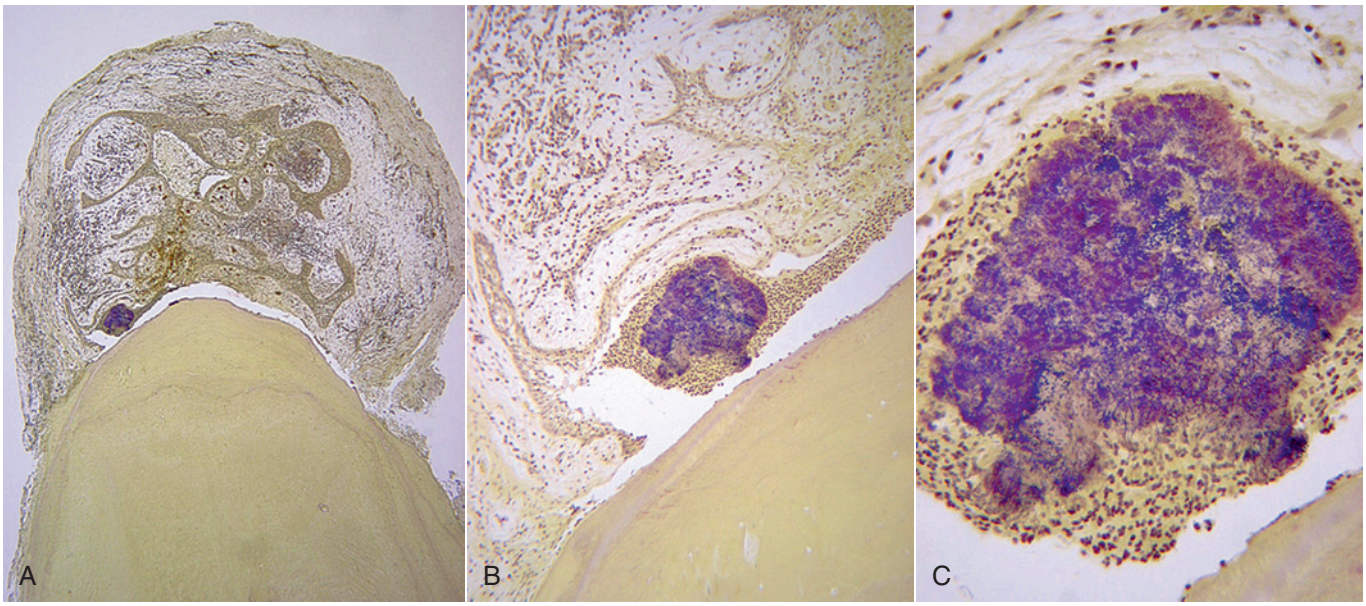

FIG. 14-30 Bacterial aggregate in an epithelialized apical periodontitis lesion, suggestive of actinomycosis. This case presented a severe flare-up in the past, but the tooth was asymptomatic at the time of extraction. **A,** Section not passing through the canal; note cyst cavity and bacterial aggregation on the left (magnification ×25). **B,** Detail of area with colony (magnification ×100). **C,** Higher magnification of colony, which is surrounded by neutrophils (magnification ×400). Sections stained with a Taylor-modified Brown and Brenn technique. (Courtesy Dr. Domenico Ricucci.)

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CHAPTER 15

Pathobiology of Apical Periodontitis

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CHAPTER OUTLINE

Apical Periodontitis
- *Prevalence*

Etiology

Infection: A Conflict Between Host and Parasites

Pathogenesis
- *Innate Immune Response*
- *Adaptive/Specific Immune Response*
- *Neurogenic Inflammation*

Diagnosis
- *Correlation Between Clinical and Histologic Findings*
- *Correlation Between Radiographic and Histologic Findings*

Histopathology

Symptomatic Apical Periodontitis
- *Cell Biology*
- *Inflammatory Mediators*
- *Histopathology*
- *Clinical Features*
- *Outcomes*

Asymptomatic Apical Periodontitis: Apical Granuloma, Chronic Apical Periodontitis
- *Cell Biology*
- *Inflammatory Mediators*
- *Histopathology*
- *Clinical Features*
- *Outcomes*

Asymptomatic Apical Periodontitis with Cyst Formation: Radicular Cyst, Chronic Apical Periodontitis with Cyst Formation
- *Cell Biology*
- *Inflammatory Mediators*
- *Histopathology*
- *Clinical Features*
- *Outcomes*

Asymptomatic Apical Periodontitis with Reactive Bone Formation: Condensing Osteitis or Chronic Focal Sclerosing Osteomyelitis
- *Cell Biology*
- *Histopathology*
- *Clinical Features*
- *Outcomes*

Periapical Lesions of Nonendodontic Origin

Extraradicular Endodontic Infection

Apical Periodontitis and Systemic Diseases

Genetic and Systemic Disease Risk Factors of Persistent Apical Periodontitis
- *Genetic Risk Factors*
- *Systemic Disease Risk Factors*

Wound Healing of Apical Periodontitis
- *Periapical Wound Healing After Nonsurgical Root Canal Therapy*
- *Periapical Wound Healing After Surgical Endodontic Therapy*
- *Can Radicular Cysts in Apical Periodontitis Lesions Regress After Nonsurgical Endodontic Therapy?*

Factors Influencing Periapical Wound Healing After Endodontic Therapy

Periradicular tissues consist of cementum, periodontal ligament, and alveolar bone. *Cementum* is a mineralized, avascular connective tissue and consists of three different types. Acellular afibrillar cementum covers the teeth at and along the cementoenamel junction. Acellular extrinsic fiber cementum is confined to the coronal half of the root. Cellular intrinsic fiber cementum is present on the apical half of the root where no acellular extrinsic fiber cementum has been laid down.[192] Many growth factors, such as insulin-like growth factor-1 (IGF-1), fibroblast growth factors (FGFs), epidermal growth factor (EGF), bone morphogenetic proteins (BMPs), transforming growth factor-β (TGF-β), and platelet-derived growth factor (PDGF), are contained in the cementum matrix.[47,88,164] These growth factors may be released under certain conditions, as they have been shown to be associated with cementoblast proliferation, migration, and differentiation during cementum wound healing.[88]

The *periodontal ligament* is a soft, specialized connective tissue that connects the cementum to the alveolar bone. Periodontal ligament contains heterogeneous cell populations and extracellular matrix (ECM).[144,192] The cells of the periodontal ligament include osteoblasts, osteoclasts, fibroblasts, epithelial cell rests of Malassez, macrophages, cementoblasts, and undifferentiated mesenchymal cells (stem cells).[192] Fibroblasts, osteoblasts, and epithelial cells are differentiated cells that have retained the ability to undergo limited cell divisions and proliferation upon stimulation by appropriate signals. Multipotent mesenchymal stem cells of the periodontal ligament are capable

of differentiating into cementoblast-like cells and periodontal ligament cells as well as osteoblasts.[112,182,233] The ECM of the periodontal ligament consists of collagen fibers, fibronectin, elastin, other noncollagenous proteins, and proteoglycans. The ECM serves as stratum for cell adhesion and promotes cell spreading and cytoskeletal organization. The collagen fibers (Sharpey fiber) of the periodontal ligament connect the tooth with the alveolar bone. The periodontal ligament is highly vascularized and innervated. The tissue apical to the dentinocemental junction should be considered as part of the periodontal ligament because cementum is not a normal component of the pulp tissue.

Epithelial cell rests of Malassez (ERM), the remnants of the Hertwig epithelial root sheath that disintegrates after tooth development, are present in the periodontal ligament near the root surface in all teeth after root formation.[209] They are nests of epithelial cells connected as a network and surrounded by a basal lamina.[192,230] ERM are quiescent in normal periodontal ligament[192] but can be stimulated to proliferate in apical periodontitis.[158] They are believed to be the cellular source that when properly stimulated can form radicular cysts in certain apical periodontitis lesions.[158,184,200]

Alveolar bone or *alveolar process* is that part of bone of the jaws housing the sockets for the teeth. It consists of outer cortical plate, a central spongy or cancellous bone, and bone lining the sockets.[192] Bone matrix contains IGFs, TGF-β, BMPs, FGF, and PDGF.[40,250] These growth factors are essential for osteoblast progenitor cell proliferation, migration, and differentiation during bone wound healing.[161]

The response of the periradicular tissues to various injuries is similar to that of other connective tissues elsewhere in the body. The response is manifested as an inflammatory reaction regulated by both innate and adaptive immune mechanisms. Although microbial infection of the pulp in the root canals is the primary cause of apical periodontitis,[119,178,184] the pathologic changes of the periapical tissues in apical periodontitis are usually not directly caused by microbes themselves, but rather by their toxins, noxious metabolic by-products, and disintegrated pulp tissue in the root canal system. These irritants are capable of inducing both innate and adaptive immune responses; they can either activate nonantigenic pathways or serve as antigens to activate adaptive responses. The subsequent inflammatory responses are diverse and can involve changes in microvasculature, transmigration of blood-borne cells and plasma proteins out of the blood circulation into the tissue space, and activation of sensory nerves. In addition, endothelial cells, mast cells, platelets, fibroblasts, neutrophils, macrophages, dendritic cells, innate and adaptive immune cells, immunoglobulins, inflammatory mediators, proinflammatory cytokines, chemokines, and neuropeptides are also involved in the inflammatory response. Apical periodontitis can be protective or destructive, depending on the dynamic interaction between microbial insult and the host's defenses in the periapical tissues. Unfortunately, the bacterial biofilm formed in the root canal system with necrotic pulp is protected from host's defenses and antibiotic therapy because of a lack of blood circulation in the root canal system. Consequently, any attempt of wounded periradicular tissues to repair/regenerate is futile, because bacterial toxins and noxious metabolic by-products in the root canal system continuously egress into the periapical area and irritate the periapical tissues. Emerging lines of evidence suggest that under most conditions, bacterial biofilms in the complex root canal system can be greatly reduced, but not eliminated, by conventional endodontic procedures such as mechanical instrumentation, antiseptic irrigation, and intracanal medication. If microbes in the root canal system are effectively eliminated or entombed within the root canal by filling material, and the root canal system is adequately sealed and protected from coronal microleakage, then periradicular tissues have the ability to restore their original structures by means of a repair/regeneration process. Nevertheless, the presence of posttreatment apical periodontitis may be due to persistent microbial biofilms,[76] and this recognition has spurred considerable research into treating biofilms (see Chapters 13 and 14).

APICAL PERIODONTITIS

Prevalence

Epidemiologic study of apical periodontitis documents that the prevalence of apical periodontitis varies among patients aged 20 to 30 (33% prevalence of apical periodontitis), 30 to 40 (40%), 40 to 50 (48%), 50 to 60 (57%), and older than 60 years of age (62%).[203] Most studies on the prevalence of apical periodontitis are from European and Scandinavian countries.[70,117,237] According to a survey by the American Dental Association in 1990, an estimated 14 million root canal treatments were performed in the United States alone.[9] Apical periodontitis is a prevalent health problem.[71]

ETIOLOGY

The etiology, pathogenesis, and histopathology of apical periodontitis are similar to that of marginal periodontitis (see also Chapter 25). Both diseases are caused by bacterial infection and involve pathologic changes of alveolar bone, periodontal ligament, and cementum. Marginal periodontitis affects coronal periodontal tissues, whereas apical periodontitis affects apical periodontal tissues. Bone loss is one of characteristic features in both diseases: crestal bone is lost in marginal periodontitis, and apical bone undergoes resorption in apical periodontitis.

Apical periodontitis can be caused by both exogenous and endogenous factors. Exogenous factors include microbes and their toxins and noxious metabolic by-products, chemical agents, mechanical irritation, foreign bodies, and trauma. Endogenous factors include the host's metabolic products, such as urate and cholesterol crystals,[188] as well as cytokines or other inflammatory mediators that activate osteoclasts.[252] These irritants can activate nonantigenic pathways or antigenic pathways to induce innate and adaptive immunoinflammatory responses, respectively.

In the root canal system, infection of the pulp tissue caused by caries or other pathways is the primary cause of apical periodontitis.[119,178,184,261] The classic study by Kakehashi and colleagues[119] demonstrated that pulp necrosis and periradicular inflammation developed in conventional rats when the pulps of teeth were exposed to oral microorganisms. However, in germ-free laboratory rats, no pulp necrosis and periradicular inflammation occurred even when the pulps of teeth were exposed to the oral environment and packed with sterile food debris. A similar response occurs in humans. Using bacterial culturing, it has been demonstrated that traumatized human teeth with intact crowns and necrotic pulps *without bacterial contamination* did not show radiographic evidence of periapical

bone destruction. In contrast, if bacteria were isolated from traumatized teeth with intact crowns and necrotic pulps, then radiographic evidence of periradicular bone destruction was observed.[261] These important findings have been replicated in nonhuman primate experiments. When the pulps of intact vital teeth were intentionally devitalized under aseptic conditions and left in the root canals with bacteria-tight, sealed coronal restoration for 6 months to 1 year, no periradicular inflammatory reaction was observed.[156,178] Taken together, there is considerable evidence that bacteria constitute a major etiologic factor in the development of apical periodontitis.

Bacterial toxins (e.g., lipopolysaccharide [LPS], lipoteichoic acid [LTA]) and noxious metabolic by-products that egress from the root canal system into the periapical tissues are capable of inducing a periapical inflammatory reaction.[53,64,226,311] These substances can activate the innate immune system via receptors that recognize the stereotypic pathogen-associated molecular patterns (PAMPs) that are found in the structure of these toxins. Different classes of microbes express different molecular patterns that are recognized by different pattern recognition receptors (PRRs) or Toll-like receptors (TLRs) on host cells, such as phagocytes, dendritic cells, and B lymphocytes.[1,171,172] PRRs or TLRs are encoded in the germline. In mammalian species, there are at least 10 TLRs, and each appears to have a distinct function in innate immune recognition.[171] For example, LPS can stimulate sensory nerve fibers to release calcitonin gene–related peptide (CGRP) and substance P (SP)[61,107] to cause vasodilation and increased vascular permeability. LPS and lipoproteins can also activate TLRs on dendritic cells to stimulate T lymphocyte differentiation.[4] Certain subtypes of TLRs recognize the common shared structural features of various toxins (i.e., PAMPS). Because the TLRs are synthesized before an infection, they are classified as part of the innate immune system.

Apical periodontitis can be caused either by entry into the periapical tissues of bacterial toxins, enzymes, and noxious metabolic by-products or by direct invasion of the periapical tissues by microbes originating from the root canal system. It is important to differentiate between apical inflammation and apical infection. *Apical inflammation* is the periapical tissue reaction to irritants emerging from the root canal system that manifests as vasodilation, increased vascular permeability, and exudation. In contrast, *apical infection* is due to the physical presence of pathogenic microorganisms in the periapical tissues that subsequently produce tissue damage. There can be infection without inflammation, for instance, in a severely immunocompromised patient. There can also be inflammation without infection, such as in a myocardial infarct, cerebral infarct, and physical or chemical injury.[165] In diseases caused by infection, bacteria are usually present in the involved tissues or organs,[294] such as acute necrotizing gingivitis, marginal periodontitis, actinomycosis, tuberculosis, and bacterial bronchitis. Although apical periodontitis is primarily an infectious disease, bacteria are usually not present in the periapical tissues but in the root canal system,[139,184,296] except in certain cases of apical periodontitis associated with abscess formation,[201,291,303] or with a draining sinus tract,[90,203,301] or extraradicular endodontic infection.[260,284] One major current hypothesis is that apical periodontitis is triggered by entry into the periapical tissues of bacterial toxins, enzymes, and noxious metabolic by-products.[269] The mere presence of bacteria (colonization) in some apical periodontitis lesions does not necessarily denote a periradicular infection. Periapical infection is related to both virulence and the number and specific combinations of microorganisms in the periapical tissues.[264] Bacteria may be temporarily present in the inflamed periradicular tissues only to be killed by the host's defense mechanisms when the focus of infection in the root canal system is effectively eliminated by mechanical instrumentation, antiseptic irrigation, and intracanal medication. For instance, the majority of apical periodontitis lesions with abscess formation or draining sinus tracts heal satisfactorily after nonsurgical root canal treatment without the need for systemic antimicrobial therapy.[203]

Primary root canal infection in untreated root canals is a polymicrobial mix with approximately equal proportions of gram-positive and gram-negative species, dominated by obligate anaerobes (see Chapter 14).[262,264] In root-filled teeth with apical periodontitis, gram-positive microorganisms, with a relatively equal distribution of facultative and anaerobic species, appear to dominate other microorganisms.[176,265] A high prevalence of *E. faecalis* is frequently observed in filled root canals associated with persistent apical periodontitis.[94,124,176,206,244,264] These issues are described in greater detail in Chapter 14.

Physical (overinstrumentation, overfilling) and chemical (irrigants, intracanal medication, root canal filling materials) insults,[230] as well as traumatic injury[10,11] to the periapical tissues, can also cause apical periodontitis, depending on the severity of injury and cytotoxicity of the chemicals. Foreign bodies, such as root canal filling materials, have been shown to cause persistent periapical inflammation.[133,187,205,313] However, the possibility of bacterial contamination in foreign body–induced apical periodontitis lesions was not carefully ruled out in many studies, so it is possible that the foreign bodies served as carriers for the microorganisms. In addition, foreign bodies have the odd property of favoring infection,[315] as they can lower the infectious dose of bacteria (LL) and cause granulocytes to develop phagocytic defect or loss of ammunition (LL, OK).[314] Although most root canal filling materials are not inert and are capable of inducing certain degrees of inflammation, in general they are biocompatible and well tolerated by periapical tissues.[99]

It has been demonstrated histologically that periodontal disease could cause inflammatory pulpal and periapical disease.[140]

INFECTION: A CONFLICT BETWEEN HOST AND PARASITES

Every infection is a race between the capabilities of the microorganism to multiply, spread, and cause disease and the ability of the host to control and finally eliminate the microorganisms.[294] The host has physical barriers—surface epithelium, enamel, and dentin—as well as innate and adaptive immune defenses to prevent pulpal and periapical infection. Nevertheless, parasites also possess weapons, leading to inhibition of phagocytosis, inhibited lysosomal function, reduced killing by phagocytes, inactivation of complement system and immunoglobulins, and specific mechanisms that permit invasion of the host's physical barriers.[294] Infection of a tissue can manifest different histopathologic features as a result of specific host-parasite interactions that occur. Many infections are asymptomatic in more than 90% of individuals.[294] For instance, pulp necrosis and chronic apical periodontitis caused by root canal

infection are usually asymptomatic, and patients are often surprised to find out that this infection has been present for a sufficient time to lead to destruction of periapical bone. Therefore, there is no simple correlation between infection and clinical symptoms of apical periodontitis, except in cases of symptomatic apical periodontitis and acute apical abscess.

PATHOGENESIS

When pulps are infected/inflamed, many innate and adaptive immune cells release elevated amounts of various inflammatory mediators, including cytokines, chemokines, and neuropeptides. As the pulpal inflammation spreads, the inflammatory mediators begin to alter the physiology of the periapical tissues. Clinically, the observable changes on radiographic examination are widening of the periodontal ligament space or development of apical osteolytic lesions due to bone resorption. The loss of bone is mainly caused by activated osteoclasts. Many cytokines, such as interleukin (IL)-1, IL-11, IL-17, and tumor necrosis factor α (TNF-α), are found to have the ability to induce osteoclast progenitor cell differentiation and activation.[24,252] The inflammation-induced bone resorption in the periapical tissues is accompanied by recruitment of immune cells that essentially build a defensive line against the spread of microbial invasion from the root canal.[170] The pathogenesis of apical periodontitis involves innate and adaptive immune responses as well as sensory nerve response in the periapical tissues. Immune cells present in human periradicular lesions consist of lymphocytes, macrophages, plasma cells, neutrophils, dendritic cells, and natural killer (NK) cells with the former two types as the majority.[151,169] The characteristic features of innate and adaptive immunity are summarized in Table 15-1.

TABLE 15-1
Features of Innate and Adaptive Immunity

Property	Innate	Adaptive
Recognition	Structures shared by groups of related microbes (conserved molecular patterns)	Antigens of microbes and of nonmicrobial antigens (details of molecular structure)
Diversity	Limited	Very large
Memory	None	Yes
Receptors	Encoded in the genome	Encoded in gene segments (somatic recombination)
Blood proteins	Complement	Antibodies
Cells	Macrophages Neutrophis	Lymphocytes Antigen-presenting cells NK cells
Action time	Immediate activation of effectors	Delayed activation of effectors
Response	Costimulatory molecules, cytokines	
(IL-1, IL-6)		
IL-2	Clonal expansion	
Chemokines (IL-8)	Effector cytokines (IL-4, IFN*r*)	

IFN, interferon; *IL,* interleukin; *NK,* natural killer.
Data from Janeway CA, Medzhitov R: Innate immune recognition, *Annu Rev Immunol* 20:197, 2002; Abbas AK, Lichtman AH, Pober JS: *Cellular and molecular immunology*, ed 5, Philadelphia, 2003, Saunders.

Innate Immune Response

Specificity of Innate Immune Response

In recent years, the concept of the nonspecific nature of innate immunity has changed since identification of a network of germline-encoded receptors, the pattern-recognition receptors (PRRs) mentioned earlier, that recognize specific molecular motifs of microorganisms.[5,171] PRRs can be expressed on the cell surface (macrophages, dendritic cells, neutrophils, NK cells, B cells), in intracellular compartments, or secreted into the blood and tissue fluids.[115] There are numerous microbial constitutive and conserved products such as the pathogen-associated molecular patterns (PAMPs), also noted earlier. Importantly, the PRRs of the innate immune system recognize PAMPs.[5,171]

The specificity of innate immunity is due to the recognition of PAMPs of microorganisms by PRRs, such as *Toll-like receptors* (TLRs), of the host's cells. Activation of PRRs triggers numerous host responses, including opsonization, activation of complement and coagulation cascades, phagocytosis, activation of proinflammatory signaling pathways, and induction of apoptosis.[115] For example, TLR4/CD14 is the receptor for the gram-negative bacterial LPS. TLR4-mutated C3H/HeJ mice (LPS hyporesponsive) have reduced response to gram-negative bacteria and are highly susceptible to infection by *Salmonella typhimurium* or *Neisseria meningitidis*.[48] Importantly, there is a reduced expression of IL-1 and IL-12 and decreased periradicular bone destruction in TLR-4 deficient mice when teeth are subjected to pulpal exposures and infection with a mixture of four anaerobic pathogens: *Prevotella intermedia, Fusobacterium nucleatum, Streptococcus intermedius* (G+), and *Peptostreptococcus micros* (G+).[106] In addition, LPS is shown to be capable of inducing pain via direct activation of TLR4/CD14 expressed on nociceptive sensory neurons.[293] Thus, the TLR4 PRR receptor is importantly involved in odontogenic infections.

Components such as lipoteichoic acid (LTA) of gram-positive bacterial cell walls can also stimulate innate immunity in a way similar to LPS. TLR2 plays a major role in detecting gram-positive bacteria and is involved in the recognition of a variety of microbial components, including LTA, lipoproteins, and peptidoglycan. The importance of TLR2 in the host defense against gram-positive bacteria has been demonstrated using TLR2-deficient ($TLR2^{-/-}$) mice, which were found to be highly susceptible to challenge with *Staphylococcus aureus* or *Streptococcus pneumoniae*.[66,270] LTA also stimulates leukocytes to release inflammatory mediators, including TNF-α, IL-1β, IL-6, IL-8, and prostaglandin (PG) E2, which are known to play a role in various phases of the inflammatory response. All of these inflammatory mediators have been detected in periapical samples, and each has a well-known tissue-damaging effect by activating various host responses.

The innate immune response to bacterial infection induces expression of proinflammatory cytokines, chemokines, and costimulators, which are essential for activation and influence

of the nature of adaptive immune response.[4,172] The innate immune system is capable of recognizing nonself and self-antigens, whereas the adaptive immune system does not; thus, many autoimmune diseases are disorders of adaptive immunity.[172]

Nonspecific Innate Immune Response

The primary nonspecific innate immune defense mechanism in apical periodontitis is phagocytosis of microbes by specialized phagocytes such as polymorphonuclear leukocytes (PMNs) and macrophages. Tissue inflammation leads to the recruitment of PMNs from the blood circulation into the periradicular tissue. Activated PMNs exhibit an abrupt increase in oxygen consumption, the well-known respiratory burst, resulting in the release of oxygen radicals, a family of extremely destructive, short-lived substances that destroy nearby microorganisms and host cells.[15] Phagocytosed microbes or foreign particles are exposed to a toxic environment containing specific and azurophil granules and oxygen-derived free radicals and are eventually degraded.[194] PMNs also possess an extracellular killing mechanism via neutrophil extracellular traps (NETs), which are extracellular structures composed of chromatin with specific proteins from the neutrophilic granules. Upon activation (e.g., by IL-8, LPS, bacteria, fungi, activated platelets), neutrophils start a cellular program called *apoptosis* that leads to their death and the formation of NETs, which have antimicrobial activities.[30,77] Besides their role in the innate immunity as professional phagocytes, macrophages also serve as antigen-presenting cells by expressing MHC (major histocompatibility complex) class II molecules that interact with antigen-specific clones of T-helper lymphocytes. Circulating monocytes are the precursors of both tissue macrophages and many dendritic cell subsets.[1,126,234] The details of the immunologic activities of neutrophils and macrophages in periapical pathosis are described in Chapters 12 and 13.

Adaptive/Specific Immune Response

The specificity of adaptive immunity is regulated at genetic levels in B and T lymphocytes through a complex process leading to the generation of molecules that recognize and bind to foreign or self-antigens. These molecules are specific receptors on T cells (T-cell antigen receptors or TCRs) and on B cells (B-cell antigen receptors or BCRs; also termed *immunoglobulins*). TCRs on T cells interact with antigens that are presented by antigen presenting cells expressing MHC molecules along with other accessory molecules, whereas BCRs on B cells interact with antigens directly. BCRs may be secreted in the blood circulation or in the tissues as antibodies. The variable region of both TCR and BCR proteins are rearranged at the genomic level via genetic recombination of the V(D)J segments. The estimated total diversity after this recombination for TCR is approximately 10^{21} and for BCR is approximately 10^{16} that generates the repertoire of different individual T and B cell clones.[1,116] Each T or B cell clone generated in the bone marrow carries a specific TCR and BCR. They undergo a positive and negative selection process through which most clones are deleted via apoptosis because they bind to self-antigens. This initial "negative screening" process reduces the potential for autoimmune disorders. Only those that do not interact with self-antigens are released into the lymphatic system and blood circulation. The naive T cells circulate back and forth between the lymphatic system and blood circulation until they encounter foreign antigens presented by antigen-presenting cells. About 97% of T cells undergo apoptosis, and only a small percentage of these cells are exported to the periphery as mature T cells.[1]

The interaction between TCR and the antigen peptide/MHC complex and costimulators activates T cells, leading to the synthesis of T-cell growth factor, IL-2, and its receptor that causes T-cell clonal expansion/proliferation. Some of these T cells differentiate into armed, effector T cells, and others become memory cells. There are a number of T-cell subpopulations, categorized by their functions: (1) T helper cells (T_H), (2) T regulatory cells (T_{reg}), (3) T suppressor cells (T_S), and (4) T cytotoxic (cytolytic) (T_C) cells.[1,56,290] Some of them can be distinguished by their cell surface markers, cytokine profiles, or transcriptional factors. See also Chapter 13 for additional details.

Upon antigen stimulation, naive CD4 T cells proliferate and differentiate into T_H0, which are subsequently committed to develop into T_H1 or T_H2 cells. Monocytoid DC (dendritic cell) (DC1) induces T_H1-type responses; plasmacytoid DC (DC2) selectively induces T_H2 responses. Each subset of T_H cells has distinct functions and cytokine profiles. T_H1 cells mainly produce IL-2 and interferon (IFN)-γ, which activate macrophages and induce B cells to produce opsonizing antibody. T_H2 cells produce IL-4, -5, -10, and -13, which activate B cells to make neutralizing antibody. T_H0 can also develop into T_H17 under the activation of IL-6 and TGF-β and produce IL-17, a powerful pro-inflammatory cytokine. Overall, T_H1 and T_H2 have mutually inhibitory effects.[1] The development of CD4 T_H cells involves the encounter of antigen presented by antigen-presenting cells (APCs) in association with class II MHC. All cells express MHC class I, but only certain cells express class II MHC. These class II MHC–expressing cells constitute the body's population of APCs and consist of (1) dendritic cells, (2) macrophages, (3) B cells, (4) vascular endothelial cells, and (5) epithelial cells. The former three are considered "professional" APCs, because they are dedicated to this function. The latter two APCs are quiescent under normal conditions but can be induced to express class II MHC when exposed to elevated concentrations of IFN-γ.[1,116]

Dendritic cells and macrophages phagocytose foreign antigens, whereas B cells utilize the membrane-bound immunoglobulin to bind and internalize antigens. Other APCs endocytose foreign antigens into the cytoplasm for antigen processing. The processed antigens are partially degraded into small peptides. Many of them are 10 to 30 amino acids long and capable of binding onto the newly synthesized class II MHC molecules before the antigen/class II MHC complex is transported to the surface of cells and presented to TCRs of $CD4^+$ T cells.

Although controversial, evidence has suggested that $CD8^+$ T_S and $CD8^+$ CTLs represent distinct subpopulations of $CD8^+$ T cells. T_S are MHC class I–restricted $CD8^+/CD28^-$ T_S cells, which act on antigen-presenting cells (APC) by a contact-dependent manner, rendering them tolerogenic to T_H cells. They inhibit the proliferation of T_H cells.[50,316] T cytotoxic cells ($CD8^+T_C$), also known as *cytolytic T lymphocytes* (CTLs), are a subset of T cells that kill target cells expressing MHC-associated peptide antigens. The majority of T_C express CD8 and recognize antigens degraded in the cytosol and expressed on the cell surface in association with class I MHC molecules of the target cells. Functional T_C acquire specific membrane-bound

cytoplasmic granules, including a membrane pore-forming protein called *perforin* or *cytolysin* and enzymes called *granzymes*.[1]

The role of B cells in adaptive immunity is mainly the production of antibodies that constitute the host humoral immune response. The V(D)J gene recombination occurs in both heavy and light (κ and λ) chains. A recombinase system, which is an enzymatic complex consisting of several enzymes—including recombination activating gene 1 and 2 (RAG-1, RAG-2), terminal deoxynucleotidyl transferase (TdT), DNA ligase IV, Ku proteins, and XRCC4—is essential for successful recombination. This recombinase system is also used for TCR recombination. Mature IgM/IgD coexpressing B cells undergo isotype switching via a process called *switch recombination* after encountering antigen. The rearranged V(D)J gene segment recombines with a downstream C region gene (γ, ε, or α), and the intervening DNA sequence is deleted. This gives rise to other classes of immunoglobulins (IgG, IgE, IgA) besides IgM. In addition to isotype switching, activated B cells also undergo somatic mutation in the V region gene, leading to affinity maturation of antibodies and alternative splicing of VDJ RNA to membrane or secreted immunoglobulin mRNA. A large quantity of antibody is secreted when B cells terminally differentiate into plasma cells.[1,116] The ability of antigens to selectively stimulate the differentiation of plasma cells supports the clinical finding that plasma cells isolated from periapical lesions secrete antibodies specific for the particular bacteria found in the adjacent root canal system.

The immune response and the role of lymphocyte subpopulations in apical periodontitis lesions were investigated by employing lymphocyte-deficient rodents as study models. T-cell deficiency appears to accelerate bone loss at the early phase of apical periodontitis lesions (2 weeks) but does not affect the overall course of lesion development.[271,295] Using RAG (recombination activating)-2 SCID mice (both T- and B-cell-deficient), it was found that approximately a third of the RAG-2 mice developed endodontic abscesses, whereas no immunocompetent controls had abscesses.[273] In another study, specific RAG 2 knockout (k/o) mice were used to determine which immune element was important for the defense mechanism in endodontic infection. The results demonstrated that B cells, but not T cells, played a pivotal role in preventing dissemination of endodontic infection.[105] Therefore, both T and B cells mediate the observed immune responses in apical periodontitis lesions.[170]

Neurogenic Inflammation

Certain primary afferent nerve fibers, upon stimulation by various irritants, release neuropeptides, which cause vasodilation, protein extravasation, and recruitment/regulation of immune cells such as macrophages, neutrophils, mast cells, and lymphocytes. This phenomenon is termed *neurogenic inflammation*.[26,29] Pivotal neuropeptides in the induction of neurogenic inflammation are CGRP, for vasodilation, and SP, for the induction of protein extravasation. Neuropeptides and their receptors are widely distributed throughout the body. During inflammation, there is a sprouting of afferent fibers[38,39,96] and local increases in inflammatory mediators that trigger neuropeptide release, leading to neurogenic inflammation.[26,43] Besides the cardinal functions of the key neuropeptides that cause the first sign of inflammation—vasodilation and increased vascular permeability—the role of these neuropeptides in the process of inflammation is now known to be far more complex. In the development of chronic apical periodontitis lesions, neuropeptides are also involved in immune regulation, bone resorption, and wound healing. At sufficient concentrations, SP increases the secretion of IL-1, TNF-α, and IL-6 from macrophages and stimulates T-lymphocyte proliferation and enhances antigen-induced IFN-γ production by T cells.[253] Certain neuropeptides, such as SP, upregulate immune and inflammatory responses, whereas other neuropeptides, such as vasoactive intestinal peptide (VIP) and neuropeptide Y (NPY), inhibit inflammatory responses. Synergistic interactions among neuropeptides, such as CGRP and other inflammatory mediators, eicosanoids, and bradykinin, suggest a complex interplay between these molecules in the immune response.[83,211,253]

In chronic apical periodontitis lesions, specific receptors for SP and CGRP are expressed in certain immune cells, including macrophages and lymphocytes. Both CGRP and VIP may play a role in inhibiting bone resorption by suppressing osteoclastic functions. The level of VIP in apical periodontitis lesions is inversely related to lesion size. Osteoclast cell culture studies have demonstrated that the presence of greater concentrations of VIP leads to a decrease in their ability to form lacunae of osseous resorption, causing rapid cytoplasmatic contraction and reduced cell mobility. VIP exerts an effect on macrophages to block the production of TNF-α, IL-6, and IL-12, suggesting that VIP could have a role in controlling growth of apical periodontitis lesions.[43]

The major innate and adaptive immune responses and neurogenic inflammation in the pathogenesis of apical periodontitis caused by root canal infection are illustrated in Fig. 15-1.

DIAGNOSIS

Correlation Between Clinical and Histologic Findings

Clinical diagnosis of inflammatory periapical disease is mainly based on clinical signs or symptoms, duration of disease, pulp tests, percussion, palpation, and radiographic findings (see also Chapter 1). In contrast, a histologic diagnosis is a morphologic and biologic description of cells and extracellular matrix of diseased tissues. The clinical diagnosis represents a provisional diagnosis based on signs, symptoms, and testing results, whereas the histologic diagnosis is a definitive diagnosis of tissue disease.

Similar to pulpitis,[231,238] apical periodontitis is not always symptomatic or painful. Although many inflammatory mediators (histamine, bradykinin, prostaglandins) and proinflammatory cytokines (IL-1, IL-6, TNF, nerve growth factor [NGF]), as well as neuropeptides (SP, CGRP), are capable of sensitizing and activating nociceptive sensory nerve fibers,[96,97] other mediators such as endogenous opioids and somatostatin released by inflammatory cells during inflammation are able to inhibit firing of sensory nerve fibers.[102,236] Activation of nociceptive sensory nerve fibers may also be related to concentrations of inflammatory mediators. The complexity of these findings supports the clinical observation that there is no good correlation between clinical symptoms and histopathologic findings of apical periodontitis.[27,159] For example, many teeth with apical periodontitis are free of symptoms.

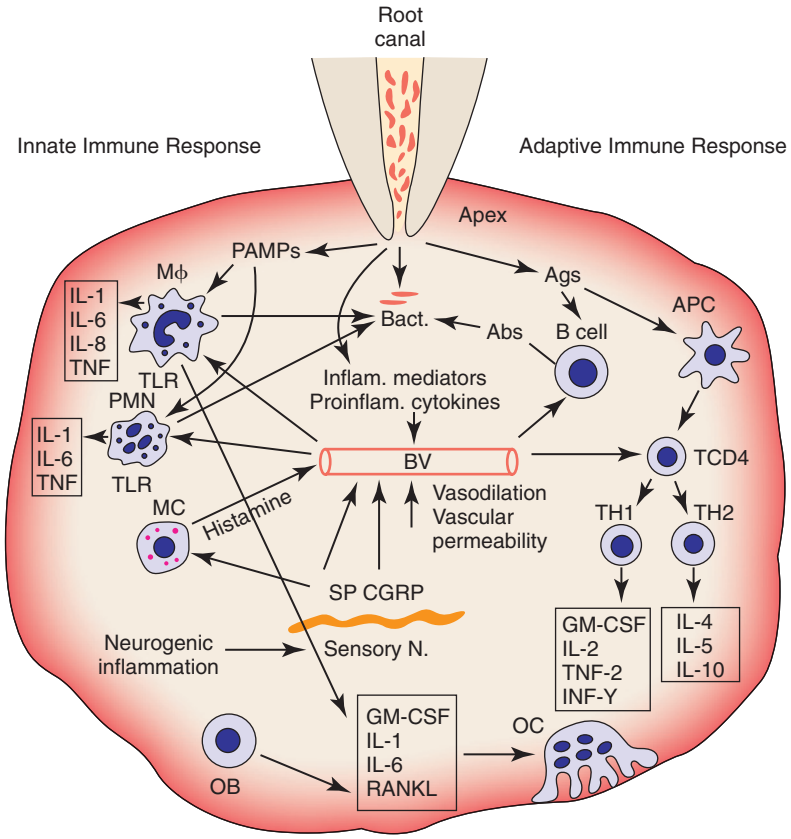

FIG. 15-1 Major innate and adaptive immune responses and neurogenic inflammation in the pathogenesis of apical periodontitis. *APC,* antigen presenting cell; *GM-CSF,* granulocyte/monocyte colony-stimulating factor; *MC,* mast cell; *Mφ,* macrophage; *OB,* osteoblast; *OC,* osteoclast; *PAMPs,* pathogen-associated molecular patterns; *PMN,* polymorphonuclear leukocyte; *RANKL,* receptor activator of nuclear factor κB ligand; *TLR,* Toll-like receptor. (Courtesy Dr. Lin.)

Correlation Between Radiographic and Histologic Findings

Radiography is designed to detect pathologic changes at tissue not cellular levels. Even using sensitive imaging systems such as ultrasound, cone-beam computed tomography, and other technologies, it is impossible to detect the presence of inflammatory cells or other subtle changes in the periapical tissues. Using conventional radiographic and histologic methods in the same cadavers, evidence of inflammation was often observed in the periapical tissues of endodontically treated teeth with normal radiographic features.[17,36,85] This finding is supported by the fact that lesions localized in the cancellous bone may not be visible radiographically unless they involve cortical bone (see also Chapter 2).[20,22,109] In addition, radiographic findings are unable to predict asymptomatic apical periodontitis (granuloma) from asymptomatic apical periodontitis with cyst formation (radicular cyst).[141,267] Accordingly, radiographic findings and histopathologic features of apical periodontitis have a poor correlation based on available case series studies.

The absence of clinical symptoms and negative periapical radiographic findings of endodontically involved teeth does not necessarily indicate absence of apical periodontitis.[17] By the same token, clinical success of endodontically involved teeth (i.e., absence of signs and symptoms and negative periapical radiographic findings) after nonsurgical root canal therapy does not necessarily imply complete histologic healing of periapical lesions. Thus, currently available diagnostic methods used in endodontics, such as percussion, palpation, and pulp tests (cold, heat, electric), are not sensitive enough to provide histologic diagnosis of an inflammatory periapical disease. In fact, all endodontic tests are basically used to examine the functions of nociceptive sensory nerves and not the pathologic changes of the pulp or periapical tissues. Until we have more advanced and sophisticated clinical diagnostic technologies, we will continue to face the problem of clinical diagnosis of inflammatory periapical disease. Nevertheless, the treatment of various types of apical periodontitis lesions is basically the same: nonsurgical root canal therapy. Interestingly, this treatment does provide a consistently good clinical outcome, as measured by either clinical signs of success or survival of the treated tooth. Future research should focus on developing testing methods that provide greater insight into the status of the periapical tissue.

HISTOPATHOLOGY

The study of the histopathology of diseased tissues and organs has gone through an interesting evolution. It began at macroscopic, light microscopic, and then electron microscopic observations of diseased tissues and organs. Nowadays, histopathology focuses on cellular and molecular biology of diseased tissues and organs. Biochemical disorders occur inside the cells before light and electron microscopic observable morphologic changes

of cell injury. Cell death (protein denaturation) occurs before observable morphologic changes of cell necrosis. Gene transformation or mutation occurs before observable morphologic changes of neoplastic cells.[165]

Based on etiology, clinical signs and symptoms, and duration of the disease process, the World Health Organization (WHO) classifies disease of periapical tissues into many categories.[307] There are also many classifications of inflammatory periradicular disease in several endodontic textbooks[110,203,282] and by the American Board of Endodontists, depending on clinical manifestations and histologic appearances. In addition, the American Association of Endodontists held a consensus conference on diagnostic terms in the fall of 2008. Traditionally, there has been a lack of consensus on clinical diagnostic terminology of pulpal and periapical disease in the endodontic community because of the paucity of studies with high levels of evidence. Because this chapter focuses on pathobiology of apical periodontitis, inflammatory periapical disease will be classified based on histologic appearances and cell biology of the injured periapical tissues. To avoid confusion between histologic and clinical diagnosis, readers are encouraged to read the chapters related to clinical examination, radiographic interpretation, and clinical diagnosis of inflammatory periapical disease (see Chapters 1, 2, and 3).

The histopathologic analysis of a diseased tissue or organ only shows structural changes of cells and extracellular matrix at the time the tissue or organ is removed. Therefore, it does not represent the complete kinetics or spectrum of disease development. Histologic classification of apical periodontitis is based on types of cells participating in inflammatory responses in the periapical tissues. In general, inflammation can be divided into acute and chronic responses, depending on types of cells present at the site of injured tissue.[137,165,248,299] Acute inflammatory response is characterized by participation of neutrophilic leukocytes and chronic inflammatory response by participation of macrophages and lymphocytes. However, many factors, such as severity of tissue injury, specific etiology, host's resistance, and particular tissue involved, can modify the course and morphologic variations as well as cell biology of both acute and chronic inflammatory responses.[137,165] Acute and chronic inflammatory responses are in fact not rigid phases of a single programmed event but two overlapping responses with partially different triggering mechanisms and programs.[165]

SYMPTOMATIC APICAL PERIODONTITIS

It is a general belief that the development of apical periodontitis follows total pulp necrosis. This belief is based on (1) the pulpal strangulation theory due to a generalized increase in pulpal interstitial pressure inside the uncompromised pulp space during pulpal inflammation that causes collapse of venules and cessation of blood flow[96] and (2) animal and human studies that concluded that uncontaminated necrotic pulps that are intentionally devitalized or accidentally traumatized are generally incapable of inducing periapical inflammation unless they are infected.[156,178,261] However, if the vital pulps become infected due to caries or other pathways, periapical inflammation can develop even when inflamed, but vital tissue is still present in the apical portion of the root canal. Most of our information related to the histopathology of apical periodontitis comes from analysis of longstanding, chronic human lesions caused by caries or from time-course studies of development of apical periodontitis induced by artificial root canal infection in animals. In these instances, the moment of transition from pulpitis to apical periodontitis was not captured. In fact, apical periodontitis has been demonstrated to be a direct extension of apical pulpitis into the periapical tissues before total pulp necrosis caused by root canal infection (Fig. 15-2).[135,154,155,214,215,268] For example, Kovacevic and colleagues[135] studied the transition from pulpitis to apical periodontitis in dogs' teeth by artificial exposure of pulps to the

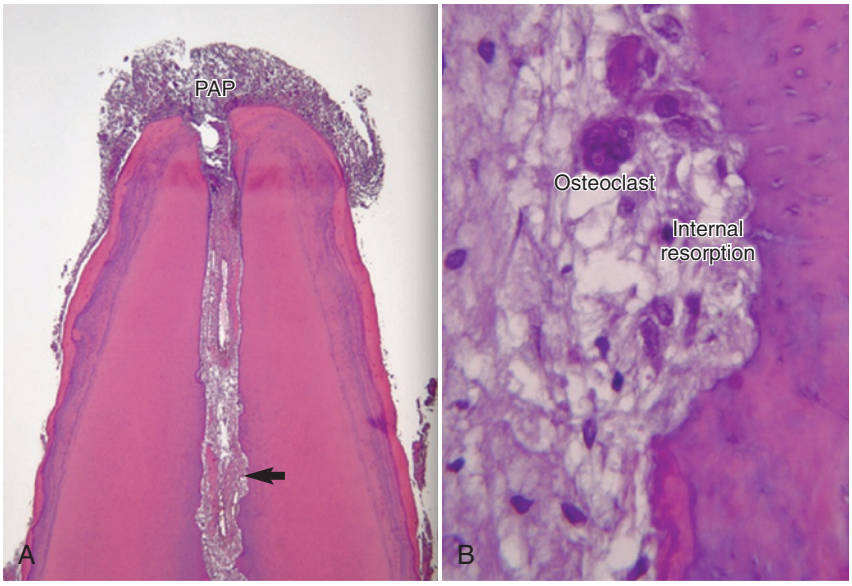

FIG. 15-2 A, Inflammation of the pulp tissue in the apical root canal extends into the periapical tissues in a mature tooth (H&E, magnification: ×100). B, Arrow in (A). High magnification of the pulp tissue in the apical root canal in A. The pulp tissue is vital and infiltrated with chronic inflammatory cells. Note resorption of canal wall and multinucleated clast cells (H&E magnification: ×200). (Courtesy Dr. Domenico Ricucci, Rome, Italy.)

oral cavity and observed that pulpitis was coupled with an acute apical periodontitis. Similarly, Kimberly and Byers[128] demonstrated that periapical changes, including sprouting of nerve fibers, appeared 3 to 5 weeks following establishment of irreversible pulpitis subsequent to pulp exposure lesions in animals. Yamasaki and coworkers[310] and Stashenko and colleagues[255] also showed that periapical inflammatory infiltrates, increased osteoclast numbers, and bone destruction were apparent well in advance of total pulpal necrosis, with vital pulp tissue still present in the apical portion of the root canal. The biologic basis for these observations appears to hinge on the apical development of pulpal infection/inflammation leading to the diffusion of many inflammatory mediators, proinflammatory cytokines, chemokines, and bacterial toxins into the periapical area[154] prior to total pulpal necrosis.

The development of acute apical periodontitis largely reflects the innate immune system and is the first line of active defense against irritants from the root canal. Acute apical periodontitis is an immediate defense reaction to irritants and does not require exquisite specificity and memory. Characteristic features of acute apical periodontitis are similar to the typical acute inflammatory reaction and consist of vasodilation, increased vascular permeability, and transmigration of leukocytes from the blood vessels into perivascular tissue space. The beneficial actions of acute inflammation are (1) infiltration of leukocytes to the injured tissue to phagocytose and kill microbial agents; (2) accumulation and activation of humoral factors such as immunoglobulins, complement factors, and plasma proteins in the injured tissue to recruit more neutrophils and macrophages; and (3) neutralization or degradation of bacterial toxins and their harmful metabolic by-products.[137,165]

Cell Biology

The inflammatory response is a dynamic interaction between host defense mechanisms and microbial insults. The interlacing activation and control pathways of cellular and humoral components involved in the inflammatory response are complex. The cells involved—neutrophils, monocytes/macrophages, platelets, mast cells, T lymphocytes, B lymphocytes, NK cells, dendritic cells, endothelial cells, fibroblasts, eosinophils, and basophils—each have numerous functions that are activated and modulated by a multiplicity of biochemical messengers.[1,137,165] *Cell activation* means that the cell acquires the ability to perform one or more new functions or to perform normal functions at a higher rate[1]; it often results in transcription of new genes and synthesis of new proteins.[1]

Mast Cells

Histamine stored in the cytoplasmic granules of mast cells is the mediator that first appears in acute inflammation to induce vasodilation and increased vascular permeability; mast cells are the designated triggers of acute inflammation. They are widely distributed in perivascular tissue spaces and originate in the bone marrow from precursor cells. Mature mast cells contain numerous cytoplasmic granules, which are the source of vasoactive mediators. The preformed histamine is released by mast cell degranulation and can be triggered by (1) physical stimuli such as cold, heat, and mechanical trauma; (2) binding of IgE-specific antigen to mast cells and membrane-bound IgE antibodies; (3) binding of complement components (C3a and C5a) to their complementary receptors on mast cells; and (4) stimulation by neuropeptide (SP) and cytokines (IL-1, IL-8).[1,29,137,165] In addition, activated mast cells secret cytokines (TNF-α, IL-1, IL-3, IL-4, IL-5, IL-6, IL-13), prostaglandins, and leukotrienes to enhance inflammatory defense mechanisms.[1,37,126]

Endothelial Cells

Endothelial cells are important players in the inflammatory response. Without the participation of endothelial cells, the host is unable to deliver its cellular and humoral defense components from the circulating blood to the site of tissue injury. Inflammatory mediators, complement components, proinflammatory cytokines, nitric oxide, neuropeptides, and bacterial toxins can all affect endothelial cells, resulting in vasodilation and increased vascular permeability.[1,137,165] IL-1 and TNF released by activated macrophages and NK cells can stimulate endothelial cells to express intercellular adhesion molecules (ICAMs), such as ICAM-1, ICAM-2, ICAM-3, vascular cell adhesion molecule (VCAM), and platelet endothelial cell adhesion molecule (PECAM), which enhance leukocyte adhesion to endothelial cells and transmigration through the blood vessels.[1,137,148,165] IL-1, TNF, and LPS also can activate endothelial cells to synthesize chemokine (IL-8), a potent chemotactic mediator for neutrophils.[1]

Polymorphonuclear Neutrophilic Leukocytes

Polymorphonuclear neutrophilic leukocytes (PMNs) are the principal effector cells in acute apical periodontitis. They are derived from bone marrow stem cells. Neutrophils have a lobulated nucleus and contain primary or azurophil (elastase and myeloperoxidase) and secondary or specific (lysozyme and other protease) granules in their cytoplasm.[126,137,165] Neutrophils are only present in the blood circulation. They are the first leukocytes to transmigrate through the blood vessels into perivascular tissue space and then are directed toward the wound or irritants, peaking at 24 to 48 hours. The transmigration of neutrophilic leukocytes from the blood vessels into perivascular space involves complex cellular and molecular biology. Following vasodilation and increased vascular permeability, leukocyte margination, rolling, capture, and activation in the blood vessel and then transmigration through the blood vessel are mediated by an intricate interaction of cell adhesion molecules expressed on leukocytes (L-selectin, integrins) and on endothelial cells (P- and E-selectins, ICAM, VCAM, PECAM-1). Neutrophil rolling is mediated by interaction between leukocyte selectin ligands and P-selectins on endothelial cells. Leukocyte sticking is mediated by interaction between leukocyte integrins and ICAMs and VCAMs on endothelial cells. Leukocyte transmigration is mediated by interaction between PECAM-1 on both leukocytes and endothelial cells.[165] Chemokines (IL-8) increase the affinity of leukocyte integrins for their ligands on endothelial cells.[1] Once transmigrating through the junction between endothelial cells and basement membrane into the perivascular tissue space, neutrophilic leukocytes are directed toward stimuli by chemotactic factors or chemotaxins, such as bacterial products (fLMP), C3a, C5a, leukotriene B4 (LTB4), platelet-activating factor (PAF), fibrinopeptides, dead cells, and chemokines (IL-8) by receptor-mediated mechanisms.[1,137,165,248]

Neutrophils can be activated by bacteria and pathogen-associated molecular patterns (also known as the TLRs, described earlier). They can also be stimulated by IL-1, TNF, and chemokines produced by activated macrophages and NK cells to enhance the phagocytic activity of infectious agents and

synthesis of defensins, which are broad-spectrum antibiotics.[1] Neutrophilic leukocytes are terminally differentiated cells and short lived—within hours to a few days. Most neutrophilic leukocytes in acute inflammatory response die as a result of apoptosis or programmed cell death. The apoptotic neutrophils are phagocytosed by macrophages.[89,137,165] However, some neutrophilic leukocytes die after a furious battle against microbial infection and release intracellular proteolytic lysosomal enzymes, oxygen-derived active metabolites, nitric oxide, proinflammatory cytokines, eicosanoids, and matrix metalloproteinases into tissue to intensify inflammation and tissue damage.[137,165] The release of lysosomal enzymes by neutrophils and macrophages can also occur by lysosomal suicide due to rupture of phagolysosome in the cytosol, regurgitation during phagocytosis of irritants, or frustrated phagocytosis of indigestible foreign bodies.[137,165] The main effector functions of neutrophilic leukocytes are phagocytosis, killing of microbes, and release of inflammatory mediators (including proinflammatory cytokines) to recruit more leukocytes to prevent spread of infection.

Macrophages

Macrophages make their appearance as a second wave in acute apical periodontitis within 48 to 96 hours. Macrophages are blood-borne but have a counterpart in the connective tissue.[165] Blood monocytes transmigrate through the blood vessel into the tissues and become macrophages. Mature macrophages have an irregular-shaped nucleus and contain abundant lysosomes and many phagocytic vesicles (phagosomes). Monocytes use mechanisms similar to neutrophils to adhere to endothelial cells in high endothelial venule–expressing adhesion molecules for mononuclear leukocytes (ICAM for macrophages and VCAM for lymphocytes); they then transmigrate through the blood vessel and are directed toward the site of tissue injury by chemotactic factors.[165]

Macrophages can be activated by bacteria, PAMPs, and interferon-*r* (INF-*r*) and produce numerous products: lysosomal enzymes, coagulation factors, bioactive lipids, reactive oxygen species, chemokines, cytokines/growth factors, and angiogenesis factors.[137,165,248] Macrophages are the most dynamic and versatile leukocytes. The main functions of macrophages are numerous. They include phagocytosis of microbes and foreign bodies, production of inflammatory mediators, initiation of the immune response, cleanup operation of necrotic cells and tissue, induction of systemic effects (fever, acute phase reaction, cachexia), synthesis of molecules or cytokines affecting cell and vascular growth in wound healing, as well as antibacterial and antiviral defenses.[165] Tissue macrophages are not terminally differentiated cells and are able to undergo mitosis. Their life span is from several weeks to months.

Activated neutrophilic leukocytes and macrophages are capable of phagocytosing and killing pathogens. They recognize microbes through TLRs and receptors for the Fc fragment of immunoglobulin IgG as well as complement component C3b on their cell surface. C3b opsonization and antibody coating of microbes enhance recognition and phagocytosis of pathogens by activated neutrophilic leukocytes and macrophages.[1,137,165] Activated neutrophilic leukocytes are effective in phagocytosing and killing extracellular microbes, whereas macrophages activated by IFN-*r* produced by NK cells and T_H1 cells are more effective in phagocytosing and killing intracellular microbes.[1] Killing of phagocytosed microbes by neutrophilic leukocytes and macrophages is mediated through oxygen-dependent and oxygen-independent mechanisms. Oxygen-dependent mechanisms are more effective in killing all kinds of bacteria than oxygen-independent mechanisms.[1,137,248] The effector molecules of an oxygen-dependent system are hydrogen peroxide, superoxide anion, hydroxyl radical, singlet oxygen, and hypochlorite. An oxygen-independent system is also important in killing microbes and is dependent on lysozymes, defensins, and lactoferrin contained in phagocyte granules.[1,137,165,248] Some antimicrobial peptides and other oxygen-independent mechanisms possessed by phagocytes are specialized for killing of certain groups of microbes.[1,248]

Microbes phagocytosed by phagocytes are enclosed in membrane-bound phagosomes in the cytosol. The phagosomes fuse with lysosomes to form phagolysosomes. Irritants such as microbes, foreign protein antigens, and dead cells inside the phagolysosome are destroyed or degraded by proteolytic enzymes stored in lysosomes.[1] There are also granules that are fused with the nascent phagosome and release their contents into the phagosome. Some of these granules have direct antimicrobial action, such as defensins and the bactericidal permeability-increasing protein azurocidin. Others are proteases, such as elastase and cathepsins, lactoferrin, and peroxidase myeloperoxidase. Lysosomal enzymes, reactive oxygen intermediates, and nitric oxide released by neutrophils and macrophages indiscriminately kill not only bacteria but also tissue cells. Much of periapical tissue damage that occurs during acute inflammation can be attributed to release of proteolytic lysosomal enzymes and matrix metalloproteinases from disintegrated neutrophilic leukocytes and macrophages rather than to bacteria and their toxins.[1,165]

Platelets

Platelets normally circulate in the blood but also play an important role in inflammation. They are small cytoplasmic fragments derived from the megakaryocyte.[126] Platelets are essential for blood clotting, hemostasis, and fibrinolysis. Platelets produce vasoactive amines (PAF, serotonin), chemokines, and growth factors (PGDF, FGF, TGF) during inflammation.[165]

Natural Killer Cells

NK cells may also be players in acute apical periodontitis. They are a subset of lymphocytes found in blood and lymphoid tissues. NK cells are derived from the bone marrow stem cells but lack the specific T-cell receptor for antigen recognition.[1] NK cells also possess TLRs for microbial constitutive and conserved products. The effector functions of NK cells are to lyse virus-infected cells without expressing class 1 MHC molecules and to secret IFN-*r* to activate macrophages. Antibody-coated cells, cells infected by viruses, some intracellular bacteria, and IL-2 released by activated macrophages can activate NK cells. Viruses have been isolated in apical periodontitis lesions.[218,249] NK cells kill target cells by antibody-dependent cell-mediated cytotoxicity because NK cells express receptor for the Fc fragment of IgG.[1] NK cells provide a link between the innate and adaptive immune systems.[115]

Inflammatory Mediators

Numerous biochemical mediators are involved in the acute innate inflammatory response. They are mainly derived from

TABLE 15-2

Major Mediators in Inflammation

Mediator	Source	Effector Cells and Tissues
Vasodilator		
Histamine	Mast cells Platelets	Endothelial cells
Prostaglandins	Leukocytes Mast cells	Endothelial cells
Nitric oxide	Endothelial cells Macrophages	Vascular smooth muscle
CGRP	Sensory nerve	Endothelial cells
Fibrin degradation products	Plasma	Endothelial cells
Increased Vascular Permeability		
Bradykinin	Plasma	Endothelial cells
Leukotrienes C4, D4, E4	Leukocytes Mast cells	Endothelial cells
PAF	Leukocytes Mast cells Endothelial cells	Endothelial cells
C3a, C5a	Plasma	Endothelial cells
Fibrinopeptides	Plasma	Endothelial cells
Substance P	Sensory nerve	Endothelial cells
Increased Expression of Endothelial Adhesion Molecules (Selectin, ICAM, VCAM, PEAM)		
TNF	Activated macrophages NK cells	Endothelial cells
IL-1	Activated macrophages NK cells	Endothelial cells
Chemokines	Macrophages Neutrophils Endothelial cells Fibroblasts	Endothelial cells
Leukocyte Activation and Chemotaxis		
C3a, C5a	Plasma	Leukocytes
Leukotriene B4	Mast cells Leukocytes	Leukocytes
Chemokines (IL-8)	Macrophages Neutrophils Endothelial cells Fibroblasts	Leukocytes
Fibrinopeptides	Plasma	Leukocytes
Bacterial products (fMLP)	Bacteria	Leukocytes
TNF	Activated macrophages Dead cells	Leukocytes
Opsonins		
C3b, C5b, immunoglobulins	Plasma	Microbes
Tissue Damage		
Lysosomal enzymes	Neutrophils Macrophages	Cells and tissues
Free oxygen radicals	Activated leukocytes	Cells and tissues
Nitric oxide	Macrophages	Cells and tissues
Bacterial products (LPS)	Bacteria	Cells and tissues

CGRP, calcitonin gene–related peptide; *fMLP,* formyl-methionyl-leucyl-phenylalanine; *IL-1,* interleukin-1; *PAF,* platelet activation factor; *NK cell,* natural killer cell; *TNF,* tumor necrosis factor.

Data from Kumar V, Abbas AK, Fausto N, et al: *Robbins and Cotran pathologic basis of disease*, ed 8, Philadelphia, 2010, Saunders; Slauson DO, Cooper BJ: *Mechanisms of disease*, ed 3, St Louis, 2002, Mosby; Majno G, Joris I: *Cell, tissues, and disease*, ed 2, Oxford, 2004, Oxford University Press; Abbas AK, Lichtman, Pober JS: *Cellular and molecular immunology*, ed 5, Philadelphia, 2003, Saunders.

plasma and cells. The main biologic functions of inflammatory mediators are to cause vasodilation and increased vascular permeability and recruit inflammatory cells, mainly neutrophilic leukocytes and macrophages from blood circulation to the site of tissue injury. Some mediators can also cause tissue damage. The major mediators involved in vascular changes, cell recruitment, and tissue damage in the acute inflammatory response are listed in Table 15-2.

All listed inflammatory mediators have been shown to be present in apical periodontitis.[278,279] Bradykinin is the product of kinin-system activation; fibrinopeptides, the products of blood-clotting system activation; and fibrin degradation products, the products of fibrinolytic-system activation. Kinin, fibrinolytic, and clotting systems are initiated by activated Hageman factor. Prostaglandins and leukotrienes are the products of arachidonic acid metabolism. Activated phospholipase A2 splits cell membrane phospholipid molecules into arachidonic acid and platelet activating factor. Arachidonic acid molecules can be processed along two pathways: the cyclooxygenase pathway, which leads to production of prostaglandins and thromboxanes, and the lipoxygenase pathway, which produces leukotrienes.[137,165]

Complement components, such as C3a, C3b, C5a, C5b, and C5-C9, are products of the complement cascade, which can be activated by two pathways. The classic pathway is initiated by activation of C1 by multimolecular aggregates of IgG or IgM antibody complexed with specific antigen. The alternate pathway is activated by microbial cell components (lipopolysaccharide, teichoic acid) and plasmin. C3a and C5a are anaphylatoxins that stimulate mast cells and basophils to release histamine. They also cause phagocytes to release lysosomal enzymes. C3b is an opsonin and can coat bacteria to enhance phagocytosis by phagocytes. In addition, C3b can also bind to antibody bound to antigen or microbes. C5a is a strong

chemotaxin for neutrophils. C5b-C9 is a membrane attack complex and able to cause cell lysis if activated on the host cell and bacterial cell membrane.[137,165]

Besides inflammatory mediators, the inflammatory response is also dependent upon the timing and extent of various cytokine and chemokine secretions. IL-1, TNF, IL-6, IL-12, and IFN-γ are present in the acute inflammatory response.[1,91] IL-6 is produced by activated mononuclear phagocytes, endothelial cells, and fibroblasts in response to microbial infection and other cytokines, such as IL-1 and TNF. IL-6 stimulates the synthesis of acute-phase proteins by hepatocytes.[1] The major source of IL-12 is activated mononuclear phagocytes and dendritic cells. IL-12 provides a link between innate and adaptive immune responses.[1] IFN-*r* is produced by activated NK cells, T_H1 cells, and cytotoxic T cells and can activate macrophages and enhance their microbial killing ability. IL-1β and TNF are associated with apical bone resorption during chronic apical periodontitis.[252]

Chemokines are a large family of structurally homologous cytokines that stimulate leukocyte movement and regulate the transmigration of leukocytes from the blood vessel to tissue space.[1] They are produced by leukocytes, endothelial cells, and fibroblasts. The secretion of chemokines is induced by microbial infection, TNF, and IL-1.[1,91,240] Different types of leukocytes express different chemokine receptors.[1]

Neuropeptides are released via axon reflexes of afferent sensory neurons in response to various stimuli. Neuropeptides, as mediators of the inflammatory process, are described in detail in the Neurogenic Inflammation section of this chapter. Inflammatory mediators such as histamine, kinins, prostaglandins, and proinflammatory cytokines are capable of sensitizing and activating sensory nerves to release neuropeptides.[29,96,97,213,225]

Histopathology

The acute inflammatory response is practically immutable in all vascularized living tissues, largely due to the programmed actions of the innate immune system. Initially, blood vessels are engorged by a local infiltration of inflammatory cells, mainly activated neutrophilic leukocytes and some macrophages in the apical periodontal ligament of the infected/inflamed root canal. In addition, sprouting of sensory nerve fibers has been shown early in the inflamed periapical tissues.[39,128] Several studies have also demonstrated the presence of inflamed vital pulp tissue with intact nerve fibers in the apical portion of the root canal in association with apical periodontitis.[154,214,215,255,310] This explains the clinical observation that a patient may experience some pain if an instrument is introduced into the canal short of the apex in some teeth with apical periodontitis lesions.

In primary acute apical periodontitis, apical bone destruction is usually not observed radiographically because the duration of acute response is short, and activated neutrophilic leukocytes and macrophages are not able to resorb bone. Only osteoclasts are capable of resorbing bone, and they have to differentiate from the monocyte/macrophage cell lineage in the blood circulation. However, in a rat model experiment, Stashenko and colleagues[255] showed histologically that periapical inflammatory cell infiltration increases osteoclast numbers, and that bone destruction was apparent well in advance of total pulpal necrosis.

Bacteria are usually not present in acute apical periodontitis lesions.

Clinical Features

As discussed earlier, there is no correlation between clinical and radiographic findings and histologic appearance of inflammatory periapical disease. Teeth with acute apical periodontitis are usually symptomatic and painful to bite and percussion, which results from mechanical allodynia and hyperalgesia.[125] Pain is induced by sensitization and activation of nociceptive sensory nerve fibers by inflammatory mediators, proinflammatory cytokines, nerve growth factor, and pressure.[125] Sprouting of sensory nerve fibers in inflamed periapical tissues could also increase receptive field size in teeth with apical periodontitis.[38,39] Radiographic examination usually does not show periapical bone destruction of the involved tooth in acute apical periodontitis, although occasional slight widening of the apical periodontal ligament space and loss of the apical lamina dura of the involved tooth may be present.

Outcomes

The fundamental purpose of the acute inflammatory response is to restore the structural and functional integrity of damaged tissue by eliminating irritants as soon as possible.[137,165,299] Tissue damage can also occur in acute inflammation by release of lysosomal enzymes, toxic oxygen radicals, and nitric acid from disintegrated neutrophilic leukocytes and macrophages into tissue.[165] Depending on the dynamic interaction between host defenses and microbial insults, acute apical periodontitis can result in (1) restitution of normal periapical tissues if irritants are immediately eliminated by root canal therapy, (2) abscess formation if massive invasion of periapical tissues by highly pyogenic bacteria occurs, (3) organization by scarring if extensive destruction of periapical tissues results, or (4) progression to chronic apical inflammation if irritants continue to persist.

Abscess is a focal localized collection of purulent exudate in a tissue or an organ.[137] It is a morphologic variation of acute and chronic inflammation.[137,165,299] The development of an abscess in apical periodontitis lesions is probably caused by invasion of a combination of specific pyogenic bacteria in the inflamed periapical tissues.[32,201,263,291] Neutrophilic leukocytes are the predominant cells in acute apical periodontitis with abscess formation. Abscess begins as a furious battle between highly virulent pathogens and an army of neutrophilic leukocytes. The pathogens produce massive toxins to kill neutrophils. As neutrophils attack the pathogens, they secrete lysosomal enzymes that digest not only the dead cells but also some live ones. Many neutrophils die fighting against pathogens. The resulting purulent fluid is poorly oxygenated and has a low pH. The bactericidal ability of leukocytes appears to be impaired because of the deprivation of oxygen and interference of respiratory burst. The purpose of respiratory burst is to generate bactericidal agents.[15,165] However, the phagocytic activity of leukocytes is not impaired in anaerobic conditions.[165]

Histologically, apical abscess formation is characterized by local collection of suppurative or purulent exudate composed of dead and live neutrophilic leukocytes, disintegrated tissue cells, degraded extracellular matrix, and lysosomal enzymes released by dead neutrophilic leukocytes. It also contains dead and live bacteria and bacterial toxins released by dead bacteria in the inflamed periapical tissues. Abscess formation also involves destruction of periodontal ligament and sometimes periapical bone—especially in chronic apical periodontitis

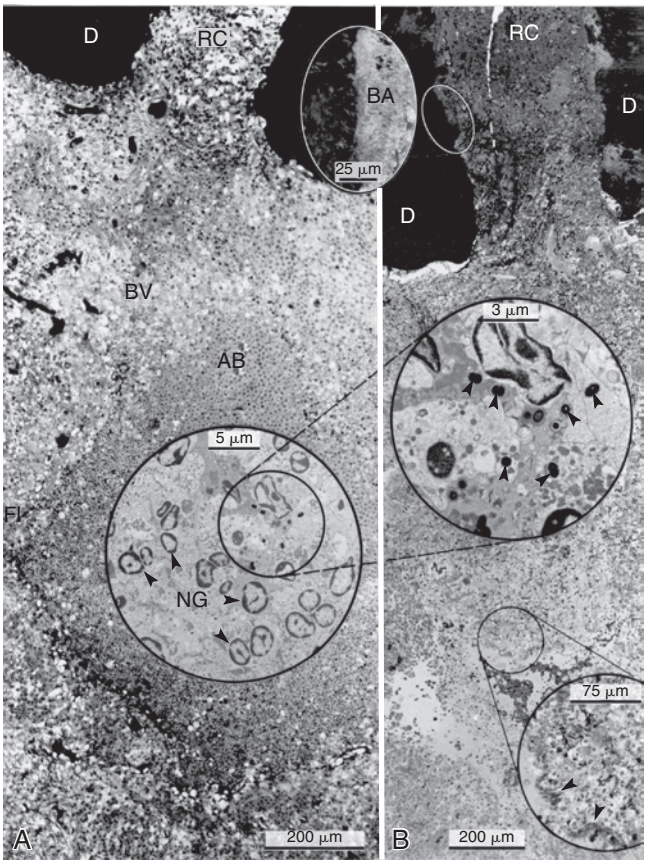

FIG. 15-3 Structure of a secondary periapical abscess. **A,** Axial section of an abscessed apical periodontitis. The microabscess (AB) contains a focus of neutrophils (NG inset in **A**). Note the phagocytosed bacteria in one of the neutrophils (further magnified in large inset in **B**). A secondary abscess forms when bacteria (BA in oval inset) from the apical root canal (RC) advance into the chronic apical periodontitis lesion (**B**). Note the tissue necrosis immediately in front of the apical foramen and the bacterial front in the body of the lesion *(arrowheads in lower inset). BV,* blood vessels; *D,* dentin. (*A,* magnification: ×130; *B,* magnification: ×100; oval inset, magnification: ×400; inset in *A,* magnification: ×2680; upper inset in **B,** magnification: ×4900; lower inset in **B,** magnification: ×250.) (From Nair PNR: Apical periodontitis: a dynamic encounter between root canal infection and host response, *Periodontol 2000* 13:121, 1997.)

with abscess formation—and a surrounding layer of viable neutrophilic leukocytes and a band of fibrovascular granulation tissue. Both layers are thought to serve as protective barriers to prevent the spread of infection. Epithelial cell proliferation is scanty in acute apical periodontitis with abscess formation (Fig. 15-3).[44,215,230]

Clinically, teeth with acute apical abscess formation usually have symptoms such as pain to biting and percussion. The periapical area of the involved tooth may be tender to palpation. Intraoral or extraoral swelling is often present. Because of a sudden outpouring of suppurative exudate in the periapical area, tissue pressure increases such that mechanical stimuli are capable of activating terminals of nociceptive neurons in the inflamed periapical tissues. The severe pain of an acute apical abscess can be due to activation of nociceptors by inflammatory mediators and sensitization to mechanical stimuli due to increased interstitial pressures.[232] If the periapical purulent exudate can be evacuated through the root canal (or through incision and drainage when indicated) during root canal therapy, the patient usually experiences an immediate relief of acute pain. Radiographically, the involved teeth may show slight widening of the apical periodontal ligament space to loss of apical lamina dura. In chronic apical abscess, the involved teeth may be symptomatic or asymptomatic. If an intraoral or extraoral draining sinus tract is present, swelling is usually absent. Radiographic bone destruction is obvious in teeth with chronic apical abscess formation.

In most cases, if the source of infection in the root canal is eliminated by root canal therapy, the abscess will heal by reabsorption of the pus in teeth with an acute apical abscess. Phagocytes will kill all bacteria in the abscess. The continued influx of leukocytes stops because the chemotactic stimuli have been removed, and the existing neutrophilic leukocytes die of apoptosis. Finally, macrophages move in to clean up necrotic neutrophilic leukocytes and disintegrated tissue cells. In chronic apical abscess formations, wound healing will take place mainly by means of regeneration and to some degree by tissue repair. However, if bacterial virulence and numbers of pathogens overwhelm the host's defenses, the abscess may break through the cortical bone, periosteum, and oral mucosa or facial skin to develop an intraoral or extraoral draining sinus tract. Sometimes, the uncontrolled abscess may spread along the fascial planes of the head and neck to develop serious cellulitis (see also Chapter 18).[142,230]

ASYMPTOMATIC APICAL PERIODONTITIS: APICAL GRANULOMA, CHRONIC APICAL PERIODONTITIS

If pathogens in the root canal are not eliminated, the symptomatic apical periodontitis may progress to become an asymptomatic apical periodontitis. Asymptomatic apical periodontitis is characterized by the persistence of inflammatory stimuli, adaptation of the host's response to stimuli, presence of adaptive immune responses, and initiation of the repair process.[137,165,248,299] Chronic inflammation is good news and bad news. The good news is that the host's defenses are able to maintain an active defense against the invading microorganisms and toxins; the bad news is that the host response is inadequate to eliminate these factors.[165]

Asymptomatic apical periodontitis is a form of adaptive immune response that requires exquisite specificity and memory. The adaptive immune response enhances bacterial killing compared with the innate immune response. Traditionally, the terms *asymptomatic chronic apical periodontitis* and *periapical granuloma* are used interchangeably. A granuloma is a focal area of *granulomatous inflammation,* which is a histologic term for a chronic inflammatory reaction.[137,165,294] Granulomatous inflammation is characterized by the presence of activated macrophages with modified epithelioid cells in diseases such as tuberculosis, leprosy, syphilis, cryptococcosis, sarcoidosis, rheumatic fever, and foreign body granuloma.[101,137,165,304] The presence of poorly digestible irritants (nonantigenic or antigenic), T cell–mediated immunity to irritants, or both appears to be necessary for granuloma formation.[1,137,165] A granuloma is relatively avascular,[137,165] whereas a chronic apical periodontitis is very vascular. Histologically, some but not all chronic apical periodontitis lesions may show some features of granulomatous inflammation,[137,165] so the terms *apical granuloma* and *asymptomatic chronic*

apical periodontitis should not be used interchangeably. A *granuloma* is best considered a histologic term used to describe a specific form of chronic inflammation such as foreign body granuloma or immune granuloma.[137,165]

The foreign body reaction is a specific subtype of chronic inflammation.[137,165,299] Foreign materials such as root canal filling materials, paper points, cotton fibers, and surgical sutures can trigger a foreign body giant cell granuloma.[133,187,313] If activated macrophages are unable to engulf large indigestible foreign particles, they can fuse to form giant cells on the surface of the particles and continuously release lysosomal enzymes, inflammatory mediators, and proinflammatory cytokines as a result of frustrated phagocytosis. Giant cells appear to be at least as active metabolically as a regular macrophage.[165] In addition, foreign bodies can favor infection in several ways, as they can be a source of bacterial biofilm[51,197] and they lower the infectious dose of bacteria to induce infection. For example, if a small, sterile plastic cage is implanted under the skin of a guinea pig, as few as 100 *Staphylococcus aureus* are sufficient to infect the tissue, whereas even 10^9 bacteria (i.e., a million-fold increase in dose) fails to produce an abscess in normal guinea pig skin.[315] Finally, foreign bodies can make the infection hard to treat because bacteria in biofilms can switch on appropriate genes and cover themselves with a thick layer of biopolymer that is resistant to both host defense mechanisms and antimicrobial agents.[118,165]

Cell Biology

Macrophages and Lymphocytes

Macrophages and lymphocytes are the primary players in asymptomatic apical periodontitis.[3,52,79,122,132,258,259,280,312] Lymphocytes are blood-borne and have counterparts in the connective tissue.[165] Macrophages play a dual role in host defenses. In the innate immune response, activated macrophages phagocytose microbes, dead cells, and foreign bodies and produce inflammatory mediators and proinflammatory cytokines to enhance the host defense against stimuli. In the adaptive immune response, activated macrophages function as APCs. They phagocytose and present the processed foreign antigens in association with MHC to T cells. Thus, activated macrophages are effector cells of adaptive immune response.

Lymphocytes are the only cells in the body capable of specifically recognizing and distinguishing different antigenic determinants; they are responsible for the two defining characteristics of the adaptive immune response: specificity and memory. The functions of lymphocytes were described earlier in the section on the adaptive immune response.

Dendritic Cells

Dendritic cells play a vital role in asymptomatic apical periodontitis. They have been shown to be present in apical periodontitis lesions in rats.[120,202] Dendritic cells are accessory immune cells derived from bone marrow stem cells and may be related to the mononuclear phagocyte lineage.[1] They function as antigen-presenting cells for naive T lymphocytes and are important for the initiation of adaptive immune responses to protein antigen.[1] Activated dendritic cells produce IL-12, which is a key inducer of cell-mediated immunity.

Osteoclasts

Periapical bone destruction is a hallmark of asymptomatic apical periodontitis. During the chronic stage of apical periodontitis, both osteoclast and osteoblast activity decrease,[297] so the periapical osteolytic lesion remains stationary. Based on magnified radiographic and automated image analysis, periapical bone destruction was observed at 7 days after the pulps of experimental teeth were exposed to oral microorganisms in animal studies. A period of rapid bone destruction took place between 10 and 20 days, with slower bone resorption thereafter.[297] The stationary phase of bone resorbing activity was correlated to asymptomatic apical periodontitis.[297] Increased expression of bone resorptive cytokines such as IL-1, IL-6, and TNF was related to the period of active bone resorption.[298] T_H cells appear to outnumber T_S cells during the active stage of periapical bone destruction in induced rat periapical lesions, but T_S cells dominate T_H cells during the stationary stage of bone destruction.[256]

Bone resorption is caused by osteoclasts. The formation of osteoclasts involves differentiation of the osteoclast precursor from the monocyte-macrophage cell lineage in bone marrow. Several cytokines and growth factors, such as granulocyte/macrophage colony-stimulating factor (GM-CSF), RANKL (receptor activator of nuclear factor κB ligand), osteoprotegerin (OPG), IL-1, IL-6, TNF, as well as prostaglandins, bradykinin, kallidin, and thrombin, have been shown to mediate osteoclast progenitor cell differentiation.[24,40,65,147,180,208,272] Parathyroid hormone is capable of stimulating osteoblasts to synthesize GM-CSF and RANKL. Bone stromal cells and T cells also produce RANKL. Osteoclast progenitor cells express receptor activator of nuclear factor κB (RANK). OPG, a decoy receptor for RANKL secreted by osteoblasts, negatively regulates the differentiation of osteoclasts by absorbing RANKL and reducing its ability to activate the RANK pathway.[126]

RANKL activates the RANK pathway on osteoclast progenitor cells, resulting in differentiation of these cells along the osteoclast lineage. Proinflammatory cytokines, IL-1, TNF, and IL-6 also mediate the differentiation of osteoclast progenitor cells to osteoclasts. The differentiation of the mononuclear osteoclast progenitor cells terminates with fusion to latent multinucleated osteoclasts, which are finally activated to become bone-resorbing osteoclasts. The mature osteoclasts then attach to mineralized bone surface after osteoblasts have primed the unmineralized bone surface and released chemotactic factor to attract osteoclasts.[190] Osteoclasts attach to bone by the vitronectin receptor (integrin superfamily), expressed preferentially in the sealing zone. Vitronectin has binding sites for the arginine-glycine-aspartic amino acid (RDG) sequences present in many extracellular matrix proteins, including osteopontin, bone sialoprotein, and fibronectin, on the surface of the exposed mineralized bone.[81] When bound to bone extracellular matrix, osteoclasts develop a ruffled border. Inside the ruffled border, osteoclasts use ATP to drive H^+ pumps, leading to the acidification of the extracellular compartment. They subsequently secrete proteolytic lysosomal enzymes and carbonic anhydrase to degrade both the mineralized and unmineralized components of bone.[16,18,28,272] (See Fig. 15-5 for the mechanism of bone resorption by osteoclasts in apical periodontitis.)

The resorption of root cementum or dentin in apical periodontitis lesions is less well understood than resorption of bone. Bone is constantly remolding (resorption and deposition) through physiologic and functional processes and thus is much easier to study. In contrast, cementum and dentin are more stable. The cells responsible for dental hard-tissue resorption are called *odontoclasts*.[219] It has been shown in

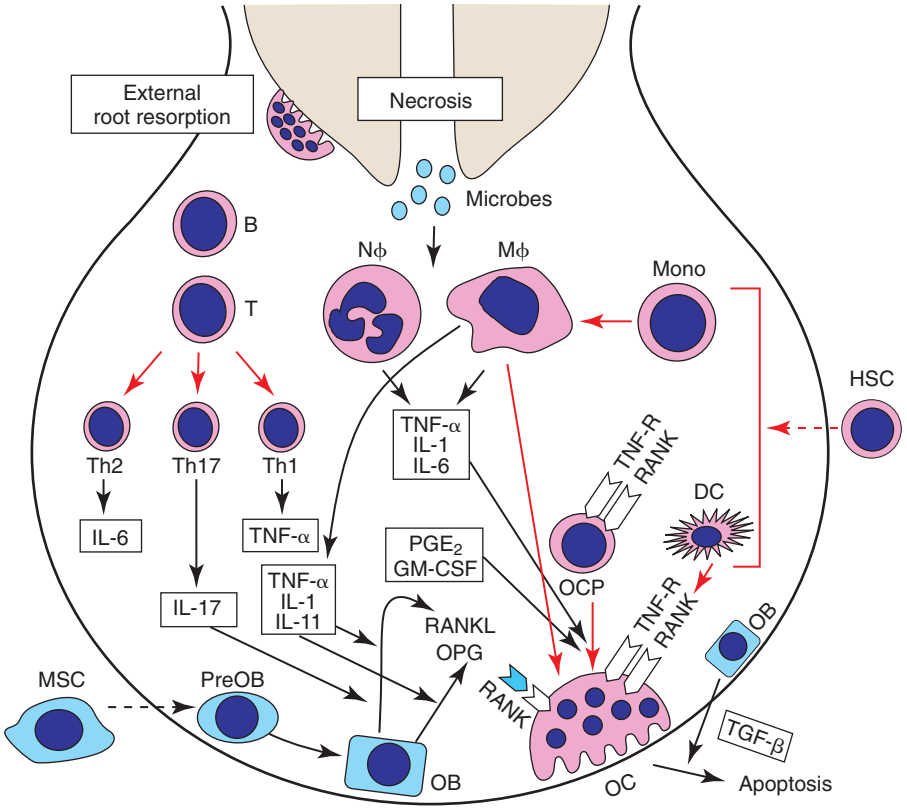

FIG. 15-4 Bone resorption by osteoclasts in apical periodontitis. *DC,* dendritic cell; *HSC,* hematopoietic stem cell; *Mφ,* macrophage; *MSC,* mesenchymal stromal cell; *Nφ,* neutrophil; *OB,* osteoblast; *OC,* osteoclast; *OCP,* osteoclast precursor; *OPG,* osteoprotegerin; *RANK,* receptor activator of nuclear factor κB; *RANKL,* receptor activator of nuclear factor κB ligand. (Courtesy Dr. Huang.)

ultrastructure and gene expression studies that odontoclasts and osteoclasts are similar.[220,221] Therefore, it is believed that the cellular mechanisms of bone, cementum, and dentin resorption are similar.[221] Nevertheless, little is known about how the precursors of the odontoclasts appear and what causes odontoclast differentiation and activation to resorb dentin and cementum. Bone resorption by osteoclasts in apical periodontitis is illustrated in Fig. 15-4.

Epithelial Cell Rests of Malassez

Proliferation of epithelial cell rests of Malassez (ERM) is evident in approximately 52% of inflammatory periapical lesions collected from extracted teeth.[186] It is a form of pathologic (inflammatory) hyperplasia. This type of hyperplasia is caused by stimulation of growth factors and cytokines produced during the inflammatory response. Hyperplasia is a self-limiting process and is reversible when the causative stimulus is eliminated.[137,165,248] During periapical inflammation, innate and adaptive immune cells and stromal cells (e.g., activated macrophages, neutrophilic leukocytes, NK cells, T cells, fibroblasts) in the periapical tissues produce many inflammatory mediators (e.g., prostaglandin, histamine), proinflammatory cytokines (e.g., IL-1, IL-6, TNF), and growth factors (e.g., PDGF, epidermal growth factor [EGF], keratinocyte growth factor [KGF], FGF). These inflammatory mediators, proinflammatory cytokines,[35,46,87,173,223] and growth factors[78,111,160,177,198,276] are capable of stimulating epithelial cell rests to proliferate (Fig. 15-5). The extent of cellular proliferation appears to be related to the degree of inflammatory cell infiltration.[214,215]

Fibroblasts

Fibroblasts are important cells in chronic inflammation and wound healing. They are derived from undifferentiated mesenchymal cells and exist in all connective tissues. They synthesize and secret proteoglycans, glycoproteins, and precursor molecules of various types of collagens and elastin.[126] In chronic inflammation, fibroblast migration and proliferation are triggered by multiple growth factors (TGF-β, PDGF, EGF, FGF) and the fibrogenic cytokines, IL-1 and TGF-α, produced by activated platelets, macrophages, endothelial cells, and inflammatory cells.[137,165] In turn, activated fibroblasts produce an array of cytokines such as IL-1, IL-6, and granulocyte/macrophage colony-stimulating factor, which influence leukocyte development. Fibroblasts stimulated by inflammation also produce matrix metalloproteinase to degrade the proteins that comprise the extracellular matrix.[165,287]

Fibrovascular granulation tissue is also a prominent feature of chronic apical periodontitis as a repair process. Migration and proliferation of endothelial cells from preexisting capillaries and venules into the site of chronic inflammation is called *neovascularization*. It is mediated by angiogenesis factors, such as vascular endothelial growth factor (VEGF) and TGF-β, produced by activated macrophages, platelets, and endothelium.[165,248] Neovascularization supplies oxygen and nutrients

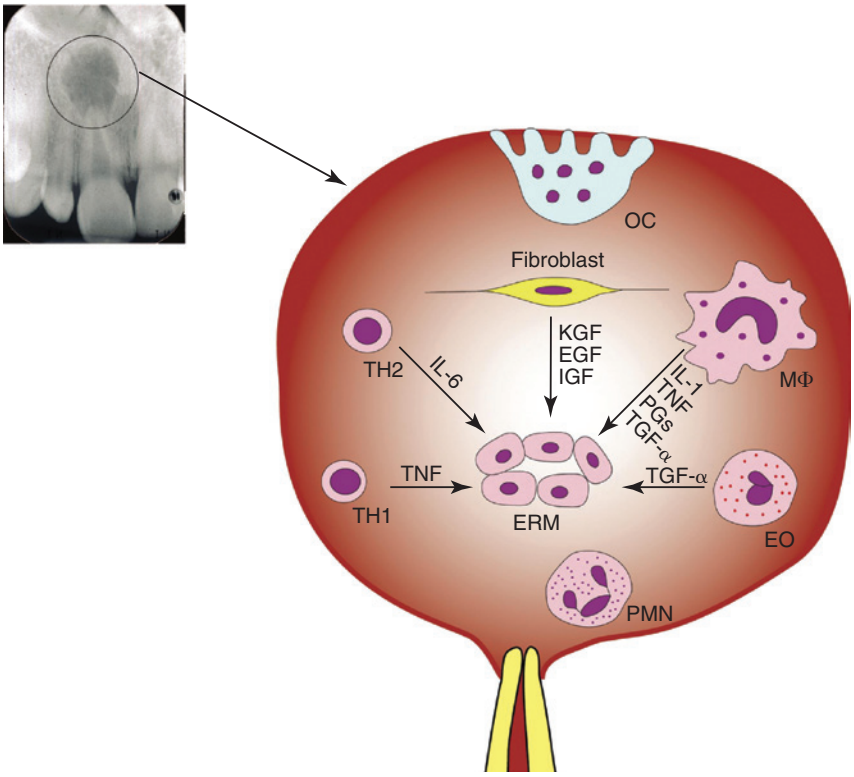

FIG. 15-5 Schematic illustration of the major mechanisms that activate proliferation of epithelial cell rests in apical periodontitis. (From Lin LM, Huang GT-J, Rosenberg P: Proliferation of epithelial cell rests, formation of apical cysts, and regression of apical cysts after periapical wound healing, *J Endod* 33:908, 2007.)

for the support of metabolically active macrophages and fibroblasts during wound healing.

Inflammatory Mediators

Many inflammatory mediators present in symptomatic apical periodontitis are also expressed in asymptomatic apical periodontitis. In addition, several different cytokines and growth factors are secreted by cells such as activated macrophages, lymphocytes, dendritic cells, and fibroblasts, in the chronic/adaptive inflammatory response described earlier.

Histopathology

Macrophages and lymphocytes are predominant cells in asymptomatic apical periodontitis lesions (Fig. 15-6). Occasionally, foamy macrophages and giant cells are seen, especially associated with cholesterol crystal deposits, which are products of disintegrated cell membranes. Cholesterol crystals are present in approximately 18% to 44% of all apical periodontitis lesions.[33,286] Bone resorption is a hallmark of asymptomatic apical periodontitis. Multinucleated osteoclasts may sometimes be seen in resorptive Howship lacunae. Proliferation of epithelial cell rests is often present in asymptomatic apical periodontitis lesions. ERM proliferate in three dimensions and form irregular strands or islands of epithelium, which are often infiltrated by varying degrees of inflammatory cells (see Fig. 15-6, *B*). A key feature of asymptomatic apical periodontitis is a proliferation of fibrovascular granulation tissue that is an attempt to prevent further spread of infection/inflammation and to repair wounded periapical tissues.

Little information is available concerning cemental changes in apical periodontitis. Using a scanning electron microscope, Simon and associates[242] observed that projections, depressions, and fibers of cementum were haphazardly arranged in root canal infection under scanning electron microscopy. There was an increase of mineralized projections, cementum lacunae, and surface resorptions and a decrease in fibers. All these changes may provide a more favorable condition for attachment of bacterial biofilm on the apical external root surface in extraradicular infection.

It is a general belief that asymptomatic apical periodontitis lesions are usually devoid of innervation, so local anesthesia may not be necessary if teeth with asymptomatic apical periodontitis require root canal therapy. However, light and transmission electron microscopic studies have demonstrated that teeth with chronic apical periodontitis were well innervated (Fig. 15-7).[153,168] This explains why instruments accidentally introduced into inflamed periapical tissues without local anesthesia can cause the patient pain.

External root resorption that involves cementum or both cementum and dentin usually occurs in asymptomatic apical periodontitis lesions.[143,292] Fortunately, cementum and dentin appear to be less readily resorbed than bone by inflammatory processes.[92] However, the radiographic presence of root "blunting" should be interpreted by the astute clinician as evidence for resorption of dentin and cementum, and the working length of the instrumentation and obturation phases of nonsurgical endodontic treatment should be adjusted by a corresponding amount.

Using light and transmission electron microscopy and microbiologic culturing, bacteria have been shown to be present in many asymptomatic apical periodontitis lesions.[2,114,284,300] Inadvertent contamination is a major concern

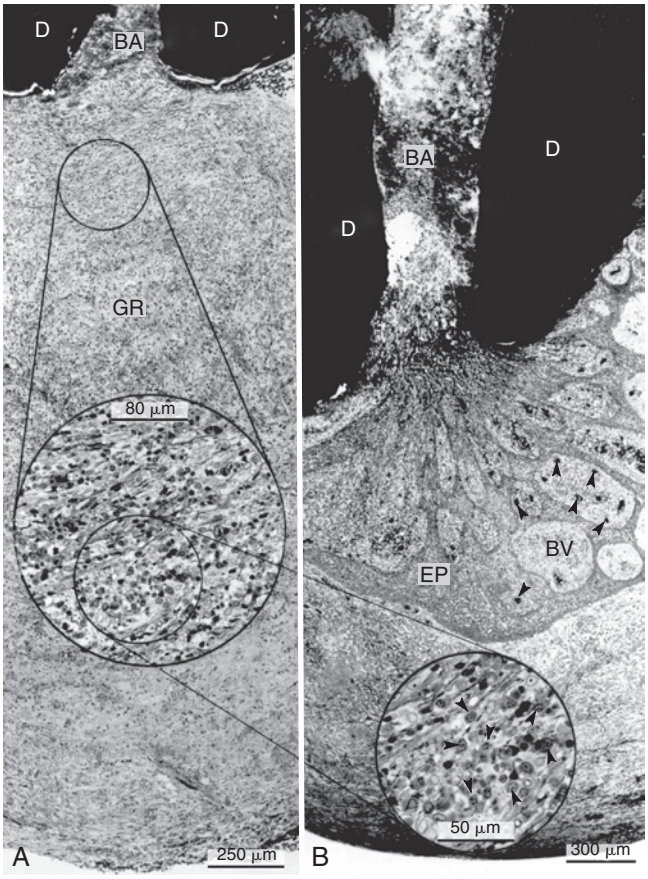

FIG. 15-6 Chronic asymptomatic apical periodontitis without **(A)** and with **(B)** epithelium (EP). The root canal contains bacteria (BA) in **A** and **B**. The lesion in **A** has no acute inflammatory cells, even at the mouth of the root canal with visible bacteria at the apical foramen (BA). Note the collagen-rich maturing granulation tissue (GR) infiltrated with plasma cells and lymphocytes (inset in **A** and **B**). *BV,* blood vessels; *D,* dentin. (**A,** magnification: ×80; **B,** magnification: ×60; inset in **A,** magnification: ×250; inset in **B,** magnification: ×400.) (From Nair PNR: Apical periodontitis: a dynamic encounter between root canal infection and host response, *Periodontol 2000* 13:121, 1997.)

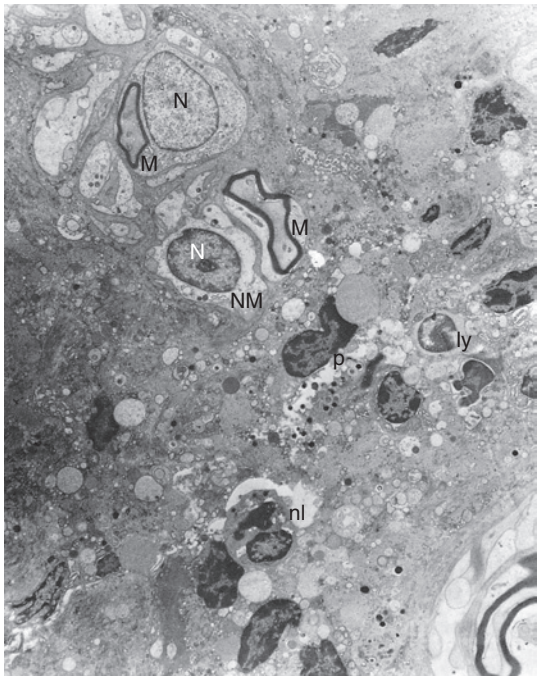

FIG. 15-7 Presence of intact myelinated and nonmyelinated nerve fibers in chronic apical periodontitis lesion. *ly,* Lymphocyte; *M,* myelinated nerve fiber; *N,* nucleus of Schwann cell; *nl,* neutrophilic leukocyte; *NM,* nonmyelinated nerve fiber; *p,* plasma cell (EM, magnification: ×1600). (From Lin L, Langeland K: Innervation of inflammatory periapical lesions, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 51:535, 1981.)

when culturing bacteria from these lesions. Under meticulous transmission electron microscopic examination, Nair[183] was unable to observe bacteria in most asymptomatic apical periodontitis lesions. If bacteria are present in the inflamed periapical tissues, they are usually found inside the phagocytes (Fig. 15-8).[154] Importantly, the mere presence of bacteria in the inflamed periapical tissues (colonization) does not necessarily imply periapical infection. Bacteria have to be able to establish an infectious process, such as survival and tissue destruction, in the inflamed periapical tissues to be considered etiologic factors for the resulting infection. Most apical periodontitis lesions are not infected; nonsurgical root canal therapy of teeth with apical periodontitis can achieve a high success rate, provided root canal infection is controlled.

Clinical Features

Involved teeth are usually asymptomatic and show an ill-defined or well-defined periapical radiolucent lesion. The chronic apical abscess and associated sinus tract are usually asymptomatic. Occasionally, the sinus tract from an apical abscess may drain along the root surface and opens into the gingival sulcus. This leads to the development of a deep, narrow pseudopocket that often mimics a periodontal pocket or a vertical root fracture (see Chapter 25 for details).

Outcomes

Asymptomatic apical periodontitis may result in (1) regeneration or repair of the periapical tissues after root canal therapy, (2) severe periapical tissue destruction, (3) acute exacerbation, (4) development of an abscess with an intraoral or extraoral draining sinus tract, or (5) development of a serious cellulitis.

ASYMPTOMATIC APICAL PERIODONTITIS WITH CYST FORMATION: RADICULAR CYST, CHRONIC APICAL PERIODONTITIS WITH CYST FORMATION

The radicular cyst is unique because no cysts in the body have similar pathogenesis. It is believed that the radicular cyst is likely formed by inflammatory proliferation of epithelial cell rests in the inflamed periodontal ligament.[158,189,200,274] The radicular cyst is a pathologic cavity completely lined by nonkeratinized stratified squamous epithelium of variable thickness in a three-dimensional structure in an apical periodontitis lesion. A radicular cyst can be a "pocket cyst" (i.e., attached to the apical foramen) or a "true cyst" (no attachment to the root structure), but it cannot form by itself. Therefore, a radicular cyst should not be considered a separate disease entity from asymptomatic apical periodontitis. A radicular cyst (pocket or true) is classified as an inflammatory and not a

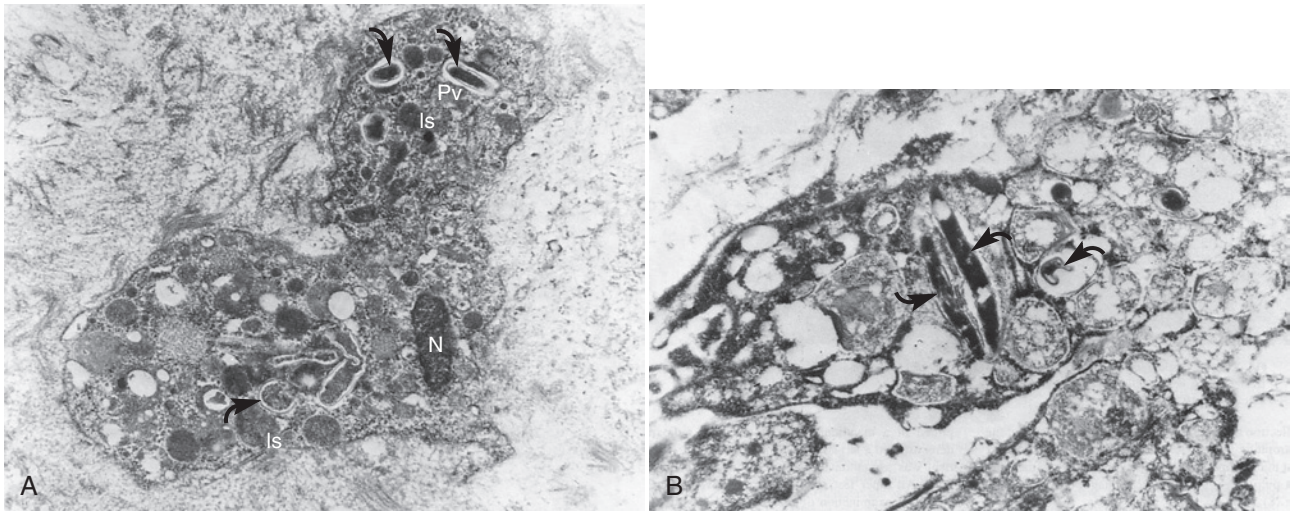

FIG. 15-8 Bacteria phagocytosed by phagocytes in chronic apical periodontitis lesion. **A,** Bacteria *(arrows)* in phagosome of a neutrophil. *ls,* Lysosome; *N,* part of nucleus; *Pv,* phagocytic vacuole (EM, magnification: ×6300). **B,** Part of the cell structure of a macrophage. Note longitudinally and cross-cut bacteria cells with unit membrane *(arrows)* in phagocytic vacuoles (EM, magnification: ×10,000). (From Lin L, Langeland K: Light and electron microscopic study of teeth with carious pulp exposures, *Oral Surg Oral Med Oral Pathol* 51:292, 1981.)

neoplastic lesion in the WHO Histological Typing of Odontogenic Tumors, Jaw Cysts and Allied Lesions.[136] The prevalence of radicular cysts in apical periodontitis lesions from extracted teeth ranges from 15% to 20%.[186]

Cell Biology

In addition to the presence of all chronic inflammatory cells, the epithelial cells are the most prominent cell type found in asymptomatic apical periodontitis with cyst formation. ERM can be considered unipotent or restricted-potential stem cells. They can be stimulated to divide symmetrically and asymmetrically into basal cells (stem cells) and suprabasal squamous cells of lining epithelium of radicular cysts.[158] Many theories about apical cyst formation have been proposed. The nutritional deficiency theory assumes that when islands of epithelium keep growing, the central cells of the epithelial island will move farther away from their source of nutritional supply and undergo necrosis and liquefaction degeneration. The products accumulated attract neutrophils and granulocytes into the necrotic area. Microcavities then coalesce to form a cyst cavity lined by stratified squamous epithelium.[274] The abscess theory postulates that when an abscess is formed in connective tissue, epithelial cells proliferate and line the abscess cavity because of their inherent tendency to cover exposed connective-tissue surface.[189,200] The theory of merging of epithelial strands proposes that proliferating epithelial strands merge in all directions to form a three-dimensional spherelike structure composed of fibrovascular connective tissue, with varying degrees of inflammatory cells trapped, that gradually degenerates due to lack of blood supply. This leads to formation of a cyst cavity.[158] Regardless of how a radicular cyst (pocket or true) is formed, it is likely caused by inflammatory proliferation (hyperplasia) of epithelial cell rests in these lesions.

It has been speculated that radicular cyst expansion is caused by increased osmotic pressure in the cyst cavity,[277] but this hypothesis overlooks the cellular aspects of cyst growth and the biochemistry of bone destruction.[72,173] Radicular cyst expansion is likely caused by degradation of fibrous connective tissue capsule by matrix metalloproteinases, which are produced by activated neutrophils, fibroblasts, and macrophages[275] and bone resorption. The ERM have also been shown to be capable of secreting a bone resorbing factor.[25] Most inflammatory mediators and proinflammatory cytokines that stimulate proliferation of epithelial cell rests also mediate bone resorption in apical periodontitis lesions.

Inflammatory Mediators

Mediators are basically similar to those present in chronic apical periodontitis.

Histopathology

Two types of cysts have been described in chronic apical periodontitis lesions. The pocket cyst's lumen opens into the root canal of the involved tooth (Fig. 15-9).[185,186,241] The true cyst is completely enclosed by a lining epithelium, and its lumen has no communication with the root canal of the involved tooth (Fig. 15-10).[185,186,241] Apical cysts are lined by hyperplastic, nonkeratinized stratified squamous epithelium of variable thickness, which is separated from the fibrovascular connective tissue capsule by a basement membrane. Both the lining epithelium and the connective tissue capsule are usually infiltrated with inflammatory cells,[235] indicating that inflammatory cells are attracted to these tissues by chemotactic irritants, either in the root canal system or in the periapical tissues. In nonproliferating epithelium, there is less inflammatory cell infiltration.[44,230] Occasionally, mucous cell metaplasia or ciliated respiratory-type cells are present in cystic epithelial lining.[195] The epithelial cells of lining epithelium do not show any characteristic features of neoplastic changes, such as pleomorphism, lack of polarity, nuclear enlargement, large nucleoli, abnormal nuclear/cytoplasmic ratio, hyperchromatism, or abnormal mitosis. Unlike odontogenic keratocysts and calcifying odontogenic cysts, the basal cells of radicular cystic lining epithelium are incapable of proliferation by themselves without stimulation of growth factors or cytokines released by innate

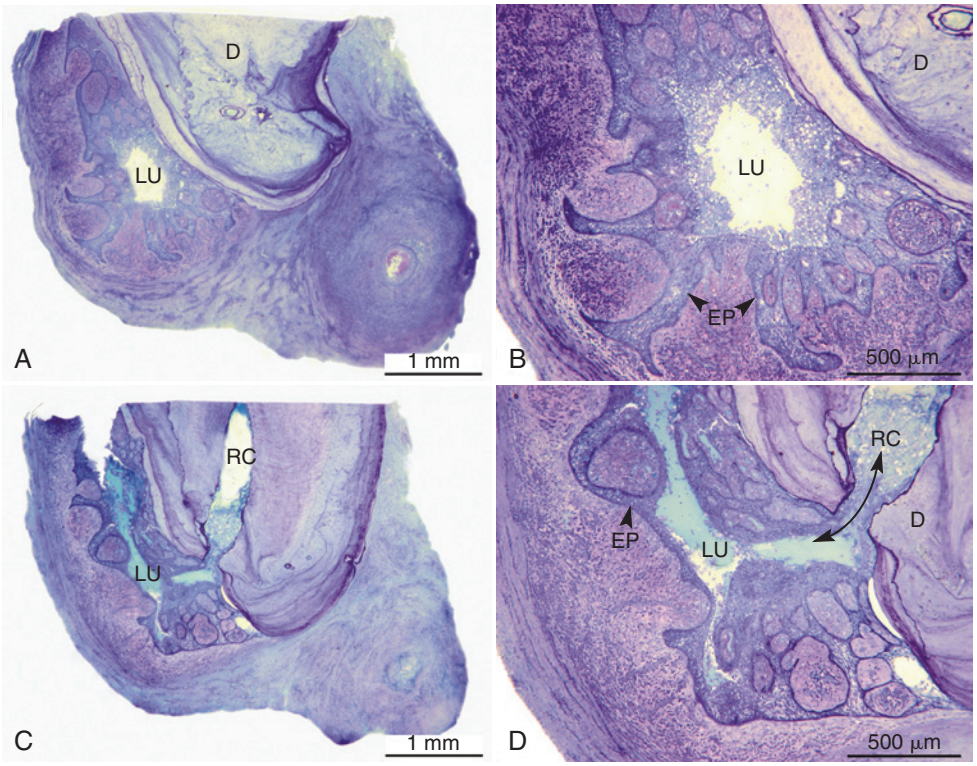

FIG. 15-9 **A,** A well-developed pocket cyst in apical periodontitis lesion. **B,** Note the saclike epithelial lumen. **C-D,** Sequential axial serial section passing through the apical foramen in the root canal plane (RC) shows the continuity of the lumen with the root canal. *D,* dentin (**A, C,** magnification: ×16; **B, D,** magnification: ×40). (From Nair PNR: Non-microbial etiology: foreign body reaction maintaining post-treatment apical periodontitis, *Endod Topics* 6:96, 2003.)

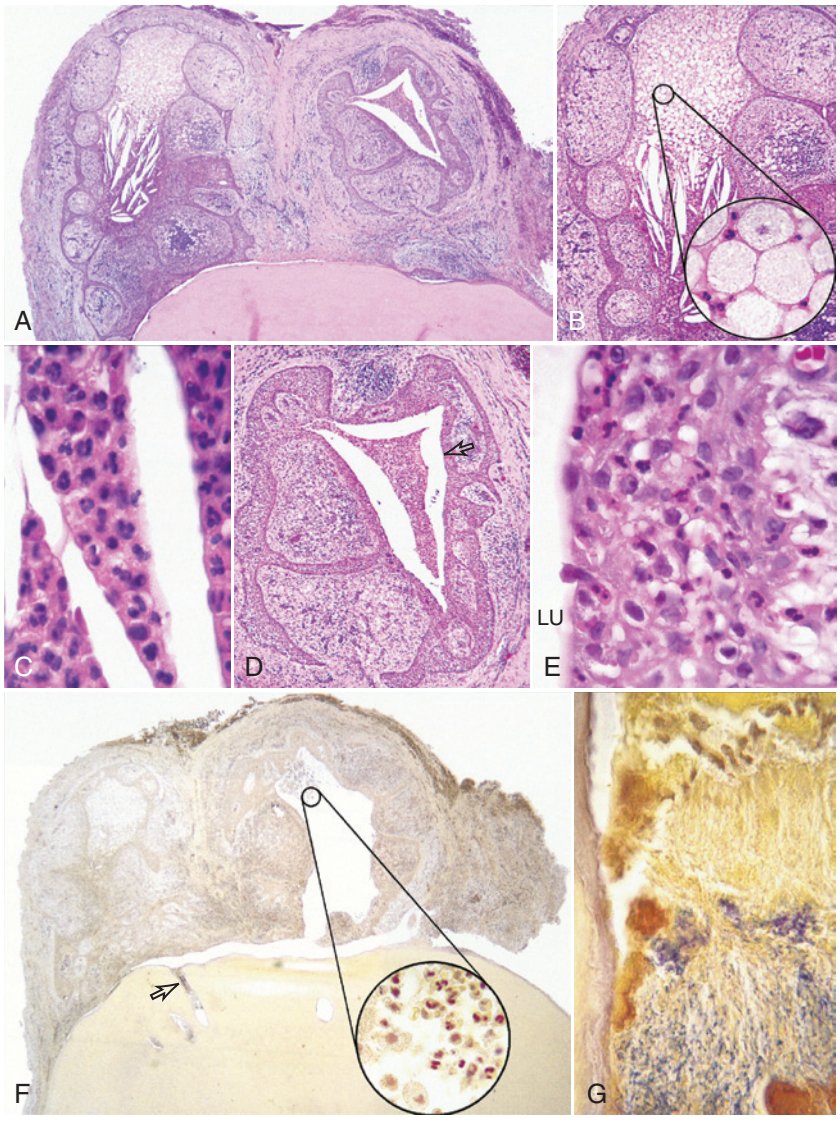

FIG. 15-10 **A,** Two distinct cysts lined by epithelium in apical periodontitis lesion. There is no evidence of communication with the foramen in serial sections (H&E, magnification: ×25). **B,** Cyst cavity on the left side in **A.** Accumulation of foamy macrophages (H&E, magnification: ×50; inset magnification: ×1000). **C,** Lower part of cavity in **B.** Dense infiltration of neutrophilic leukocytes (H&E, magnification: ×1000). **D,** Cyst cavity on the right side in **A.** Necrotic tissue debris in the lumen; infiltration of inflammatory cells in the epithelium (H&E, magnification: ×50). **E,** Cystic wall in **D.** Cyst's lining epithelium is infiltrated with acute and chronic inflammatory cells. *LU,* Lumen (H&E, magnification: ×1000). **F,** Foamy macrophages, neutrophilic leukocytes, and necrotic tissue inside the cyst *(inset),* but no bacteria (Brown & Brenn, magnification: ×25, inset magnification: ×1000). **G,** Area indicated by an *open arrow* in **F.** Bacterial colonies in the apical foramen (Brown & Brenn, magnification: ×1000). (From Ricucci D, Pascon EA, Pitt Ford TR, Langeland K: Epithelium and bacteria in periapical lesions, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 101:241, 2006.)

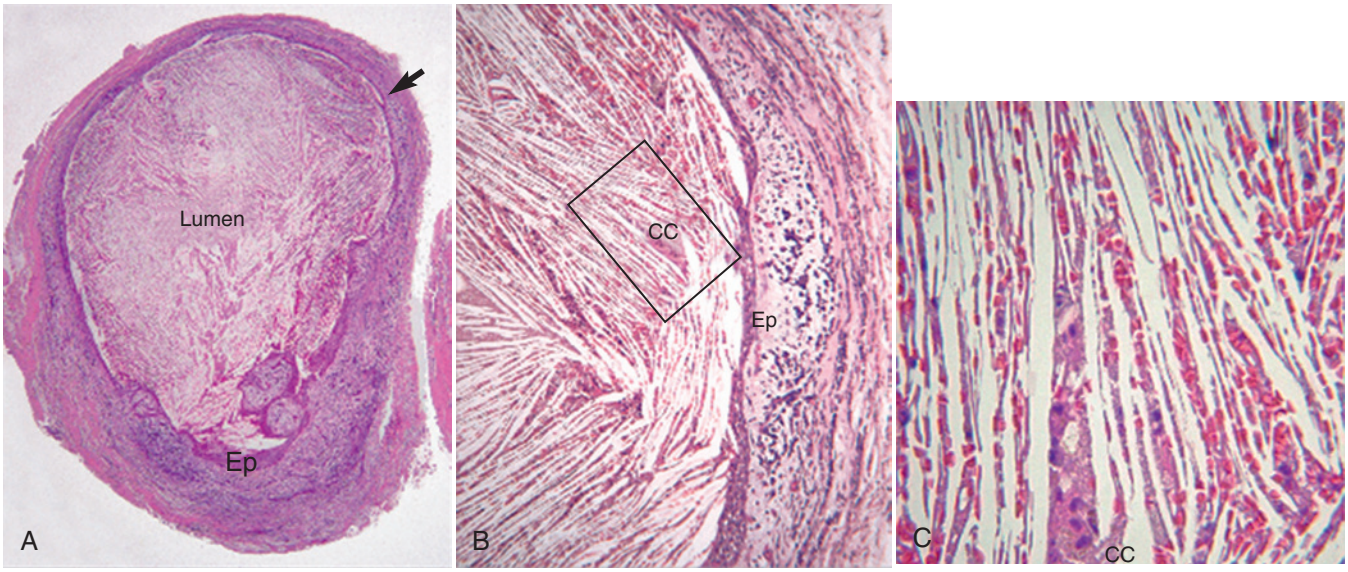

FIG. 15-11 **A,** A radicular cyst in which the lumen is completely filled with cholesterol crystals (H&E, magnification: ×25). **B,** High magnification of cholesterol crystals in **A** (H&E, magnification: ×100). **C,** High magnification of rectangular area in **B.** Multinucleated giant cells associated with cholesterol crystals (H&E, magnification: ×400). (Courtesy Dr. Ricucci, Rome, Italy.)

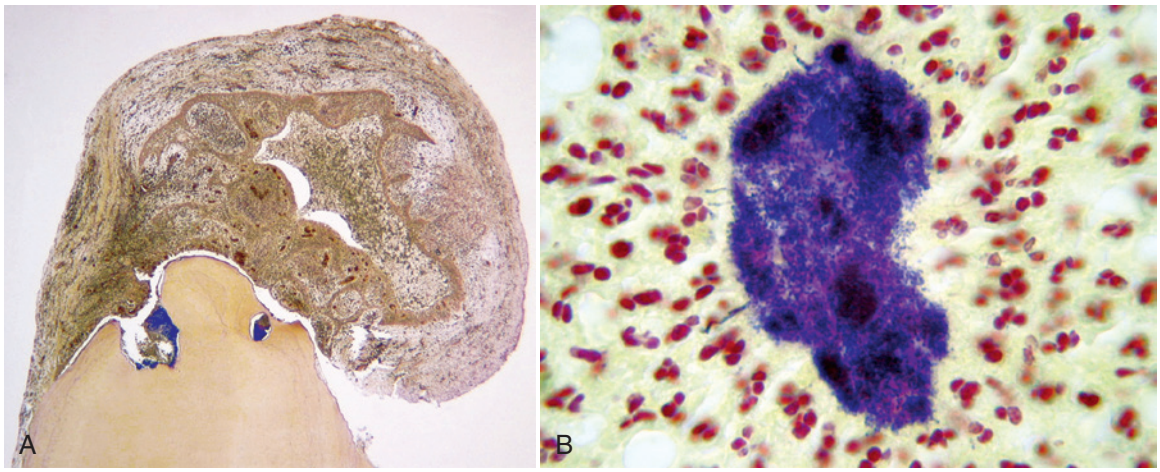

FIG. 15-12 **A,** An apical true cyst in apical periodontitis lesion. Bacterial colonies are seen in the foraminal areas (Brown & Brenn, magnification: ×25). **B,** Bacterial colonies in the cyst lumen, surrounded by inflammatory cells (Brown & Brenn, magnification: ×400). (From Ricucci D, Bergenholtz G: Histologic features of apical periodontitis in human biopsies, *Endod Topics* 8:68, 2004, Fig. 6.)

and adaptive immune cells during periapical inflammation. The lumen of cysts may contain inflammatory exudates, cholesterol crystals, clear fluid, or bacterial colonies (Figs. 15-11 and 15-12).[186,215]

Clinical Features

The involved tooth is usually asymptomatic. Periapical osteolytic lesions of endodontically involved teeth may sometimes show a well-demarcated radiolucency surrounded by a rim of radiopaque border.

Outcomes

There is no direct evidence demonstrating whether asymptomatic apical periodontitis with cyst formation (pocket and true) can or cannot regress after nonsurgical root canal therapy. Unfortunately, apical cysts cannot be diagnosed clinically and can only be diagnosed after surgical biopsy or extraction of teeth with apical periodontitis. Clinical outcome studies have shown that after proper nonsurgical root canal therapy, teeth with apical periodontitis heal in 78% of cases.[247] Therefore, it is speculated that some cysts, especially pocket cysts, may heal after nonsurgical root canal therapy.[185] Apical true cysts are less likely to heal after nonsurgical root canal therapy because of their self-sustaining nature; surgical intervention is necessary.[185] Nonetheless, similar to the periapical pocket cyst, a periapical true cyst is also formed within an apical periodontitis lesion and is not a neoplastic lesion. Any disease caused by inflammation/infection should be able to heal if the causative irritant/irritants are removed, unless the irritant/irritants are neoplasm-inducing agents or a carcinogen.

ASYMPTOMATIC APICAL PERIODONTITIS WITH REACTIVE BONE FORMATION: CONDENSING OSTEITIS OR CHRONIC FOCAL SCLEROSING OSTEOMYELITIS

Asymptomatic apical periodontitis with reactive bone formation is analogous to chronic osteomyelitis with proliferative periostitis. The etiology and pathogenesis of these two diseases are not well understood. It is generally believed that both lesions are caused by a long-term, low-grade inflammation/infection or a high resistance of local tissue to inflammation/infection.[195,210] Instead of bone resorption, the inflammation induces reactive bone formation of alveolar trabecular or spongy bone around the periapex of endodontically involved teeth.

Cell Biology

As described previously, during the chronic stage of apical periodontitis, both osteoclast and osteoblast activity decreases.[297] However, in asymptomatic apical periodontitis with reactive bone formation, osteoblasts appear to be stimulated to produce more bone. It is not clear what factor/factors stimulate osteoblasts to produce more bone mass. It could be due to increased expression of growth factors/cytokines, such as TGF-β, BMP, PDGF, and transcription factor Cbfa1 (core-binding factor family).[126]

Histopathology

There is an excessive apposition of bone mass without bone resorption in the apical area. As the bone marrow spaces become smaller and obliterated, the bone resembles compact bone that is infiltrated by a small number of lymphocytes. The compact bone has few lacunae, and many of them are empty of osteocytes. It has many prominent resting and reversal lines, similar to an idiopathic osteosclerosis or a Pagetoid appearance.[44,195,210]

Clinical Features

The lesion is usually observed in young patients, and the mandibular first molar is most commonly involved. The teeth often have gross carious lesions and can be vital or nonvital. They are usually asymptomatic. Radiographically, the lesion may have a well-defined or ill-defined radiopaque mass associated with the apex of an endodontically involved tooth. The lamina dura around the root apex is usually intact.

Outcomes

Most apical periodontitis lesions with reactive bone formation demonstrate healing after nonsurgical root canal therapy. The excessive compact bone in most cases will be remodeled to normal appearance.[69]

PERIAPICAL LESIONS OF NONENDODONTIC ORIGIN

Many periapical lesions are not of endodontic origin and should be considered in the differential diagnosis of apical periodontitis. These lesions include (but are not limited to) trauma,[10,11] foreign bodies,[133,187,313] host metabolic by-products,[188] advanced periodontal disease,[140] fibro-osseous lesions, and benign and malignant tumors.[195] A complete description of these lesions is beyond the scope of this chapter; the interested reader is encouraged to seek the cited references.

EXTRARADICULAR ENDODONTIC INFECTION

Extraradicular endodontic infection implies that bacteria have established an infectious process outside the root canal system in the periapical area.[243,260,284] Extraradicular endodontic infection can only be diagnosed after surgical biopsy or microbiologic sampling or molecular detection of therapy-resistant periapical lesions during apical surgery. The method of sample collection should be carefully reviewed, because contamination is a problem in bacteriologic study of apical periodontitis lesions during apical surgery.

Extraradicular endodontic infection can be a part of intraradicular infection or an independent entity without an intraradicular component. To be considered an independent extraradicular infection, the presence of pathogens in the root canal system must be completely ruled out, because intraradicular bacteria pose more potential problems than extraradicular bacteria in periapical inflammation. Bacteria in the complex root canal system are well protected from root canal procedures (i.e., mechanical instrumentation, antiseptic irrigation, intracanal medication), host defenses, and antimicrobial therapy because of a lack of blood circulation and areas resistant to access (e.g., isthmi; see Chapter 6). In our opinion, the independent extraradicular infection probably does not occur often and is usually associated with apical actinomycosis.[95,266] Bacteria have been observed as biofilm on the apical external root surface.[243,283] In addition, viruses have also been isolated from apical periodontitis lesions.[149,218,249] If microbiologic root canal cultures are not completely reliable (because bacteria may remain in the complex root canal system),[222] then interpretation of independent extraradicular infections as endodontic treatment failures should be carefully evaluated.

Although virulence and number of pathogens, as well as the host's defenses, determine whether extraradicular infection will develop or not, the development of extraradicular infection is still not fully understood. What are the predisposing factors to extraradicular infection? How are bacteria able to evade the host's powerful defenses in order to colonize and establish an infectious process in the periapical tissues? Are all extraradicular infections resistant to nonsurgical root canal therapy? For instance, are bacteria present in apical periodontitis with abscess formation?[32,90,263,291] Importantly, most periapical abscesses heal after proper nonsurgical root canal therapy. Similarly, most apical periodontitis lesions with draining sinus tracts also heal after proper nonsurgical root canal therapy, because the primary source of infection in the root canal system is removed. The preponderance of evidence does not support the consistent presence of an independent periapical infection in endodontic infections.

APICAL PERIODONTITIS AND SYSTEMIC DISEASES

Information concerning apical periodontitis and systemic diseases is limited. Clinical and radiographic studies have shown that there is a greater prevalence of periapical lesions in diabetics than in nondiabetics.[21] Type 2 diabetes mellitus is significantly associated with an increased prevalence of apical

periodontitis.[229] Other findings demonstrated that type 2 diabetes is associated with increased risk of a poor response by the periradicular tissues to odontogenic pathogens.[31] In rodent models, diabetic conditions enhance the development of periradicular lesions and increase mortality.[73,113,131] Mice deficient in P/E-selectin are shown to be more prone to infection-mediated early-onset periodontal disease.[196]

Genetic polymorphisms may also play a role in the individual's response to various diseases. Haplotype analyses have indicated that a variant of the IL-1β gene was likely to be most important for early-onset periodontitis risk.[62] In a Chinese male population, it was reported that the polymorphisms of *IL1A*+4845 and *IL1B*-511 might play an important role in determining generalized aggressive periodontitis susceptibility. A possible combined effect of the polymorphism of *IL1B*-511 and smoking on this type of periodontitis susceptibility was suggested.[150] In a study on patients from Chile, the heterozygous inheritance of the *IL1B*+3954 gene was found significantly higher in periodontitis cases than in control patients. The prevalence of a positive genotype (at least one allele of the two variants present at each locus) is significantly higher in cases (26%) than in controls (10%) and significantly associated with periodontitis, irrespective of smoking status and periodontitis severity.[162] Those displaying high-producer *IL6* genotype, intermediate and high-producer *IL1B* genotypes, and low-producer *TNFA* genotype tend to exhibit symptomatic dental abscesses.[57]

Apical periodontitis was once considered a focus of infection, and the microorganisms from the focus could disseminate through blood circulation to remote parts of the body, where secondary disease occurred.[108] Even though bacteremias do occur during root canal treatment of teeth with apical periodontitis when instrumentation is carried through the apical foramen into the periapical tissues, the incidence and magnitude of bacteremias is not clinically significant for a healthy person and in fact appears to be lower than that observed by flossing.[19,58,59] Microorganisms in the blood circulation are rapidly eliminated by the host's humoral and cellular defense components within minutes.[19,59] However, bacteremia may pose a potential danger to immunocompromised patients or patients with congenital heart valve disease.[58] No correlation between apical periodontitis and coronal heart disease and rheumatoid arthritis has been observed.[41,181] There is insufficient evidence to support that apical periodontitis could serve as a focus of infection and cause significant systemic diseases.[68,204]

GENETIC AND SYSTEMIC DISEASE RISK FACTORS OF PERSISTENT APICAL PERIODONTITIS

Genetic Risk Factors

Genetic polymorphism in the population plays an important role in the variation of the susceptibility and development of diseases among individuals. Knowledge on the polymorphism of key genes that are involved in disease development will help us to understand and establish prevention or treatment strategies for the diseases. Because of the completion of the Human Genome Project, many advanced molecular technologies have been developed including high-throughput screening and sequencing which further help gather the information on polymorphism in human populations. There are different types and causes of genetic polymorphisms. Single nucleotide polymorphisms (SNPs) that have a single base variation resulting from insertion or deletion of a base are considered the most common type of genetic polymorphism.[42]

Occurrence of SNP within the coding region of a gene may produce an altered protein, which may lead to altered function (termed functional SNPs). Occurrence of SNP within the promoter region of a gene may alter gene regulation, which may cause reduction/inhibition or overexpression of gene expression. Functional SNPs alter amino acid sequence or interfere with the transcriptional factor binding, whereas nonfunctional SNPs do not affect the regulation of protein expression. Many SNPs have no effect on cell function, but some could predispose people to disease or influence their response to drugs. Besides SNPs, there are single amino-acid polymorphisms (SAAPs or SAPs) of proteins, which may result from RNA editing independent of SNPs.[308]

A genetic polymorphism is defined as alleles or variants that appear in at least 1% of the population. The 1% cutoff excludes mutations that may have occurred in a single family. In most polymorphisms, one allele dominates and appears with < 99% frequency (for example, 65%) in the population. In a biallelic situation, some authors term the dominant allele as normal (N-allele or allele 1) and it occurs in < 99%, whereas the rarer allele (R-allele or allele 2) is present in >1% in the population.[227,245] It has been long known that genetic polymorphisms are associated with increased severity of inflammatory diseases.[98] Both endodontic and periodontic diseases are mainly an infectious disease caused by microbial infection. They share common features in terms of inflammation and bone resorption. Studies have implicated association of periodontitis or apical periodontitis with several gene polymorphisms such as *IL-1, IL-6, IL-8, FcγR*, tumor necrosis factor receptor superfamily member 1B (*TNFRSF1B*), (selenoprotein S) *SEPS1*, and matrix metalloproteinases (*MMPs*).[7,45,129,134,174,175,309]

It has long been observed that the proinflammatory cytokine interleukin-1 (*IL-1*) genotype is a severity factor in adult periodontal disease.[134] The specific periodontitis-associated IL-1 genotype has a variant in the *IL-1B* gene that is associated with high levels of IL-1 production. Gene polymorphism studies suggest that the frequency of *IL-1β* genotypes (*IL-1β*+3953 restriction fragment length biallelic polymorphism) is increased in patients with advanced adult periodontitis compared to early and moderate combined, and to healthy controls.[84] A more recent study showed that IL-1β levels in gingival crevicular fluid is increased with severity of disease and correlates well with clinical signs of incipient disease.[254] This suggests that some individuals, when challenged by bacterial accumulations, may respond with a higher level of inflammatory activities, therefore leading to more severe periodontitis. Both FcγRIIa (CD32) and FcγRIIIb (CD16) influence polymorphonuclear leukocyte phagocytic function and therefore their polymorphisms may also influence inflammatory disease development. It was reported that the *FcγR IIIb-NA2* allotype represents a risk factor for recurrence of adult periodontitis,[129] and the *FcγRIIIa-158V* allele and possibly *FcγRIIIb-NA2* may be associated with severity of chronic periodontitis in a Japanese population[130]; whereas the *FcγRIIa-H/H131* genotype may be associated with chronic periodontitis risk (and disease severity) in Caucasian smokers.[309] *FcγRIIIa* and *FcγRIIIb* genotypes may impose an additional risk of periodontal bone loss in a German population.[174]

The polymorphisms of two other genes involved in inflammation, *SEPS1* and *TNFRSF1B*, have been found to be involved in determining host individual susceptibility to aggressive periodontitis, and there is the potential association between *IL-6* and *FcγR* polymorphisms and the aggressive periodontitis in a Caucasian-Italian population.[224]

The genetic polymorphisms have been involved in the development of apical periodontitis reports. Two genetic conditions—carriage of allele H131 of the *FcγRIIa* gene and a combination of this allele with allele NA2 of the *FcγRIIIb* gene—have been reported to be associated with posttreatment apical periodontitis,[245] whereas polymorphism in the *FcγRIIIa* does not influence the patient's response to endodontic treatment of teeth with apical periodontitis in a Brazilian population.[246] So far there has been only one report on *IL-1* polymorphism association with apical periodontitis in an American population.[179] In this study, patients with persistent apical periodontitis showed an increased prevalence in the genotype composed of allele 2 of *IL-1β* polymorphism when compared with those who experienced complete healing after acceptable root canal therapy (70.6% versus 24.6%). This outcome suggests that specific genetic markers associated with IL-1β production may predict increased susceptibility to persistent apical periodontitis. An earlier paper that studied a Brazilian population did not find an association between IL1 polymorphism and persistent apical periodontitis; however, allele 1 (for *IL-1A* and *IL-1B*) was always detected higher in diseased than in healthy/healing individuals, although not statistically significant.[245] This lack of statistical difference could be due to small sample size or different population pools.

The polymorphism of the IL-8 gene, a potent chemokine that attracts neutrophils to the site of infection, has also been shown to influence the development of different forms of apical periodontitis.[7] *IL8/CXCL8-251 T* allele, which is associated with higher production of IL8/CXCL8, is also associated with a higher risk of developing acute suppurative form of apical periodontitis. *IL8/CXCL8 -251 A* allele, which is associated with lower production of IL8/CXCL8, is associated with chronic nonsuppurative form of apical periodontitis. SNP of other inflammation-associated genes, matrix metalloproteinases (MMPs) 2 and MMP3, also show influence on periapical lesion formation.[175] Thus, markers in MMP3 and MMP2 genes could also help predict host susceptibility to developing periapical lesions and the healing response.

Systemic Disease Risk Factors

Many systemic diseases have been linked to periodontal disease[127,199] because they contribute to either a decreased host's resistance to infection or dysfunction in the connective tissue of periodontium, increasing patient susceptibility to immuno-inflammation-induced destruction.[14] However, the similar systemic diseases do not appear to link to apical periodontitis, possibly because periodontitis is an open lesion and apical periodontitis is a closed lesion.

Diabetes mellitus (DM), a systemic disease, has been well documented to be associated with a high prevalence of apical periodontitis.[21,74,113,163,167,228] DM is a chronic disorder of carbohydrate, fat and protein metabolism, and a defective or deficient insulin secretory response.[137] Hyperglycemia is characteristic of DM and has profound effects on cell metabolisms, especially endothelial cells such as increased polyol pathway flux, increased formation of advanced glycation end products, hyperglycemia-induced activation of protein kinase C, and increased hexosamine pathway flux.[34] The tissue most frequently affected by hyperglycemia is microvasculature, whereby endothelial cells are not able to transport intracellular glucose effectively.[34] Because of damage to vasculature (atherosclerosis), blood circulation, innate and adaptive immunoinflammatory mechanism, and phagocyte function are impaired,[34,137] thus leading to pulp necrosis and predisposition to pulpal infection and subsequent apical periodontitis.[21]

Sickle cell anemia is the prototype of hereditary globinopathies, characterized by the production of structurally abnormal hemoglobulin.[137] The red blood cells are sickle shaped. Accumulation of distorted red cells could cause vasoocclusion, which could lead to anoxia, infarcts, and necrosis of tissue.[100] The occurrence of asymptomatic pulp necrosis in clinically intact permanent teeth has been reported.[13,123,239] Pulp necrosis could predispose to pulpal infection and apical periodontitis because of a lack of defense mechanism.

Sjögren syndrome is a chronic, systemic autoimmune disorder.[195] It involves salivary glands resulting in xerostomia. The lack of salivary cleansing action predisposes the patient to dental caries. If the caries is not prevented or treated early, it would impose risk of pulpal infection and apical periodontitis.

Radiation therapy of head and neck malignant lesions is associated with a high occurrence of caries.[104] If untreated, the radiation caries could lead to pulpal infection and subsequent apical periodontitis. It has been shown that a radiation dose 66 to 72.2 Gy (gray) in the head and neck region was associated with a high prevalence of caries and apical periodontitis because of a change in microflora favorable for carious development.[103]

However, there is no clear-cut direct causation or mechanism between systemic disease risk factors and the incidence or persistence of apical periodontitis without prior caries and pulp infection.

WOUND HEALING OF APICAL PERIODONTITIS

Periapical Wound Healing After Nonsurgical Root Canal Therapy

Understanding wound healing is as important as knowing the pathogenesis of disease, because satisfactory wound healing is the ultimate goal of treatment. If we are able to understand the mechanisms of periapical wound healing, we can design treatment approaches that maximize favorable conditions for wound healing to occur, such as effective disinfection of the root canal system in nonsurgical root canal therapy, control of periapical inflammation by medication, or incorporation of growth factors in bone grafts in surgical endodontic therapy (see also Chapters 12 and 21).

Interestingly, healing begins as soon as inflammation starts. When irritants (microbial and nonmicrobial) in the canal systems or in the periapical tissues are eliminated by nonsurgical or surgical endodontic therapy, inflammatory mediators are no longer produced in the periapical tissues because of the reduction of inflammatory cells. Inflammatory mediators already present are inactivated by the body's control mechanisms to prevent an inflammatory reaction from going unchecked. This process precedes wound healing. Although a great deal of information is known about what turns on the inflammation, relatively little is known about what turns off the inflammatory system after elimination of irritants. Examples of

host antiinflammatory control mechanisms are (1) enzyme destruction of inflammatory activators; (2) natural inhibitors of inflammatory mediators (opioids, somatostatin, glucocorticoids); (3) relative balance between intracellular levels of cyclic AMP (adenosine monophosphate) and cyclic GMP (guanosine monophosphate); (4) the antiphlogistic role of histamine; (5) inhibitors of the complement system[287]; and (6) antiinflammatory cytokines, such as IL-4, IL-10, IL-13, and TGF-β.[93,193] In addition, the major cellular inducer of inflammation, neutrophilic leukocytes, undergo apoptosis,[89] and the major cellular player of wound healing, macrophages, secrete antiinflammatory molecules such as lipoxins, resolvins, and protectins.[137,165,193]

The process of wound healing is tightly regulated by cell-to-cell cross-talk, cell-to–extracellular matrix interactions, and cell surface receptors, as well as the temporal and spatial expression of a variety of cytokines, growth factors, and neuropeptides and apoptosis (Table 15-3).[29,86,89,257,302] All these cellular and humoral factors operate together in either an antagonistic or synergistic manner and are precisely orchestrated during wound healing. This results in a highly organized response permitting regeneration of the original tissue architecture. Wound healing appears to be a programmed event. Much information concerning the pathogenesis of apical periodontitis has been gained from animal studies.[75,119,281] Although studies of wound healing of teeth with apical periodontitis after nonsurgical root canal therapy and endodontic surgery are available in a few animal experiments and a human study,[12,145,146] no studies of wound healing of apical periodontitis lesions with cyst formation after nonsurgical root canal therapy exist in the literature.

TABLE 15-3
Important Growth Factors/Cytokines in Wound Healing

Cytokines	Major Source	Target Cells and Major Effects
EGF	Macrophages, platelets, fibroblasts	Epithelial cells, fibroblasts, endothelial cells
FGF	Macrophages, endothelial cells	Endothelial cells (angiogenesis), mesenchymal cells
TGF-α	Macrophages, platelets, keratinocytes	Angiogenesis, fibroblasts
TGF-β	Macrophages, platelets	Similar to EGF
PDGF	Macrophages, platelets, endothelial cells	Chemoattractant for macrophages, fibroblasts
VEGF	Macrophages, epithelial cells	Angiogenesis
IGF	Fibroblasts, epithelial cells	Granulation tissue formation, reepithelialization
CSF	Multiple cells	Macrophages, granulation tissue formation
SP, CGRP	Sensory nerve	Endothelial cells, fibroblasts, keratinocytes

CGRP, calcitonin gene–related peptide; *CSF,* colony-stimulating factor; *EGF,* epidermal growth factor; *FGF,* fibroblast growth factor; *IGF,* insulin-like growth factor; *PDGF,* platelet-derived growth factor; *TGF,* transforming growth factor; *VEGF,* vascular endothelial growth factor.

Modified from Majno G, Joris I: *Cell, tissues, and disease*, ed 2, Oxford, 2004, Oxford University Press; Slauson DO, Cooper BJ: *Mechanisms of disease*, ed 3, St Louis, 2002, Mosby; Werner S, Grose R: Regulation of wound healing by growth factors and cytokines, *Physiol Rev* 83:835, 2003.

Wound healing of apical periodontitis lesions after proper nonsurgical root canal therapy follows the general principle of wound healing of connective tissues elsewhere in the body, with the formation of fibrovascular granulation tissue, removal of necrotic tissue and dead bacteria by activated macrophages, and finally repair or regeneration of the wounded tissue. Healing of apical periodontitis lesions is largely accomplished by regeneration and to some degree by fibrosis. Local tissue resident cells involved in periapical wound healing are osteoblasts and bone marrow mesenchymal stem cells in alveolar bone and multipotent stem cells in the periodontal ligament.[233] During periapical wound healing, many unwanted hyperplastic cells (e.g., endothelial cells, fibroblasts, epithelial cells) are deleted by apoptosis,[60,86,89] and the extracellular matrix is remodeled by metalloproteinases. Pathologic processes such as extensive fibrosis do not occur often, and the damaged periapical tissues can be restored mostly to their original structure by the process of regeneration.

The temporal and spatial relationship between alveolar bone, cementum, and periodontal ligament during periapical wound healing after nonsurgical root canal therapy cannot be clearly delineated. Nevertheless, wound healing appears to recapitulate the embryonic morphogenesis of damaged tissues or organs in many instances. The process of periapical wound healing after nonsurgical root canal therapy may be similar to wound healing following guided tissue regeneration in periodontal therapy: regeneration of new cementum, new alveolar bone, and new periodontal ligament.[67,285] Both nonsurgical root canal therapy and guided tissue regeneration therapy in periodontal disease are intended to remove irritants and provide a favorable microenvironment conducive to regeneration of periodontal tissues damaged by apical periodontitis and marginal periodontitis, respectively.

During periapical wound healing, the cells of viable periodontal ligament from adjacent root surfaces proliferate to cover the root surfaces in which the periodontal ligament was damaged by apical periodontitis and removed by macrophages. Proteins derived from the Hertwig epithelial root sheath (i.e., enamel matrix proteins) are required for cementoblast differentiation from ectomesenchymal stem cells in the dental follicle during tooth development.[251] The cells of the Hertwig epithelial root sheath are absent in mature teeth.[192,230]

Nevertheless, the extracellular matrix and growth factors of cementum (i.e., IGF-1, FGFs, EGF, BMP, TGF-β, PDGF) sequestered after cemental resorption in mature teeth are capable of inducing proliferation, migration, attachment, and differentiation of multipotent stem cells in the periodontal ligament into cementoblast-like cells and produce cementoid tissue on the root surface denuded of periodontal ligament.[88,233] This is similar to reparative dentin formation by pulp stem cells in direct pulp capping procedures where growth factors such as TGF-β are released from dentin matrix binding sites.[191,217,289] Root resorption that involves cementum or both cementum and dentin can only be repaired by cementoid tissue, because multipotent stem cells of the periodontal ligament are incapable of differentiating into odontoblasts that produce dentin.[233]

Bone has a remarkable capacity for regeneration in response to injury. During periapical wound healing, the osteoprogenitor cells or mesenchymal cells lining the surfaces

of endosteum—stimulated by TGF-β, BMPs, IGFs, PDGF, VEGF, and cytokines released by stromal cells, osteoblasts, platelets, and bone matrix after bone resorption—can undergo proliferation and differentiation into osteoblasts and produce bone matrix.[6,161] When one of the cortical bone plates (buccal or lingual/palatal) is destroyed, osteoprogenitor cells in the inner layer of periosteum beneath the oral mucosa—stimulated by TGF-β, BMPs, IGFs, PDGF, and VEGF—are also capable of proliferation and differentiation into osteoblasts and can produce bone matrix.[126,161] If both buccal and lingual/palatal cortical bone plates are destroyed by large apical periodontitis lesions, it is possible that the lesion may be repaired with fibrous scar tissue because of extensive destruction of the periosteum beneath the oral mucosa.[12] Accordingly, a guided tissue regeneration procedure using membrane barriers and bone grafts is recommended to prevent ingrowth of fibroblasts from periosteum or submucosa into the bony defect and to enhance periapical wound healing if periapical surgery is necessary.[54,55] The cellular and molecular mechanisms of excess scar tissue formation in periapical wound healing are not completely understood. Growth factors/cytokines may play an important role in regulating fibroblast gene expression and excess scar tissue formation.[288]

The newly regenerated periodontal ligament will finally undergo remodeling into a mature periodontal ligament, with one group of collagen fibers (Sharpey fibers) inserted into the newly formed cementum and another group of collagen fibers inserted into the newly formed alveolar bone. Thereby, regeneration of damaged periapical tissues, cementum, periodontal ligament, and alveolar bone is completed.

Periapical Wound Healing After Surgical Endodontic Therapy

The mechanism of periapical wound healing after nonsurgical and surgical endodontic therapy is similar, but the kinetics of healing of the periapical wound after endodontic surgery is faster than nonsurgical endodontic therapy.[138] In surgical endodontic therapy, a clinician removes irritants, such as necrotic cells, tissue debris, and bacteria in the periapical lesions, which is called *surgical debridement*.[157,165] In contrast, in nonsurgical endodontic therapy, activated macrophages perform bacterial killing and cleanup of periapical lesions, which is called *biologic debridement*.[165] Surgical debridement is very effective and of course quite rapid, whereas biologic debridement takes time. However, endodontic surgery is more invasive. In addition, proper case selection is more important in endodontic surgery than in nonsurgical endodontic therapy. The distinctive difference between nonsurgical and surgical endodontic therapy is that the goal of nonsurgical endodontic therapy is to remove primary microbial etiology from the root canal system, and the goal of surgical endodontic therapy is often to seal microbial etiology within the root canal system by root-end filling in most cases (see also Chapter 21).

Can Radicular Cysts in Apical Periodontitis Lesions Regress After Nonsurgical Endodontic Therapy?

Based on histology and cell biology, no studies have ever shown that apical true cysts are different from apical pocket cysts. It was suggested that pocket cysts in apical periodontitis lesions might regress after nonsurgical root canal therapy by the mechanism of apoptosis or programmed cell death, based on molecular cell biology.[158] In contrast, apical true cysts may be less likely to heal after nonsurgical root canal therapy because of their self-sustaining nature.[185] Histologically, inflammatory cell infiltration is always present in the lining epithelium or fibrous connective tissue capsule of apical true cysts.[185,214,215] This indicates there is continued presentation of irritants (such as bacteria)—present in the root canal system, the periapical tissues, or the lumen of cysts—to attract inflammatory cells to the cystic lining epithelium or fibrous connective tissue capsule.[186,214,215] It is not known if epithelial cells of apical true cysts alone are capable of acting as autocrine cells and secreting growth factors to sustain their own survival. It is important to realize that the apical true cyst is completely different from the odontogenic keratocyst, which is self-sustaining because it is a neoplastic lesion. Biologically, it is unlikely that the hyperplastic epithelial cells of inflammatory apical true cysts would suddenly transform into cells that behave like self-sustaining neoplasms. Any disease caused by infection should be able to regress after removal of its causative irritant(s), unless the irritants themselves are neoplasm-inducing agents or carcinogens, such as some viruses and human malignant tumors.[165,294] From pathogenesis, histology, and molecular cell biology, apical true cysts are similar to pocket cysts. Accordingly, apical true cysts, similar to pocket cysts, may be able to regress after nonsurgical root canal therapy by the mechanism of apoptosis if root canal infection is effectively under control.[158] This prediction is consistent with the high level of healing observed after nonsurgical root canal treatment.

In apical periodontitis lesions with cyst formation, the cysts have to regress before the periapical tissues can be restored to their original architecture. It is not known what matrix serves as a scaffold for endothelial cells, fibroblasts, and osteoblasts to migrate into the lumen of regressing cysts after nonsurgical root canal therapy. Complete regression of radicular cysts after nonsurgical root canal therapy could be due to any of several possible scenarios. Regression of radicular cysts and regeneration of bone may occur concurrently; or during regression of radicular cysts, part of the cystic lining epithelium could disintegrate due to apoptosis of local epithelial cells. Together with degradation of the basal lamina by matrix metalloproteinases, this could allow a fibrous connective tissue capsule to grow into the lumen of radicular cysts. Eventually, the cystic lining epithelium will completely regress or become remnants of epithelial cell rests remaining in the periodontal ligament.

Taken together, our knowledge of cyst mechanisms and healing offers tantalizing clinical implications and clearly supports the conclusion that more studies are required to understand the complex mechanisms of regression of inflammatory apical cysts, both pocket and true.

FACTORS INFLUENCING PERIAPICAL WOUND HEALING AFTER ENDODONTIC THERAPY

Local and systemic factors may affect periapical wound healing. Infection will complicate and prevent wound healing, foreign bodies can impair wound healing,[187,313] and nutrition can also

affect wound healing.[305] Diabetes was reported to reduce the likelihood of healing of apical periodontitis lesions after nonsurgical root canal therapy.[74] Impaired nonspecific immune response and disorders of the vascular system appeared to have a significant influence on the success rate of nonsurgical root canal therapy on teeth with apical periodontitis.[166] However, immunocompromised patients, such as HIV patients, responded as well as counterpart patients after nonsurgical endodontic therapy.[49,82,207] Although smoking has not been shown to be associated with increased incidence of apical periodontitis and prognosis of nonsurgical root canal therapy,[23,63] smoking may increase complications of periapical surgery, such as pain and swelling.[80]

Patients receiving radiotherapy of jaws and bisphosphonate therapy have a risk of developing osteonecrosis of jawbones,[212,216,306] so nonsurgical root canal therapy is recommended for these patients.[121,152] However, for endodontic surgery, the guidelines of the American Association of Oral and Maxillofacial Surgeons position paper on bisphosphonate-related osteonecrosis of the jaws are highly recommended.[8] For asymptomatic patients who have taken an oral bisphosphonate for less than 3 years and have no clinical risk factors, endodontic surgery is not contraindicated.[8] Nevertheless, the patient's physician should be consulted. Any kind of surgical procedure should be avoided for patients receiving intravenous bisphosphonate.[8] To prevent the complication of osteonecrosis, if patients are going to receive radiation of jaws or bisphosphonate therapy, nonsurgical or surgical endodontic therapy should be completed before the initiation of radiation or bisphosphonate therapy.[8]

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CHAPTER 16

Root Resorption

SHANON PATEL | CONOR DURACK | DOMENICO RICUCCI

CHAPTER OUTLINE

General Histologic Features
External Inflammatory Resorption
Introduction
Etiology and Pathogenesis of External Inflammatory Resorption
Histologic Appearance
Clinical Features
Radiographic Features
Management
Follow-up and Prognosis of External Inflammatory Resorption
External Cervical Resorption
Introduction
Etiology and Pathogenesis
Histologic Appearance
Clinical Features
Radiographic Features
Management
Internal Root Resorption
Introduction
Etiology and Pathogenesis of Internal Root Resorption
Histologic Appearance
Clinical Features
Radiographic Features and Diagnosis
Management
Chemomechanical Debridement of the Root Canal
Obturation
Summary

Dental resorption is the loss of dental hard tissues as a result of clastic activities.[90] It may occur as a physiologic or pathologic phenomenon. Root resorption in the primary dentition is a normal physiologic process except when the resorption occurs prematurely.[22,23] The initiating factors involved in physiologic root resorption in the primary dentition are not completely understood, although the process appears to be regulated by cytokines and transcription factors that are similar to those involved in bone remodeling.[60,123] Unlike bone, which undergoes continuous physiologic remodeling throughout life, root resorption of permanent teeth does not occur naturally and is invariably inflammatory in nature. Thus, root resorption in the permanent dentition is a pathologic event; if left untreated, it may result in premature loss of the affected teeth.

Root resorption may be broadly classified into two types, external and internal, based on the location of the resorption in relation to the root surface.[8,105] Internal root resorption was reported as early as 1830.[19] Compared with external root resorption, internal root resorption is a relatively rare occurrence, and its etiology and pathogenesis have not been completely elucidated.[72,91] Nevertheless, internal root resorption (IRR) poses diagnostic concerns to the clinician because it is often confused with external cervical resorption (ECR). An incorrect diagnosis may result in inappropriate treatment in certain cases.[57,58,88,89,91]

GENERAL HISTOLOGIC FEATURES

Osteoclasts are motile, multinucleated giant cells that are responsible for bone resorption. They are formed by the fusion of mononuclear precursor cells of the monocyte-macrophage lineage derived from the spleen or bone marrow; osteoblasts and osteocytes, on the other hand, are derived from skeletal precursor cells.[77,101] Osteoclasts are recruited to the site of injury or irritation by the release of many proinflammatory cytokines. To perform their function, osteoclasts must attach themselves to the bone surface. On contact with mineralized extracellular matrices, the actin cytoskeleton of an actively resorbing osteoclast is reorganized to produce an organelle-free zone of sealing cytoplasm (clear zone) associated with the osteoclast's cell membrane; this enables the osteoclast to achieve intimate contact with the hard tissue surface.[92] The clear zone surrounds a series of fingerlike projections (podosomes) of cell membrane, known as the *ruffled border,* beneath which bone resorption occurs. The resorptive area within the clear zone, therefore, is isolated from the extracellular environment, creating an acidic microenvironment for the resorption of hard tissues.

Odontoclasts, which are the cells that resorb dental hard tissues (Fig. 16-1), are morphologically similar to osteoclasts.[49] Odontoclasts differ from osteoclasts in that they are smaller, have fewer nuclei, and have smaller sealing zones, possibly as a result of differences in their respective resorption substrata.[74] Osteoclasts and odontoclasts resorb their target tissues in a similar manner.[92] The two cells have similar enzymatic properties,[84] show similar cytologic features, and create resorption depressions, termed *Howship lacunae*, on the surface of the mineralized tissues (see Fig. 16-1).[92] Odontoclasts are polarized in relation to dental tissue and have a ruffled border, located inside a clear zone, that is in intimate contact with their

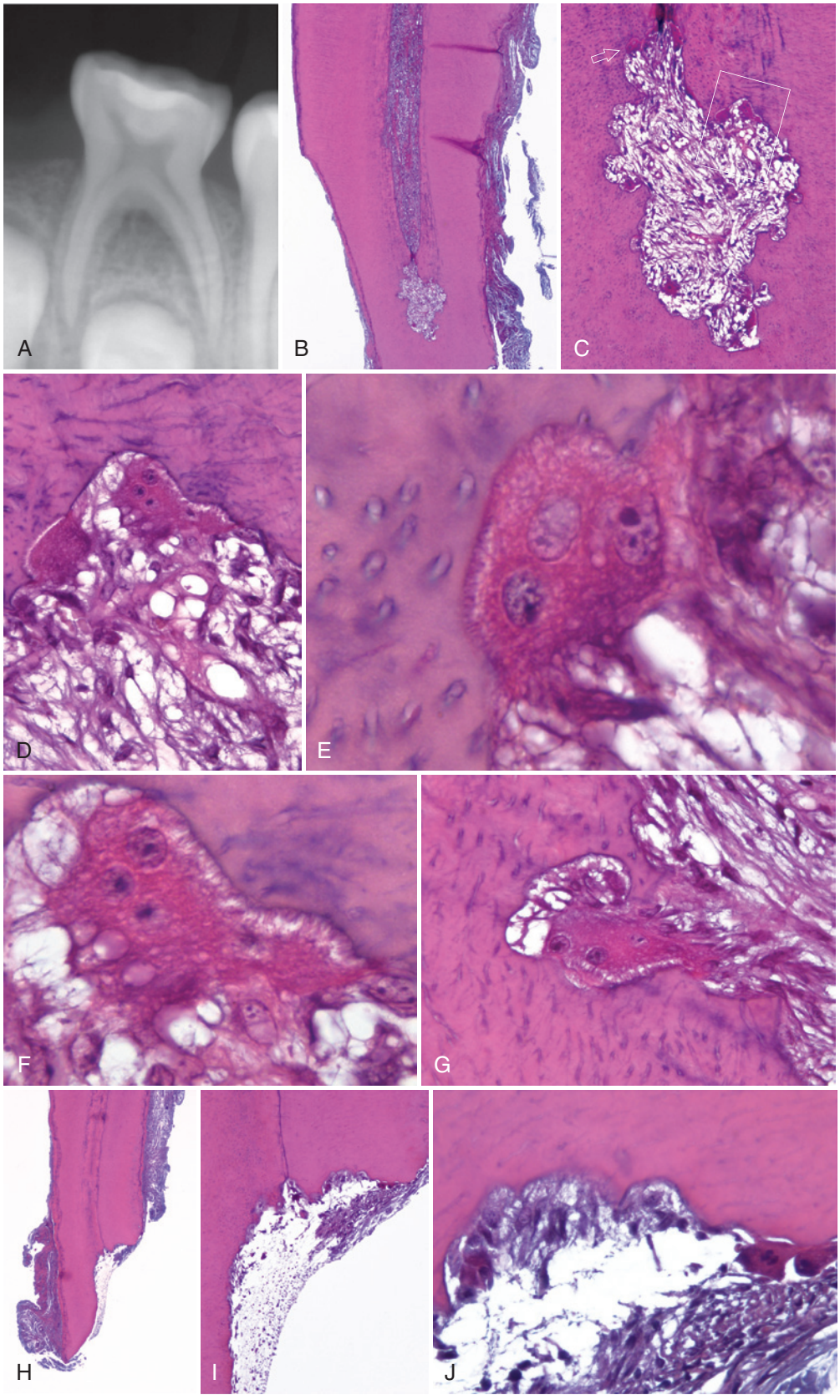

FIG. 16-1 Light microscopy images of a deciduous tooth showing both physiologic and pathologic (inflammatory) resorption. **A,** Tooth 7.5 in a 9-year-old boy. Extensive exposure of a mesial pulp horn occurred during caries excavation, and pulp capping was performed. The patient was brought to the dental office 4 months after the treatment because of severe spontaneous pain. The radiograph shows radiolucencies on both apices and interradicularly. The parents did not accept any treatment and requested extraction. **B,** Longitudinal section taken on a mesiodistal plane, passing approximately at the center of the mesial root. (Hematoxylin-eosin (H&E) stain; original magnification ×25.) **C,** Detail of the resorption area apically in **B** (original magnification ×100). **D,** Magnification of the area demarcated by the rectangle in **C.** Two odontoclasts are present in an area of dentin being resorbed, surrounded by fibroblasts and inflammatory cells (original magnification ×400). **E,** High-power view of the area indicated by the arrow in **C.** An odontoclast is in close contact with the dentin in a Howship lacuna. Its cytoplasm appears vacuolated and shows a more intense staining reaction than the cytoplasm of the adjacent cells (original magnification ×1000). **F,** High-power view of the upper odontoclast in **D.** The characteristic ruffled border can be distinguished (original magnification ×1000). **G,** Howship lacuna with an odontoclast in another area (original magnification ×400). **H** to **J,** Progressive magnification of the mesial root apex. The resorption process may be physiologic at this level (original magnification ×16, ×100, and ×400).

dental substratum.[67,92] Wesselink et al.[119] demonstrated that odontoclasts can simultaneously have two areas of ruffled border, which can resorb bone and hard dental tissue concurrently. Based on this evidence, Jones and Boyd[67] and Pierce[92] have stated that odontoclasts and osteoclasts are effectively the same cell type, differing only in their respective substrata. Wedenberg and Yumita[117] demonstrated that rat osteoclasts resorbed fully mineralized human dentin ex vivo, but did not attach to demineralized dentin or predentin. These researchers concluded that osteoclasts and odontoclasts are probably the same cell.

At high-power magnification, the cytoplasm of the odontoclasts appears vacuolated and shows a more intense staining reaction than the cytoplasm of the adjacent cells. Where odontoclasts are in contact with the tooth surface, the characteristic brush border can be seen, but the hematoxyphilic zone lining the lacunae is absent.[49] When odontoclasts are observed with an electron microscope, the most striking features are the large number of mitochondria and vacuoles in the cytoplasm and the scarcity of endoplasmic reticulum. An abundance of cytoplasmic ribosomes can be seen. The nucleoli are fairly large and centrally located in the nucleus. Close to the nucleus, the Golgi apparatus appears as a narrow zone of fine canals. Where the odontoclasts are in contact with the tooth surface, the ruffled border (corresponding to the brush border) may be observed with the light microscope. The ruffled border consists of cytoplasmic folds that create a system of canals extending 2 to 3 μm into the cytoplasm. In undecalcified sections, mineral crystals could be observed in these canals.[49]

Although mononuclear dendritic cells share a common hematopoietic lineage with the multinucleated osteoclasts, they have previously been regarded solely as immunologic defense cells. Recent studies have indicated that immature dendritic cells function also as osteoclast precursors and have the potential to transdifferentiate into osteoclasts.[102] Because dendritic cells are present in the dental pulp, it is possible they also may function as precursors of odontoclasts.

From a molecular signaling perspective, the OPG/RANKL/RANK transcription factor system[25] that controls clastic functions during bone remodeling has also been identified in root resorption.[109] The system is responsible for the differentiation of clastic cells from their precursors by means of complex cell-cell interactions with osteoblastic stromal cells. Similar to periodontal ligament cells that are responsible for external root resorption,[111] the human dental pulp has recently been shown to express osteoprotegerin (OPG) and receptor activator of NF-κb ligand (RANKL) messenger ribonucleic acids (mRNAs).[110] Osteoprotegerin, a member of the tumor necrosis factor superfamily, has the ability to inhibit clastic functions by acting as a decoy receptor that binds to RANKL and reduces its affinity for RANK receptors on the surface of clastic precursors. This results in inhibition of the regulation of clastic cell differentiation. Thus, it is possible that the OPG/RANKL/RANK system may be actively involved in the differentiation of odontoclasts during root resorption.

We know that osteoclasts do not adhere to nonmineralized collagen matrices.[106] It has been suggested that the presence of a noncollagenous, organic component in dentin (odontoblast layer and predentin) prevents (internal) resorption of the root canal wall, and precementum prevents (external) resorption of the external root surface.[114,116] Similar to osteoclasts, odontoclasts may bind to extracellular proteins containing the arginine-glycine–aspartic acid (RGD) sequence of amino acids by means of integrins.[98] The latter are specific surface adhesion glycoprotein membrane receptors containing different α and β subunits. In particular, $\alpha_v\beta_3$ integrin plays a key role in the adhesion of clastic cells.[81] Extracellular matrix proteins containing the RGD peptide sequence, particularly osteopontin, present on the surface of mineralized tissues and serve as binding sites of clastic cells.[65] The osteopontin molecule contains different domains, with one domain binding to apatites in the denuded dentin and another domain binding to integrin receptors in the plasma membranes of clastic cells. Thus, osteopontin serves as a linker molecule that optimizes the attachment of a clastic cell to mineralized tissues, mediating the rearrangement of its actin cytoskeleton.[33] It has been speculated that the lack of RGD peptides in predentin reduces the binding of odontoclasts, thereby conferring resistance of the canal walls to IRR.

Once the clastic cells have established contact with the cementum, any subsequent resorption is self-limiting unless the bound cells are subject to continued stimulation.[8] Therefore, in addition to the precipitating events discussed previously, progressive root resorption requires a source of stimulation for the resorbing cells. Stimulating factors vary and are related to the site and type of resorption, in addition to the cause of the predisposing damage to the predentin or precementum. Examples include persistent pressure and forces associated with continued orthodontic treatment; persistent impacted teeth; untreated cysts, granulomas, and tumors; endodontic inflammation and/or infection; and periodontal inflammation and/or infection.[50]

EXTERNAL INFLAMMATORY RESORPTION

Introduction

External inflammatory resorption (EIR) affects the surface of the root and is a relatively frequent sequel to dental luxation[4] and avulsion[14] injuries. It is a progressive condition with a potentially precipitous onset, and it is capable of advancing rapidly, such that an entire root surface may be resorbed within a few months if the tooth is left untreated.[10,14] It also affects teeth diagnosed with chronic periapical periodontitis (Figs. 16-2 through 16-5).[70]

The prevalence of EIR after luxation injuries ranges from almost 5%[4] to 18%.[35] It affects 30% of replanted avulsed teeth.[11,12] EIR is the most common form of external resorption root resorption after luxation and avulsion injuries.[35]

Clinical treatment of EIR is based on effective removal of the causal agent; namely, infected necrotic pulpal tissue in the root canal space.[41] Treatment should be carried out as soon as the resorptive process has been diagnosed. The earlier EIR is diagnosed and treated, the better the prognosis is for the affected tooth.[38]

The diagnosis of EIR in clinical situations is based solely on the radiographic demonstration of the process.[3] In some cases, the initial radiographic signs of EIR can be visualized as early as 2 weeks after replantation of avulsed teeth.[14] However, the limitations of conventional radiographic imaging in dentistry have been well reported. The diagnostic yield of radiographs is reduced by adjacent anatomic noise,[20,56,69,95] geometric distortion,[56] and compression of three-dimensional (3D) structures onto a two-dimensional (2D) shadowgraph.[34,87,113] These limitations may result in late diagnosis of EIR after dental trauma.

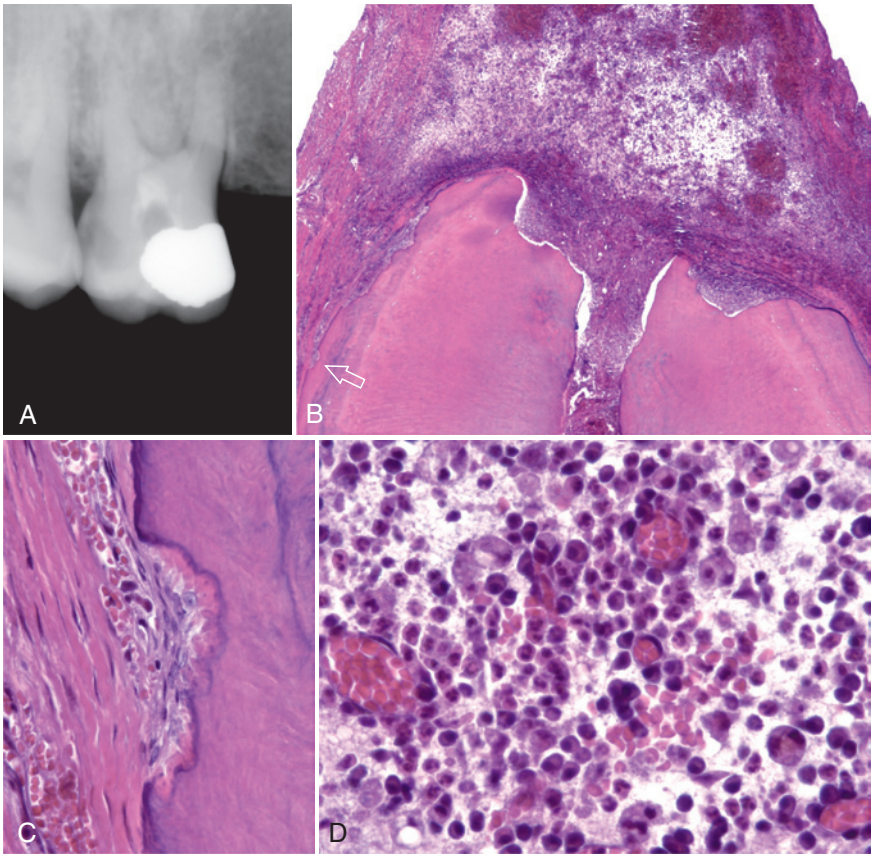

FIG. 16-2 External apical inflammatory resorption. **A,** Maxillary first molar in a 46-year-old female patient that was causing severe pain. The radiographs revealed material in the pulp chamber, a large distal caries, and apical radiolucencies. The tooth was extracted. **B,** Palatal root apex extracted with the pathologic tissue attached. Note the ingrowth of granulation tissue into the foramen and the massive resorption of the apical profile. Also note the resorption of lacunae on the left radicular profile. (H&E stain; original magnification ×25.) **C,** High magnification of the area of the external radicular profile indicated by the arrow in **B.** A resorption lacuna can be seen in the cementum. (H&E stain; original magnification ×400.) **D,** View from the center of the apical periodontitis lesion showing severe concentration of chronic inflammatory cells (mostly plasma cells) (original magnification ×400).

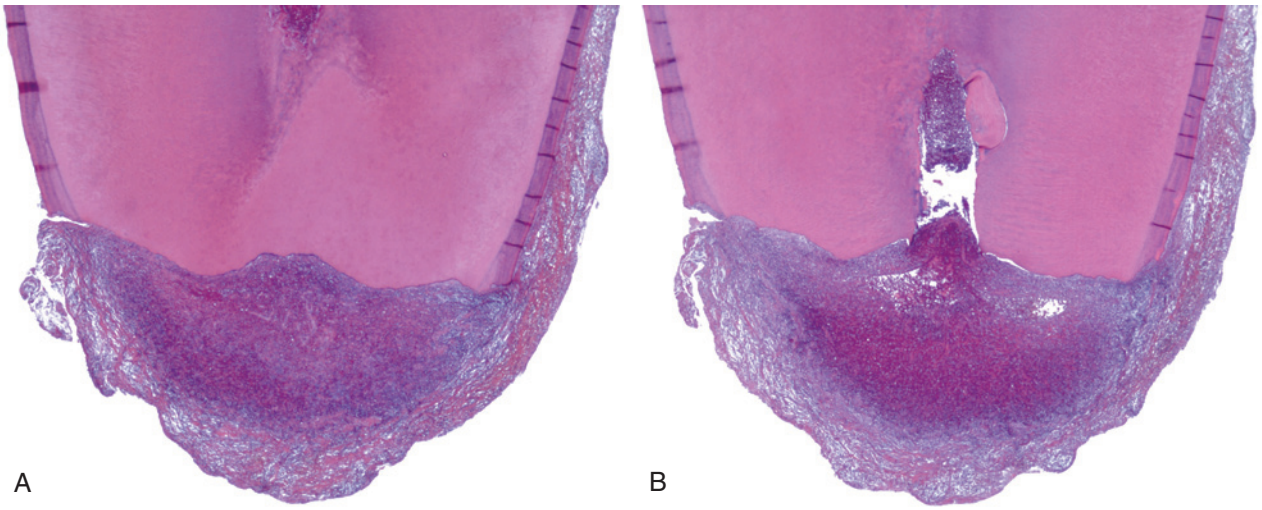

FIG. 16-3 External apical inflammatory resorption. **A,** Mandibular second premolar extracted with the periapical lesion attached. This histologic section, which did not pass through the canal, shows extensive apical resorption. (H&E stain; original magnification ×25.) **B,** Section taken approximately 120 sections away encompasses the apical foramen. In addition to resorption, the opposite phenomenon can be observed; that is, a large calcification partly embedded in the right apical dentin wall (original magnification ×25).

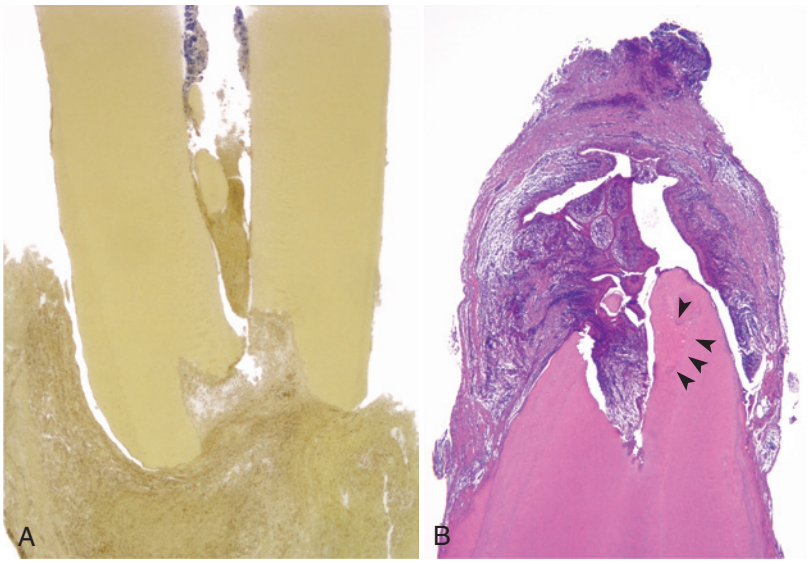

FIG. 16-4 External apical inflammatory resorption. **A,** Mesial root of a mandibular first molar extracted with a periapical lesion attached. This section passes through the canal and the foramen. Extensive resorption of the foramen has occurred, and a calcification can be seen more coronally. Note that the tissue in the apical canal and in the resorptive defect appears structured and continuous with the apical periodontitis lesion. A thick biofilm can be discerned layering the root canal walls more coronally. (Taylor's modified Brown & Brenn stain; original magnification ×25). **B,** Palatal root of a maxillary first molar extracted with the periapical lesion attached. A cyst cavity is present in the body of the lesion. Resorption of the foramen is present, and a portion of the right dentin wall has been replaced by bone *(arrowheads).* (H&E stain; original magnification ×25.)

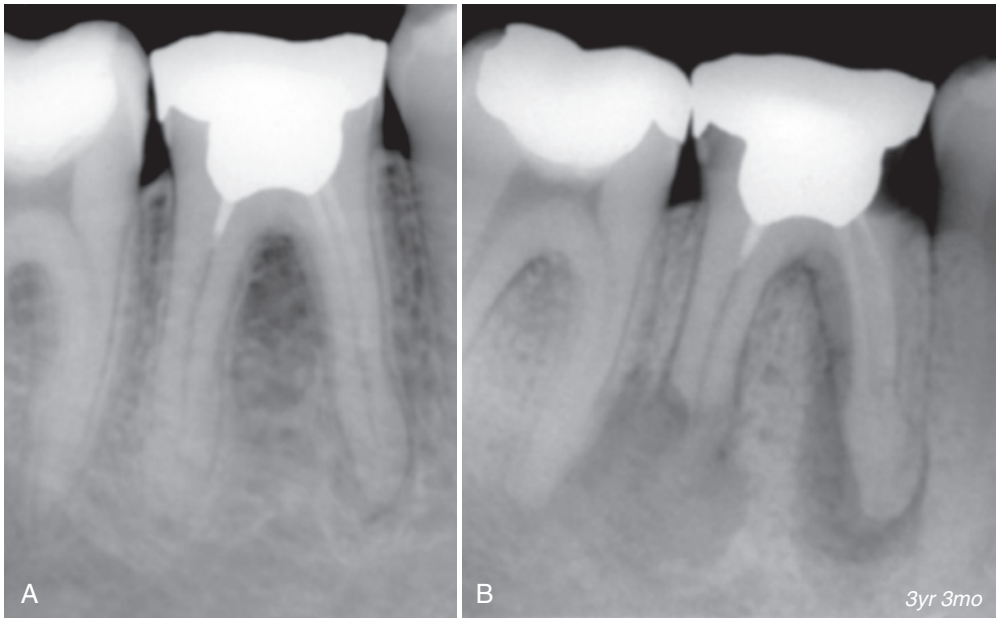

FIG. 16-5 Apical resorption. **A,** A 22-year-old male patient complained of pain on chewing. This was caused by a molar that had been treated endodontically 1 year earlier. A radiograph showed obturation material only at the root canal orifices and a periapical radiolucency on the mesial root. Endodontic retreatment was recommended, but the patient declined this treatment. **B,** The patient returned 3 years and 3 months later because of an abscess that was causing severe pain. A radiograph disclosed destructive mesial caries and large periapical radiolucencies on both roots, along with resorption of the whole apical third of the distal root.

When the occurrence of EIR is correlated with the specific types of luxation injury, it is clear that this type of resorption is often associated with the severity of the luxation injury. Andreasen and Vestergaard Pedersen[4] prospectively followed up 637 permanent luxated teeth in 400 patients for up to 10 years. In that cohort, there were no reported cases of EIR after concussion injuries and only one case after subluxation injuries, which represented only 0.5% of the total number of subluxated teeth. Six percent of extrusively luxated teeth and 3% of laterally luxated teeth developed EIR. However, EIR was a

healing complication in 38% of intrusive luxation injuries.[4] Crona-Larsson et al.[35] also reported a higher prevalence of EIR after more severe luxation injuries, with 60% of extrusively luxated teeth and 22% of intruded teeth affected by the complication. They reported EIR to be a much more frequent occurrence after lateral luxation (16.7%) and subluxation injuries (3.8%) relative to the findings of Andreasen and Vestergaard Pedersen.

Etiology and Pathogenesis of External Inflammatory Resorption

A specific set of conditions is required for the initiation and progression of EIR. Under normal conditions, permanent teeth are resistant to resorption.[115-117] Although the mechanisms that protect teeth against root resorption have not been fully elucidated, it is generally accepted that odontoclasts do not adhere to the nonmineralized layers covering the external root surface and the root canal wall (i.e., the precementum and predentin, respectively).[8,59,115-117] Similarly, it has been shown that, between episodes of physiologic resorption, the surface of bone is impervious to osteoclastic activity because it is covered by a layer of nonmineralized collagen fibrils, to which the osteoclast is incapable of binding.[32]

Traumatic dental injuries (e.g., intrusion, lateral luxation, and avulsion) and subsequent replantation often result in contusion injuries to the periodontal ligament (PDL).[8] Damage to the precementum, with a resultant breach in its integrity, is the precipitating factor in all types of external resorption.[8,9] In the subsequent wound healing process, necrotic PDL tissue remnants are excavated and removed by macrophages and osteoclasts.[44] A critical factor is that the precementum may be stripped from the root surface during the injury and the damaged cementum and bone may also be phagocytosed, resulting in exposure of the underlying dentin to osteoclastic and odontoclastic activity.[44] External root resorption may ensue, but the precise type depends on the severity of the initial injury, the stage of root development, and the pulp status of the affected tooth.[8,9] Once the clastic cells have established contact with the cementum or dentin, any subsequent resorption is self-limiting unless the cells are subject to continued stimulation.[7] As already mentioned, odontoclasts do not adhere to the nonmineralized layers covering the external root surface.[8,59,115-117] Therefore, damage to the precementum, with a resultant breach in the integrity of these layers, is the precipitating factor in all types of ERR.[8,9] Damage to the precementum and underlying tooth structure allow circulating odontoclasts to bind to the underlying mineralized dentin and cementum.[115]

The pathogenesis of EIR can be explained as follows. Contusion injuries to the PDL, after a traumatic dental injury (TDI) involving the periodontal structures, initiate wound healing, during which osteoclasts and macrophages are attracted to the site of the injury to remove the damaged tissue. The initial injury causes a breach in the integrity of the protective precementum. This permits odontoclasts to bind to and resorb the underlying mineralized cementum and dentin in a manner similar to the development of surface resorption.[9] However, EIR differs from surface resorption in that it is a progressive event that relies on microbial stimulation from the infected necrotic pulp of the affected tooth for its progression.[7] Therefore, it is more commonly associated with tooth avulsions[11,12] and moderate to severe luxation injuries, which have the potential to compromise pulp vitality.[4] If the strength of the initial osteoclast attack is sufficient to expose patent dentinal tubules beneath the cementum, a communication is created between the pulp space and the external root surface and adjacent periodontal tissues.[7,15] Microbes and/or their toxins (e.g., lipopolysaccharide, muramyl dipeptide, and lipoteichoic acid),[16] located in the root canal and the dentinal tubules, diffuse through the tubules and directly stimulate the resorbing osteoclasts. The resorption process intensifies and accelerates.[7] The resorbed mineralized tissue is replaced by granulation tissue, which ultimately invades the pulp space if the process continues.[16]

Pressure on the root surface during orthodontic treatment[71] and from impacted teeth,[122] cysts,[104] and tumors[73] may also denude the protective precementum from the root surface and therefore initiate ERR.

Histologic Appearance

The histologic appearance of EIR is characterized by saucer- or bowl-shaped areas of resorption in both the cementum and dentin, with concomitant inflammation in the adjacent periodontal membrane. Howship lacunae are a common feature of the resorption cavities, and histologic sections show that the lacunae sometimes are occupied by odontoclasts. The inflammatory reaction in the periodontal membrane appears intense and consists of a mixed-cell infiltrate that includes plasma cells, lymphocytes, and polymorphonuclear leukocytes in a granulation tissue matrix. Proliferation of capillaries in the areas of inflammation is also a feature.[15] EIR can be identified histologically 1 week after experimental replantation of teeth.[6]

Clinical Features

The tooth in question may look normal, but it will not respond positively to vitality testing. In advanced cases, signs of pulpal and/or periapical periodontitis may be present (e.g., discolored tooth, sinus present, and/or tenderness to percussion and/or palpation).

Radiographic Features

As mentioned, diagnosis of EIR is based solely on radiographic demonstration of the process.[3,14] EIR is characterized radiographically by radiolucent, concave, and sometimes ragged bowl-shaped excavations along the root surface, with corresponding and associated radiolucencies in the adjacent alveolar bone. Complete loss of the lamina dura is seen in the area of the resorption.[15] The initial radiographic signs of EIR can often be seen as early as 3 to 4 weeks after a TDI involving the periodontal tissues[15] and, if it will develop, EIR is always seen within 1 year after the injury.[5]

EIR can have a rapid onset and aggressive progression, such that complete resorption of an entire root can occur within 3 months. The diagnostic potential of a number of radiographic imaging systems has been investigated, with varying degrees of success.

Conventional intraoral radiographic imaging (digital or film based) is currently the clinical reference standard for the detection of ERR after luxation and avulsion injuries.[46,47] However, it has been well documented that this form of imaging is an inadequate method of detecting simulated ERR, especially when the cavity sizes are small.[3] Clinical studies have also

demonstrated that conventional radiography grossly underestimates the extent of inflammatory root resorption.[45]

Andreasen et al.[3] performed ex vivo experiments investigating the diagnostic accuracy of conventional radiographic imaging in the detection of simulated EIR and surface resorption. The simulated resorption cavities were located on the mesial, distal, or lingual surfaces and in the cervical, middle, or apical thirds. Nine preoperative radiographs and nine postoperative radiographs of each specimen were taken, for a total of 90 radiographs for the five specimens. The radiographs differed in the angulation between the specimen and the x-ray beam and also in the exposure time.

More than 50% of the medium and large cavities were identified, but none of the small cavities could be visualized on the radiographs, regardless of the horizontal x-ray beam angle or film density. The trabecular arrangement (noise) of the alveolar bone concealed the small cavities. Simulated resorption cavities on the proximal surfaces of the teeth were significantly easier to identify than lesions on the lingual aspect of the root. However, the position of the cavities along the root length (coronal, middle, or apical third) had no bearing on the examiners' ability to identify them. Cavities were more readily identified on high-contrast films; the availability of preoperative radiographs and multiple angled views of the specimens increased the chances of identifying the cavities.[3]

Chapnick[31] used an experimental design similar to that of Andreasen et al.[3] to further examine the efficacy of conventional radiography in the detection of simulated ERR. Multiple angled radiographs and alterations in exposure parameters were also used in this study in an attempt to maximize the diagnostic yield of the radiographs. Although the examiners were able to identify some small simulated resorption cavities, these defects were significantly more difficult to detect than medium or large ones. In agreement with Andreasen et al.,[3] Chapnick concluded that conventional radiography is an inadequate method of detecting early ERR.

Goldberg et al.[54] concluded that "*radiology is not a very accurate procedure for achieving an early and precise diagnosis of resorption defects.*"

The studies discussed to this point all used film-based conventional, intraoral radiography, Borg et al[24] concluded that charged coupled device (CCD) and photostimulable phosphor plate (PSP) digital systems are as sensitive as film-based radiography in the detection of simulated ERR. In a similar investigation, Kamburoğlu et al.[68] found that, although the CCD system used in their study performed as well as their conventional film radiographs, the PSP system was significantly less accurate at identifying the artificial cavities. In agreement with Andreasen et al.[3] and Goldberg et al.,[54] this study further reported that lesions created on the proximal root surfaces were easier to detect than those on the buccal/lingual surfaces and that the best results were achieved when multiple angled views and preoperative radiographs were available to the examiners at the same time.[68]

Clinical studies directly comparing the ability of intraoral radiographs and cone beam computed tomography (CBCT) to detect and diagnose EIR are limited. One clinical study reported that CBCT is superior to conventional radiography in diagnosing and determining the extent of nonspecific inflammatory resorption on root surfaces.[45] D'Addazio et al.[40] compared the ability of CBCT and periapical radiography to detect simulated external resorption cavities of about 2 mm in diameter in a human ex vivo model. Although both imaging modalities were 100% sensitive in the detection of the lesions, only CBCT could accurately assess the position of the defects on the root surface and their relationship to the root canal, even though multiple angled periapical radiographs of the test teeth were available to the examiners.

Alqerban et al.[1] compared the ability of two CBCT systems and conventional panoramic radiography to detect simulated external surface resorption lesions of varying sizes associated with canine impaction. The authors reported that with small and medium field of view (FOV), CBCT systems were superior to conventional panoramic radiography in the detection of simulated external resorption cavities regardless of the cavity size. There was no statistical difference between the diagnostic ability of the CBCT systems. However, conventional panoramic radiography is rarely used in endodontic-specific investigations. Intraoral radiography is currently the imaging technique of choice for assessing traumatically injured permanent teeth, which may develop EIR.[46,47]

A more recent study by the King's College London (KCL) group concluded that CBCT was a reliable and valid method of detecting simulated EIR, and performed significantly better than intraoral periapical radiography. Durack et al.[43] found that changing the exposure parameters so as to halve the radiation dose did not have a negative effect on the diagnostic yield of the reconstructed images.

Management

Clinical treatment of EIR is based on effective removal of the causal agent, the infected necrotic pulpal tissue in the root canal space. This arrests the resorption process and creates an environment conducive to hard tissue repair of the damaged root surface.[36,37,41] Therefore, it is essential to initiate root canal treatment as soon as radiographic signs of EIR are identified.[38] An exception to this is replanted teeth with closed apices; in these cases, root canal treatment should be carried out 7 to 10 days after replantation, even if there are no radiographic signs of EIR.[47] The earlier the resorption is diagnosed and treated, the better the prognosis is for the affected tooth. Failure to diagnose and treat the condition may result in tooth loss.

Effective chemomechanical debridement of the root canal space is fundamental to the success of the root canal treatment and the inhibition and cessation of EIR.[37,41] In principle, the specific root canal protocol used is irrelevant, as long as the biologic objectives are met. Long-term dressing of the root canal with calcium hydroxide may be beneficial in the treatment of established EIR; however, this protocol should be used judiciously because of the associated risk of root fracture.[13]

In many cases the EIR is extensive, rendering the tooth unsalvageable and requiring extraction.

Follow-up and Prognosis of External Inflammatory Resorption

Healing of EIR is characterized radiographically by cessation of the resorption process, resolution of the radiolucency in the adjacent bone, and reestablishment of the PDL space.[14] As mentioned previously, in untreated cases, EIR can progress so rapidly that an entire root can be resorbed within 3 months.[14,37] The prognosis is especially poor for untreated immature teeth.[14]

EXTERNAL CERVICAL RESORPTION

Introduction

External cervical resorption (ECR) is a form of root resorption that originates on the external root surface but may invade root dentin in any direction and to varying degrees. ECR generally develops immediately apical to the epithelial attachment of the tooth. In healthy teeth with a normal periodontal attachment, this is in the tooth's cervical region, a feature that gave rise to the name. However, in teeth that have developed gingival recession and lost periodontal support and/or have developed a long junctional epithelium, the resorptive defect may arise at a more apical location.

ECR has also been referred to as *invasive cervical resorption,*[62] *supraosseous extracanal invasive resorption,*[48] *peripheral inflammatory root resorption,*[53] and *subepithelial external root resorption.*[107] The authors of this chapter prefer the term *external cervical resorption* because it describes the nature and location of the lesions.

Etiology and Pathogenesis

The exact etiology and pathogenesis of ECR have not been fully elucidated. It is accepted that the resorptive process is the same for ECR as it is for any other type of resorption: a breach in the protective non-mineralized layers must exist to allow the clastic cells to bind to the underlying dentin, and the same cells must be stimulated to perpetuate the process. However, in ECR only some of the factors that predispose the root surface to clastic activity have been identified.

The anatomic profile of the cementoenamel junction (CEJ) is variable, and the junction between the enamel and the cementum in this region is not contiguous in all teeth. This may lead to exposed areas of unprotected dentin, which are vulnerable to osteoclastic activity, in the cervical region of some teeth.[83]

Heithersay[61] investigated the potential predisposing factors in 257 cases of ECR in 222 patients. Orthodontic treatment, dental trauma, oral surgery, periodontal therapy, bruxism, intracoronal restorations, delayed eruption, enamel stripping, and dental developmental defects were all identified as potential predisposing factors, either alone or in combination. Orthodontic treatment was the most common, sole predisposing factor identified, with a history of treatment in 21% of the patients and 24% of the teeth assessed. Dental trauma was the only identifiable predisposing factor for ECR in 14% of the examined teeth. Oral surgical procedures (particularly those in which the cervical region of the affected tooth was involved) were identified as the sole etiologic factor in 6% of the cases. Specific surgical procedures included extraction of partially and fully erupted third molar teeth adjacent to the affected tooth, exposure of unerupted canines or supernumerary teeth, transplantation of canine teeth, and surgical amputation of periodontally compromised teeth. Intracoronal bleaching was highlighted as the only evident predisposing factor in 5% of patients affected. A combination of predisposing factors was identified in a number of cases. For example, 4.3% of the affected teeth had a combined history of orthodontic treatment and another potential predisposing factor, primarily dental trauma and/or intracoronal bleaching. In addition, of the patients assessed, 7.7% had a history of bleaching and dental trauma; 1.8% had a history of dental trauma, bleaching, and orthodontic treatment; and 0.9% had a history of bleaching and orthodontic treatment only.

Although the data provided in this study are valuable and arguably the most comprehensive information available on the potential causes of ECR, no definitive cause-and-effect relationship has been established. In the cases affected by a combination of predisposing factors, it was impossible to determine definitively whether the development of ECR was the result of one specific event or a combination of factors, or if any of the potential causes identified were in fact contributory. In 15% of the patients examined in Heithersay's study, no potential predisposing factor was identified. Furthermore, intracoronal restorations were attributed as possible predisposing factors only when no other potential cause could be identified.[61]

There are conflicting views on the manner in which the resorptive process is sustained once the clastic cells have bound to the root dentin in ECR. One view is that microorganisms originating from the gingival sulcus provide the stimulus for continued resorption.[50,105] The opposing hypothesis, by Heithersay,[62] suggests that ECR is a type of "benign proliferative fibrovascular or fibro-osseous disorder" in which microorganisms play no active role and are either absent from the site of resorption or invade it only secondarily.

Histologic Appearance

The histologic profile of ECR is similar to that of other forms of resorption, with certain unique features reflecting the invasive nature of the process. In the early stages, granulation (fibrovascular) tissue occupies the resorptive cavity, and odontoclasts may be evident in lacunae on the resorbing front of the defect.[61] Acute inflammatory cells are often absent from the site of resorption in the early stages of ECR, but secondary bacterial colonization of the site of resorption may occur at a later stage.[39,62]

The resorptive cavity advances toward the subjacent root canal system and typically extends either circumferentially around, or in an apicocoronal direction through, the radicular dentin without communicating with the root canal. Narrow "channels" of resorption extend through the dentin and may communicate with the periodontal ligament.[61] However, perforation of the root canal wall usually occurs only at a late stage because the predentin affords protection against the resorbing cells.[61,64] Consequently, the pulpal tissue adjacent to the site of resorption has a normal histologic appearance until the root canal has been invaded.[61]

As the lesion progresses, bonelike tissue is deposited in the resorptive cavity in direct contact with the adjacent dentin; this is an attempt to repair the previous tissue destruction.[61]

Clinical Features

The clinical features of ECR are variable (Figs. 16-6 and 16-7). The process is very often quiescent and asymptomatic, especially in the earlier stages, and absence of clinical signs and symptoms is very common; the diagnosis is commonly made as a result of a chance radiographic finding. A pink or red discoloration may develop at the cervical region of the tooth; when present, this often is the feature that alerts the patient or clinician to the possible existence of a problem. The discoloration is due to the fibrovascular granulation tissue occupying the resorptive defect, which has a reduced thickness of enamel and dentin at its peripheries because of the loss of hard tissue. The granulation tissue imparts a pink hue to the tooth, through the

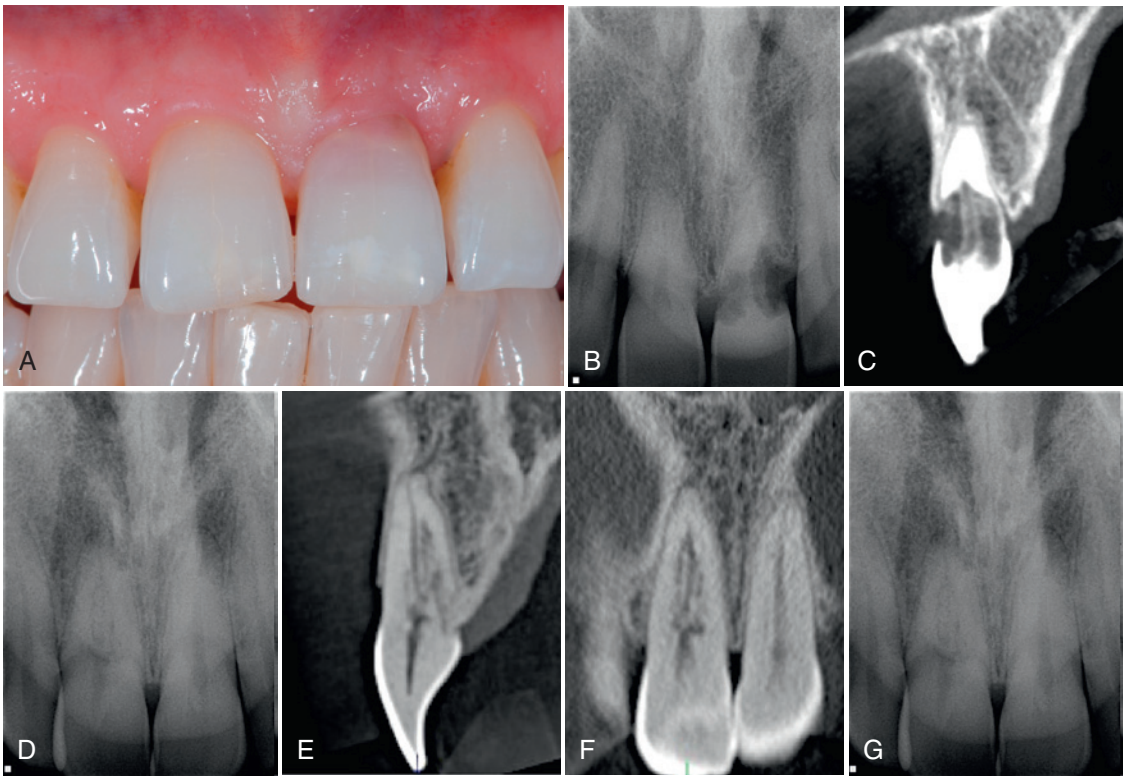

FIG. 16-6 External cervical resorption (ECR). **A,** A 55-year-old female patient presented with an asymptomatic "pink spot." She had no history of any predisposing factors. **B,** A periapical radiograph revealed radiolucent defects on the proximal aspects of the upper left central incisor; note the ragged borders. **C,** A reconstructed coronal cone beam computed tomography (CBCT) slice reveals the true extent of the ECR lesion. Note that the root canal wall appears to be intact. Inhibitory factors in the root canal wall/odontoblastic layer prevented the ECR lesion from penetrating the root canal. **D,** A 41-year-old male patient presented as a new patient. Routine radiographic examination revealed a poorly defined periapical radiolucency in the root of the upper right central incisor. The appearance is suggestive of ECR; the patient had orthodontic treatment in his early teens and remembered "knocking the tooth" at least twice when he was very young. Reconstructed sagittal **(E)** and coronal **(F)** CBCT slices revealed the true nature of the ECR lesion and showed that the lesion was not amenable to treatment. The treatment options were discussed with the patient, and it was decided to review the tooth periodically. **G,** The 4-year radiograph shows no change in size of this asymptomatic lesion.

thinned enamel and dentin, in the region of the resorption.[90] The granulation tissue may perforate the enamel or dentin at the gingival margin, giving the appearance of mild gingival hyperplasia. The discoloration, sometimes referred to as a "pink spot," can be quite subtle and is often a chance finding by the patient, the dentist or, increasingly, the dental hygienist. However, it is a relatively rare feature of ECR. Furthermore, it must occur at a site where it is readily identifiable (e.g., labial surface of an anterior tooth) to be noticed. Loss of periodontal attachment may occur in the region of the resorption, and probing of the resorptive defect or the associated periodontal pocket causes the granulation tissue to bleed profusely.[89]

As the process progresses, perforation of the root canal wall and bacterial contamination of the pulp may occur. The affected tooth may develop pulpitis and the associated clinical symptoms. Pulp necrosis and chronic periapical periodontitis may eventually develop. Clinical signs and symptoms may be the first indication of a problem with the affected tooth; they may include tooth discoloration, spontaneous localized pain, tenderness on mastication, tenderness to percussion, tenderness to palpation over the apical region of the tooth, a draining sinus, and/or buccal sulcus swelling.

Radiographic Features

The radiographic appearance of ECR depends on the location, the extent of invasion, and the relative proportions of fibro-osseous and fibrovascular tissue occupying the resorptive cavity. All ECR defects present as a radiolucency of varying radiodensity, often in but not confined to the cervical region of the affected tooth or teeth (see Figs. 16-6 and 16-7; also Figs. 16-8 and 16-9).

The lesion tends to be radiolucent when the defect is predominantly fibrovascular, granulomatous tissue. However, in cases with some fibro-osseous inclusions (i.e., more longstanding lesions), the radiolucency may adapt a more cloudy appearance. In advanced cases with extensive repair of the tissue destruction, significant deposition of fibro-osseous tissue gives the defect a mottled radiographic appearance (Fig. 16-10).

The margins of the lesion may vary from poorly to well defined, depending on the depth of the defect and the proportion and distribution of osseous inclusions in the lesion. Although lesions with irregular margins are more common, some ECR defects may have smooth and/or well-defined margins.

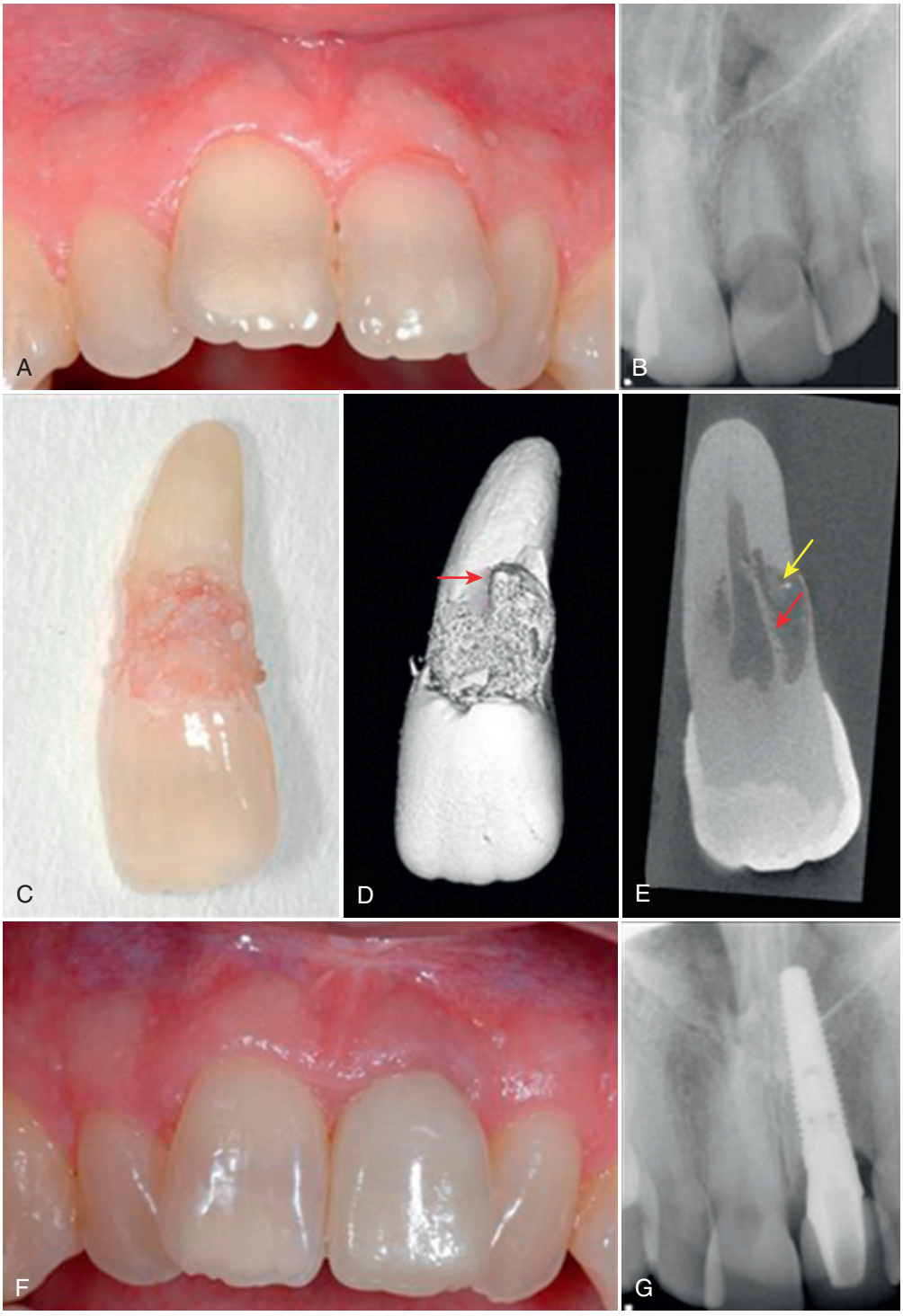

FIG. 16-7 **A**, This patient's upper left central incisor had a pink spot and was cavitated. **B**, A radiograph revealed an unusual presentation of ECR; the lesion is circular and has well-defined margins. Note that the outline of the root canal is visible and intact through the radiolucent lesion. **C**, The tooth was unrestorable and was extracted; note the large amount of granulation tissue. **D**, Three-dimensional reconstruction of the extracted tooth from microtomography data revealed bonelike tissue below the overlying granulation tissue. Note the intact root canal wall *(red arrow)*. **E**, Coronal reconstruction from a microtomography scan reveals how the predentin *(red arrow)* prevented the ECR defect from invading the root canal. In addition, bonelike tissue can be seen *(yellow arrow)*. Posttreatment view **(F)** and radiograph **(G)** after replacement of the upper left central incisor with an implant-retained crown. (From Patel S, Kanagasingam S, Pitt Ford T: External cervical resorption: a review, *J Endod* 35:616, 2009.)

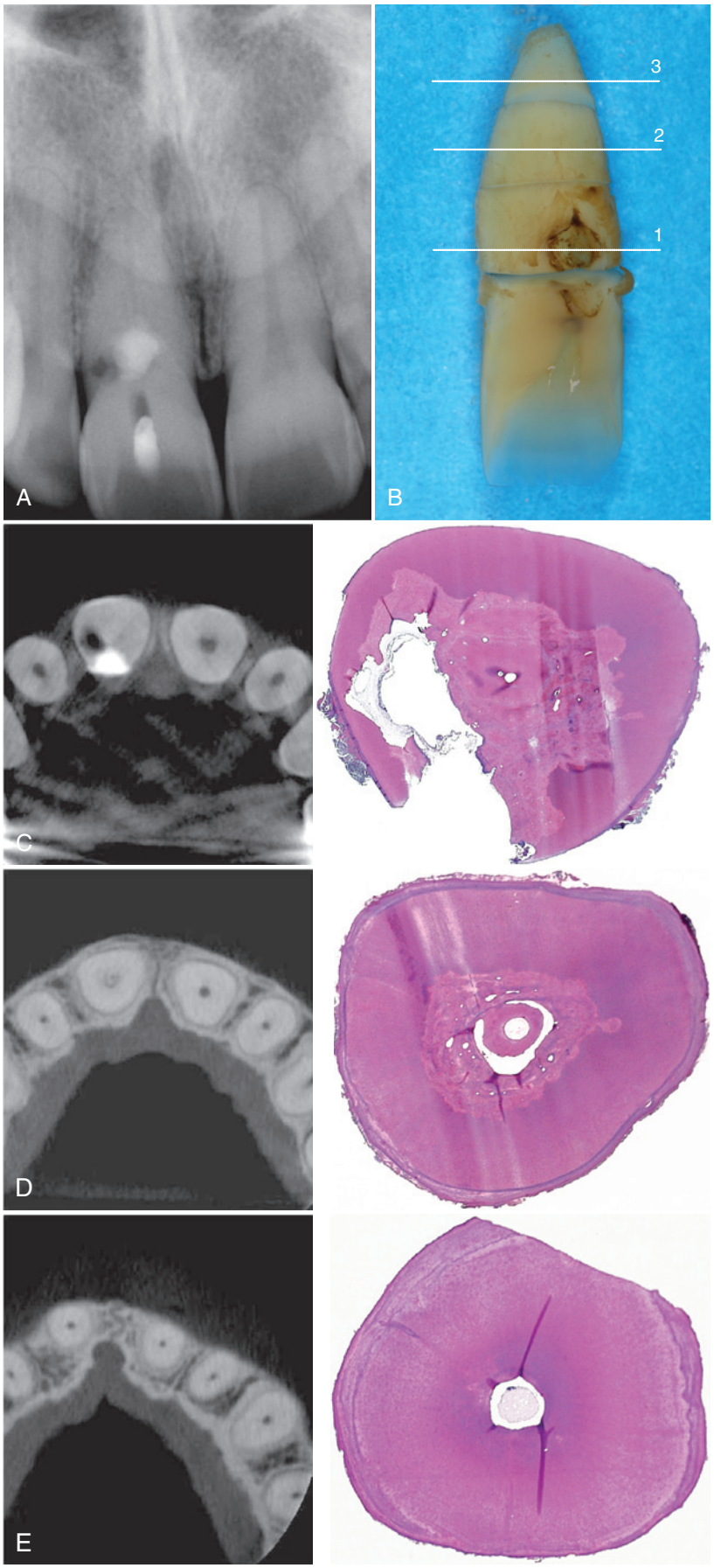

FIG. 16-8 External cervical resorption (ECR) with replacement. **A,** Radiograph of a maxillary central incisor of a 34-year-old female patient who recalled being struck in the face with a cricket ball when she was 11 years old. The dentist mistakenly diagnosed the resorptive defect as caries and attempted to manage it accordingly. The tooth was asymptomatic; however, a 4-mm periodontal probing depth was identified on the palatal aspect of the tooth. A cone beam computed tomography (CBCT) scan confirmed an ECR defect. The tooth was deemed unrestorable and was extracted with the patient's consent. **B,** Palatal view of the tooth at the end of the demineralization process, while immersed in the clearing agent. The tooth was separated into four portions, which were embedded separately in paraffin blocks. **C,** CBCT axial section passing through the coronal third at the level of line 1 in **B.** The corresponding histologic section shows that most of the dentin has been replaced by a bonelike tissue. The root canal is no longer present. (H&E stain; original magnification ×8.) **D,** CBCT axial section passing through the middle third at the level of line 2 in **B.** The root canal can be appreciated at this level, although reduced in size, but it appears to be encircled by bonelike tissue (original magnification ×8). **E,** CBCT axial section taken from the apical third at the level of line 3 in **B.** At this level the canal is the same size as that of the contralateral tooth. The histologic section confirmed the absence of bonelike tissue (original magnification ×8).

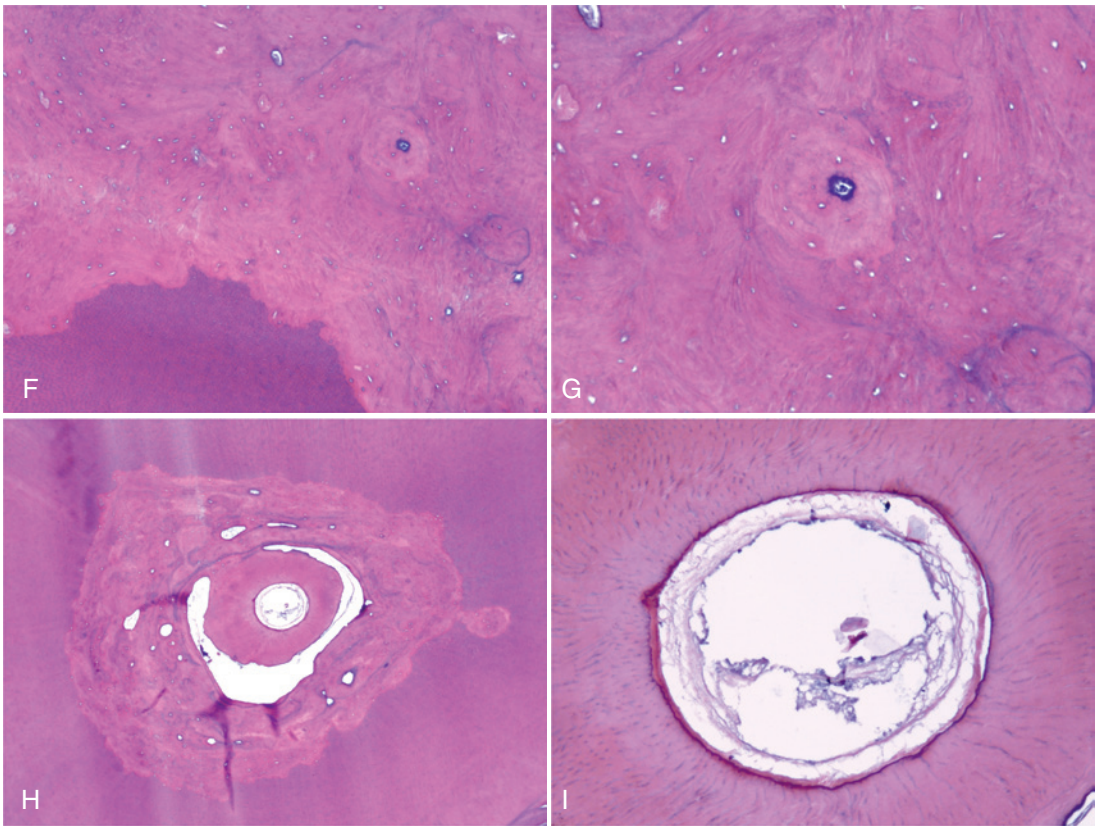

FIG. 16-8, cont'd F, Detail from C showing the transition from dentin to bonelike tissue (original magnification ×100). G, Higher magnification of F. The metaplastic tissue does not show the lamellar structure typical of bone (original magnification ×400). H, Detail of the canal in D. The metaplastic bonelike tissue has concentrically replaced a consistent portion of dentin, leaving in place a reduced layer of the original dentin (original magnification ×25). I, High-power view confirms that the tissue surrounding the canal is dentin (original magnification ×400). *Considerations:* The resorption process, followed by replacement with a bonelike tissue, started in the cervical area and extended in an apical direction, tunneling dentin circumferentially up to the transition between the middle and apical thirds of the tooth.

Despite its often cervical location, ECR may commence apical to this region, reflecting the position of the epithelial attachment of the affected tooth. In teeth with a normal periodontal attachment, the invasive nature of the process may result in the lesion extending some distance apical and/or coronal to the cervical location where it started. Furthermore, the tissue destruction at the site of onset may sometimes be minimal and/or not evident on conventional radiographs due to its location on the root surface. In these instances the lesion may appear to have originated at a location where significant tissue destruction, evident radiographically, has occurred. This may be some distance from the actual point of origin. This feature has come to light only since the advent of assessment of ECR with CBCT.

The radiographic features of ECR are very similar to those of Internal Root Resorption (IRR) (discussed later), and differentiating between them, especially in the absence of clinical signs, may be challenging. It is useful to trace the outline of the root canal walls as they approach and pass through the resorption defect on the radiograph. In cases of ECR, the outline of the canal wall should be visible and intact and should maintain its course as it passes through the defect. This is due to the fact that the resorptive lesion lies on the external surface of the root and is not in communication with the root canal; it is merely superimposed on the defect radiographically. In cases of IRR, it should be possible to trace the outline of the root canal through the resorptive defect because the defect is an extension of the root canal wall and is continuous with it.[51] Although this is a useful diagnostic feature, it does have some shortcomings. First, the outline of the root canal wall may be obscured by calcified tissue in the resorptive defect (ECR or IRR). Second, when ECR has resulted in extensive tissue destruction, perforation of the root canal wall may have allowed communication between the canal wall and the external defect.

Parallax radiographs should always be used to obtain further information about the nature of the resorptive process. In addition to a paralleled periapical radiograph, another radiograph should be taken with a shift (parallax) in the horizontal angulation of the x-ray tube in relation to the image receptor. In cases of ECR, the position of the resorptive defect moves relative to the root canal. If the lesion is located palatally/lingually, the defect moves in the same direction as the x-ray tube shift. If the lesion is located buccally, it moves in the opposite direction. This is sometimes referred to as the "same lingual, opposite buccal" (SLOB) rule. In contrast, internal resorptive defects maintain their position relative to the root canal because the defects are an extension of the root canal system.

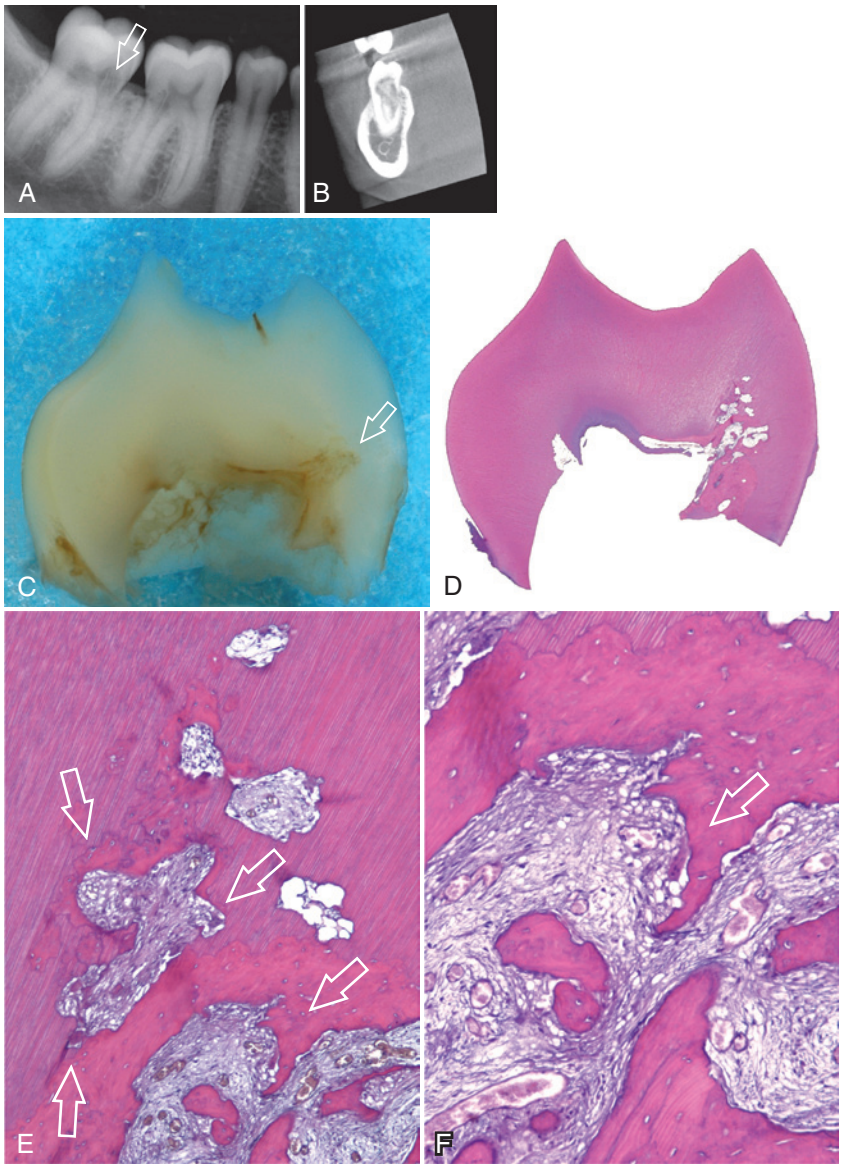

FIG. 16-9 External cervical resorption with replacement. **A,** Mandibular first and second molars in a 27-year-old female patient. The patient had symptoms of irreversible pulpitis associated with the lower right second molar; the lower right first molar was asymptomatic. **B,** CBCT scan taken through the second molar at the level of the area indicated by the arrow in **A.** Note massive resorption involving the crown and root. **C,** Mesial portion of the crown of the second molar after clearing. **D,** Section taken on a buccolingual plane. Overview shows resorption and replacement on the lingual side, corresponding to the area indicated by the arrow in **C.** (H&E stain; original magnification ×6.) **E,** Detail of the area indicated by the arrow in **C.** Several areas of resorption, with replacement by metaplastic tissue, can be seen (original magnification ×100). **F,** Magnification of the area indicated by the right lower arrow in **E.** The metaplastic tissue closely resembles bone (original magnification ×400).

CBCT has allowed 3D assessment of the nature, position, and extent of the resorptive defect, eliminating diagnostic confusion and providing essential information about the restorability and subsequent management of the tooth. CBCT is particularly useful if the clinician is not sure whether the ECR cavity has perforated the root canal wall (and thus for determining the need for root canal treatment). A CBCT scan eliminates the need for exploratory treatment. A clinical study by Patel et al.[88] comparing the ability of conventional radiographs and CBCT to diagnose and to differentiate accurately between IRR and ECR showed that CBCT was significantly more accurate (100%) than periapical radiographs at diagnosing the presence and nature of the root resorption. These researchers also concluded that the correct treatment plan should incorporate the additional information provided by CBCT.[88]

Management

The fundamental treatment objectives in ECR are to excavate the resorptive defect, halt the resorptive process, restore the hard tissue defect with an aesthetic filling material, and prevent and monitor the tooth for recurrence. Endodontic treatment of the affected tooth is necessary when the resorptive process has perforated the root canal wall. Surgical access

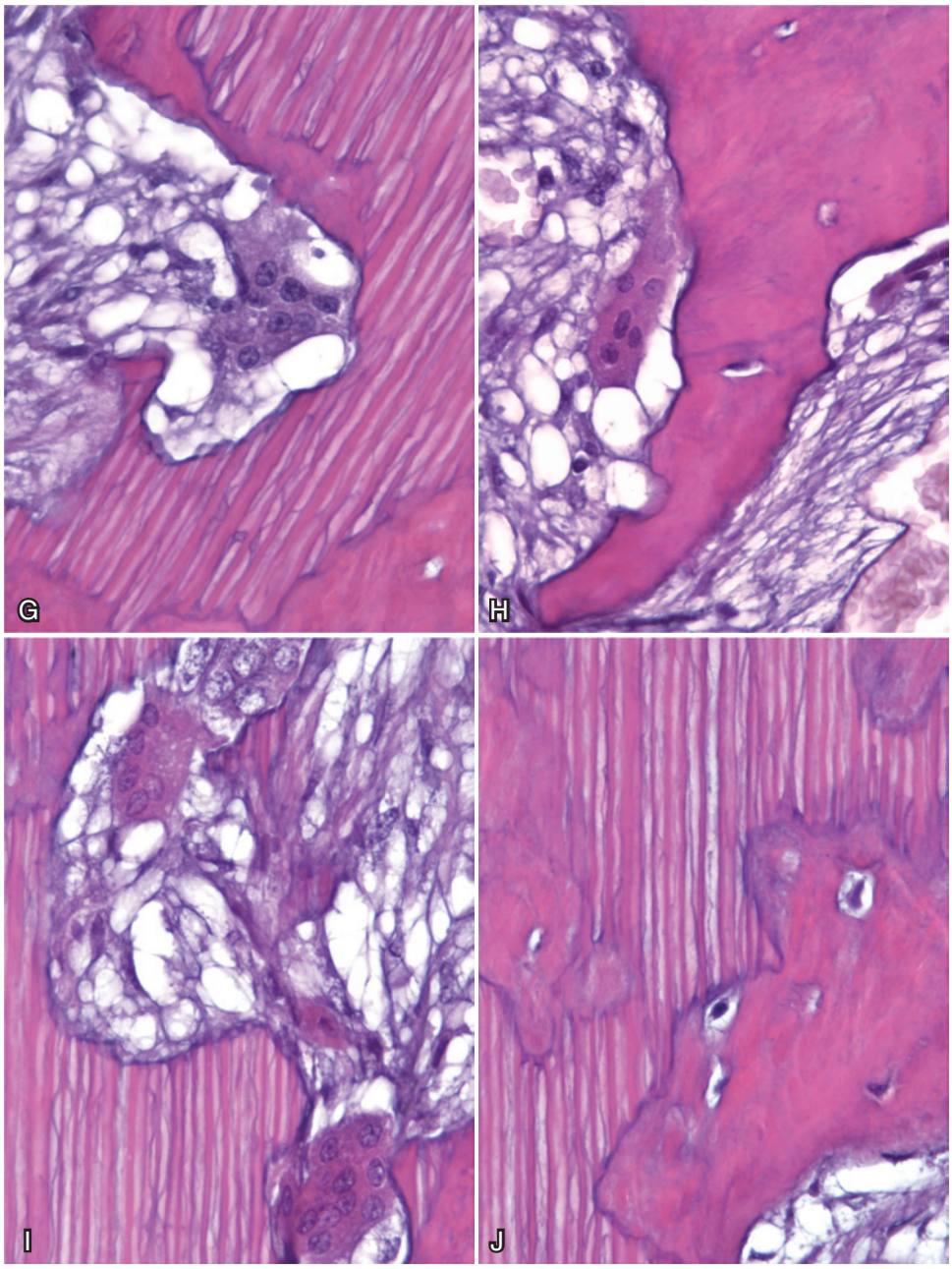

FIG. 16-9, cont'd G, High-power view of the area indicated by the right upper arrow in E. Note the lacuna in dentin occupied by a multinucleated clastic cell (original magnification ×1000). H, High-power view of the metaplastic bone tissue in the area indicated by the arrow in F. A bone trabecula is being resorbed by a typical osteoclast (original magnification ×1000). I, High-power view of the area indicated by the left lower arrow in E. A Howship lacuna with odontoclasts can be seen (original magnification ×1000). J, High-power view of the area indicated by the left upper arrow in E. Note the islands of bone tissue, with typical osteocytes, surrounded by dentin (original magnification ×1000). *Considerations:* In this case the metaplastic tissue was similar to normal lamellar bone. It is interesting that the bone tissue was undergoing remodeling, as evidenced by the presence of osteoclasts. Osteoclasts and odontoclasts can be observed in the same area and show morphologic similarity.

to the site of resorption is gained by raising a mucoperiosteal flap, the dimensions of which should allow visualization of the full extent of the defect (see Fig. 16-10). Once access has been achieved, the resorptive cavity is excavated. Fibrovascular granulomatous tissue is readily removed with a hand excavator. However, defects containing significant amounts of fibro-osseous tissue (especially when the latter is contiguous with the adjacent dentin) require discriminate removal of the tissue with ultrasonic instruments. It may be extremely difficult to differentiate between sound dentin and fibro-osseous deposits; therefore, use of the surgical operating microscope is essential. Frequent intraoperative radiographs may be necessary to ensure accurate removal of unwanted, hard resorptive tissue and to prevent unnecessary removal of sound dental tissue. The value of a preoperative CBCT scan in these cases cannot be overemphasized. The scan allows

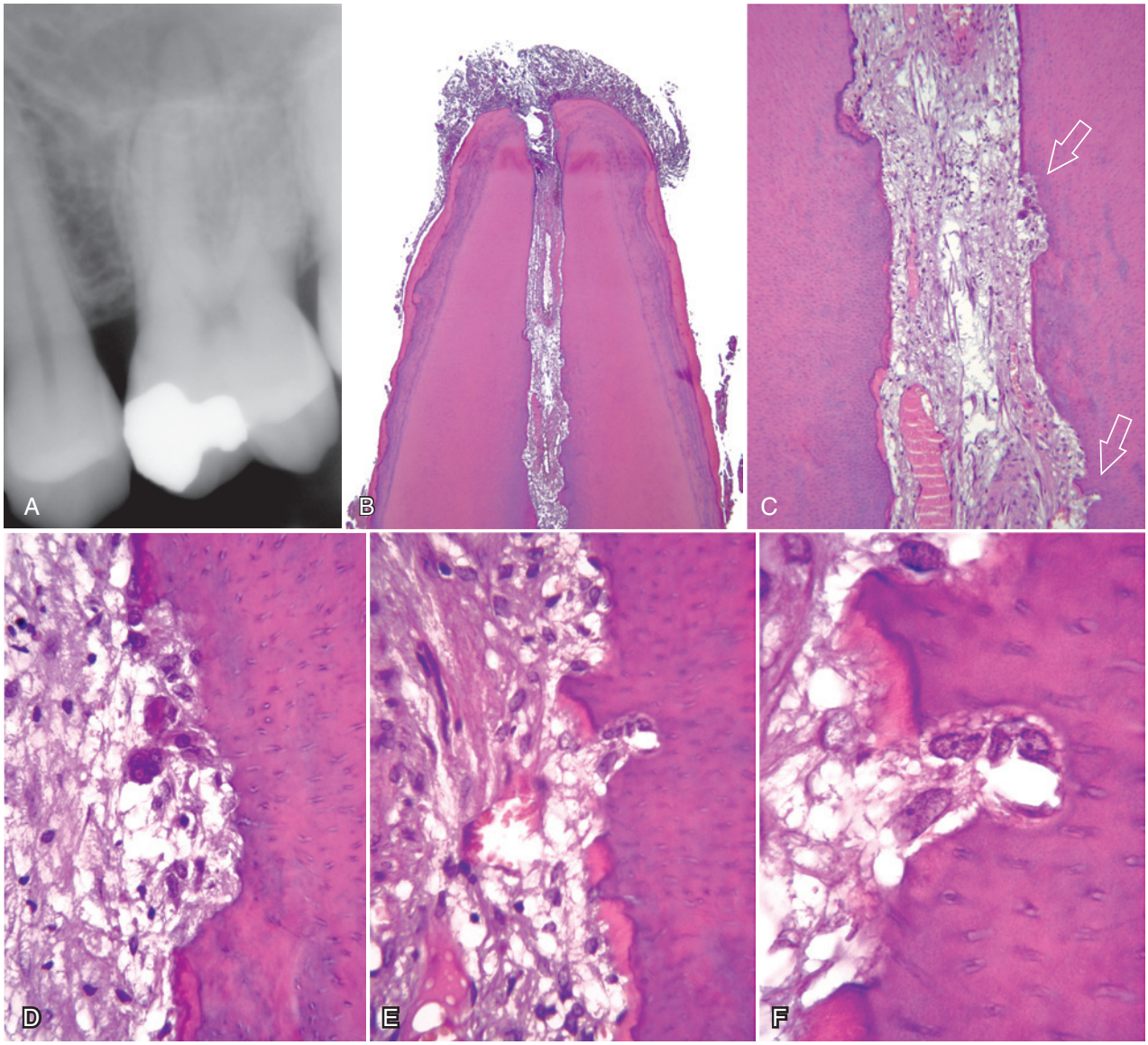

FIG. 16-10 Early internal root resorption. **A,** A 54-year-old male patient presented with a long story of pain on chewing and with cold stimuli. Recently the pain had become continuous and severe. The maxillary first molar, which had a mesial amalgam restoration, did not respond to sensitivity tests. A radiograph showed a periapical radiolucency on the palatal root. After removal of the restorative materials, a crack line involving the pulp chamber floor was diagnosed, and the tooth was extracted. **B,** Some pathologic tissue remained attached to the palatal root apex at extraction. A longitudinal section passing at the center of the canal demonstrated vital connective tissue in the apical third. (H&E stain; original magnification ×16.) **C,** Detail of the canal in **B.** Note the connective tissue with vessels and relatively few inflammatory cells, in addition to the resorption lacunae on the root canal walls (original magnification ×1000). **D,** High-power view of the area of the canal wall indicated by the upper arrow in **C.** Note the resorption lacuna with odontoclasts (original magnification ×1000). **E** and **F,** Progressive magnification of the area of the canal wall indicated by the lower arrow in **C.** Note the Howship lacuna housing clastic cells. Predentin is present in some areas of the wall, but it is absent in the areas with active resorption (original magnification ×400 and ×1000).

accurate interpretation of the full extent of the defect in all planes. True measurements can be made from the scan and related to clinical reference points, reducing greatly the need for subjective tissue removal.

Once the resorptive tissue has been removed, the cavity may be treated with a 90% aqueous solution of trichloracetic acid; this causes coagulation necrosis of the resorptive tissue without damaging the periodontal tissue.[61] The acid also penetrates and treats small channels of resorption that are not accessible to mechanical instrumentation.[89] Once the defect has been excavated and treated with trichloroacetic acid, any undermined dentin or enamel at the peripheries of the cavity are removed with a bur in a high-speed handpiece, and the cavity is restored with an aesthetically acceptable restorative material, such as composite resin or glass ionomer cement. Biodentine may prove to be a particularly suitable material for restoring these

defects[30,85] because it may combine acceptable aesthetics with the ability to support PDL attachment.

Once the cavity has been restored, the mucoperiosteal flap is replaced and secured in position. If perforation of the root canal wall has occurred, root canal treatment should be carried out. Access to the root canal system should be gained under a rubber dam before the resorptive defect is assessed. The root canal should be prepared in the area of the defect as normal, using saline as an irrigant. A tapered gutta-percha (GP) point then should be placed in the canal to maintain its patency during the excavation and restoration of the resorptive defect and to provide a barrier against which the final restoration can be condensed. The rubber dam is removed, and surgical treatment of the resorptive defect can be carried out as described, without any risk of the resorptive debris entering the root canal system. After repositioning of the mucoperiosteal flap, root canal treatment can be completed in the normal manner, without fear of extrusion of infected tissue, irrigants, or medicaments into the periodontal tissues.

Heithersay[62] advocated a nonsurgical approach to the treatment of ECR. In this technique, a 90% aqueous solution of trichloroacetic acid is applied to the resorptive tissue in the defect until coagulation necrosis occurs. The necrosed tissue is then excavated, and the defect is restored with glass ionomer cement.

The treatment of ECR depends on the severity, extent, and location of the resorptive defect and the restorability of the tooth. Heithersay[61] developed a four-stage classification system for ECR based on the depth of penetration of the resorption in a buccolingual and apicocoronal direction. He examined the prognosis of treatment in 101 cases of ECR in 94 patients using the nonsurgical protocol referred to previously, and related the success rates for treatment to the classification of the lesion.[61] He reported a 100% success rate for class I and class II lesions, a 77.8% success rate for class III lesions, and a 12.5% success rate for class IV lesions. This emphasizes the poorer outcome that can be expected for more advanced cases. A major limitation of the Heithersay classification is that it is valid only if the ECR lesion is confined to the proximal aspect of the tooth, because lesions are assessed on 2D radiographs. If the ECR lesion is located on and/or extends to the labial and/or buccal (proximal) aspects of the tooth, the true nature of the lesion cannot be accurately assessed with radiographs.[86,89]

INTERNAL ROOT RESORPTION

Introduction

Internal root resorption (IRR) is a form of root resorption that originates in and affects the root canal wall.[9] It is further classified as either inflammatory or replacement. The replacement type is associated with the deposition of mineralized tissue in the root canal space after the initial loss of dentin (initial dentin loss is a feature of both types).[9] Because their characteristics are largely similar, the two types of IRR are discussed together.

Etiology and Pathogenesis of Internal Root Resorption

For IRR to occur, the outermost protective odontoblast layer and the predentin of the canal wall must be damaged, resulting in exposure of the underlying mineralized dentin to odontoclasts (Fig. 16-11).[106,115]

The precise injurious events necessary to bring about such damage have not been completely elucidated. Various etiologic factors have been proposed for the loss of predentin, including trauma, caries and periodontal infections, excessive heat generated during restorative procedures on vital teeth, calcium hydroxide procedures, vital root resections, anachoresis, orthodontic treatment, cracked teeth, or simply idiopathic dystrophic changes in normal pulps.[5,18,28,93,112] In a study of 25 teeth with internal resorption, trauma was found to be the most common predisposing factor, responsible for 45% of the cases examined.[29] The suggested etiologies in the other cases were inflammation as a result of carious lesions (25%) and carious/periodontal lesions (14%). The cause of the internal resorption in the remaining teeth was unknown. Other reports in the literature support the view that trauma[5,116,118] and pulpal inflammation/infection[58,118] are the major contributory factors in the initiation of internal resorption.

Wedenberg and Lindskog[115] reported that IRR could be a transient or progressive event. In an in vivo primate study, the root canals were accessed in 32 incisors with the predentin intentionally damaged. The access cavities in half of the teeth were sealed; those in the other half were left open to the oral cavity. The teeth were extracted at intervals of 1, 2, 6, and 10 weeks. The authors noted only a transient colonization of the damaged dentin by multinucleated clastic cells in the teeth that had been sealed (i.e., transient internal root resorption). Those teeth were free of bacterial contamination, and no signs of active hard tissue resorption occurred. The teeth that were left unsealed during the experimental period showed signs of extensive bacterial contamination of pulpal tissue and dentinal tubules. Those teeth demonstrated extensive and prolonged colonization of the damaged dentin surface by clastic cells and signs of mineralized tissue resorption (progressive internal root resorption). As previously mentioned, damage to the odontoblast layer and predentin of the canal wall is a prerequisite for initiation of internal root resorption.[116] However, progression of internal root resorption depends on bacterial stimulation of the clastic cells involved in hard tissue resorption. Without this stimulation, the resorption is self-limiting.[116]

For IRR to continue, the pulp tissue apical to the resorptive lesion must have a viable blood supply; this provides clastic cells and their nutrients, and the infected necrotic coronal pulp tissue provides stimulation for those clastic cells (see Fig. 16-11).[105] Bacteria may enter the pulp canal through dentinal tubules, carious cavities, cracks, fractures, and lateral canals. In the absence of a bacterial stimulus, the resorption is transient and may not advance to the stage that can be diagnosed clinically and radiographically. Therefore, the pulp apical to the site of resorption must be vital for the resorptive lesion to progress (see Fig. 16-11). If left untreated, internal resorption may continue until the inflamed connective tissue filling the resorptive defect degenerates, advancing the lesion in an apical direction. Ultimately, if left untreated, the pulp tissue apical to the resorptive lesion undergoes necrosis and the bacteria infect the entire root canal system, resulting in apical periodontitis.[96]

Histologic Appearance

Wedenberg and Zetterqvist[118] reported on the histologic nature of IRR. The authors specifically examined the histologic,

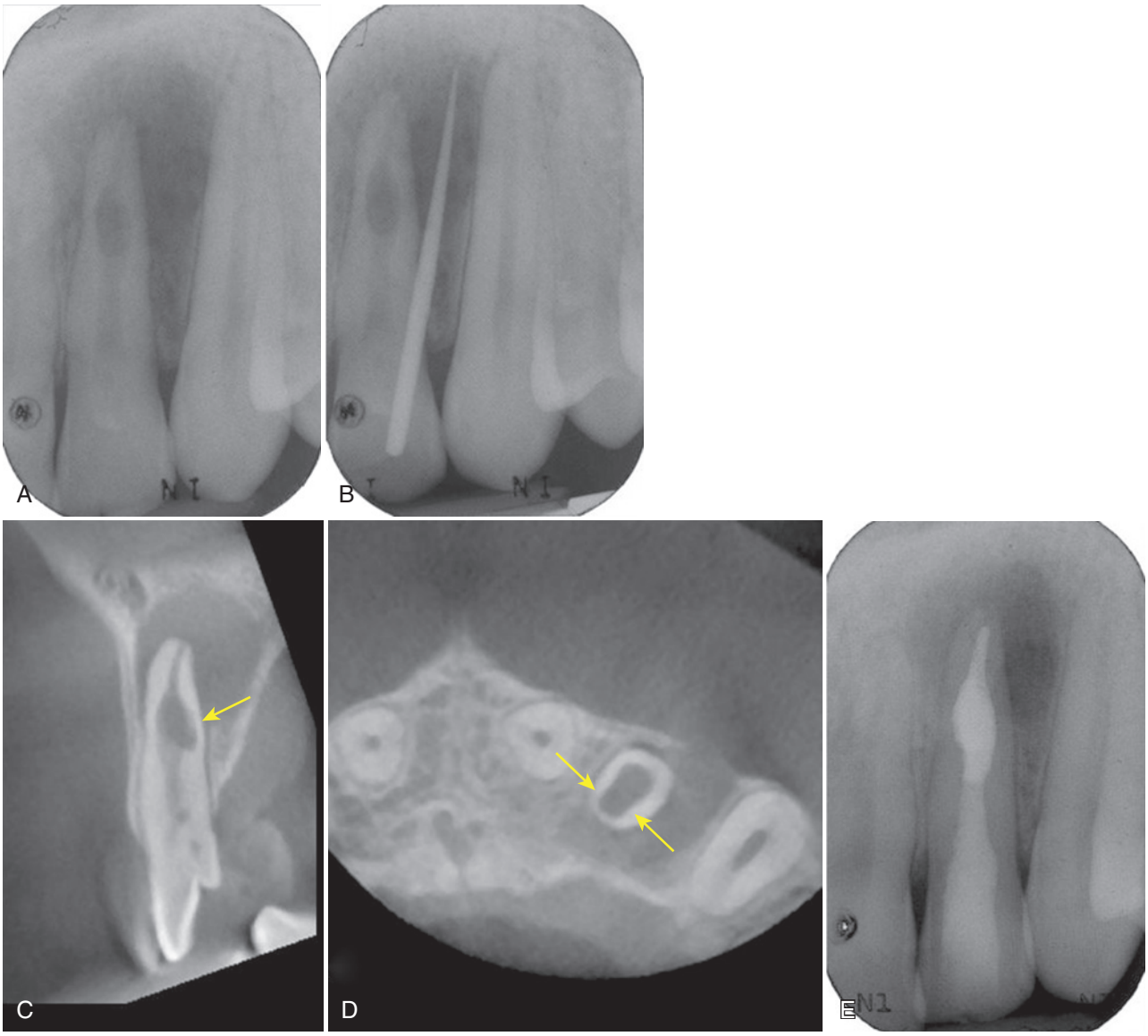

FIG. 16-11 A and B, Parallax views of the maxillary left lateral incisor showing internal root resorption with necrosis. A gutta-percha point has been used to track the sinus. The reconstructed sagittal (C) and axial (D) slices from a CBCT scan reveal that the lesion has resorbed the palatal aspect of the root *(arrows)* and has nearly perforated the root wall. E, The tooth has been obturated with gutta-percha using a thermoplasticized technique. (From Patel S, Ricucci D, Durack C, Tay F: Internal root resorption: a review, *J Endod* 36:1107, 2010.)

enzyme histochemical, and scanning electron micrographic (SEM) features of the resorptive process in a small sample of primary and secondary teeth extracted due to progressive IRR. The histologic and enzyme histochemical profiles were identical for the two groups, but the resorptive process appeared to occur more rapidly in the primary teeth. The pulpal tissue was populated to varying degrees in all teeth with an inflammatory infiltrate composed predominantly of lymphocytes and macrophages, with some neutrophils. The connective tissue in the pulp spaces was less vascular than healthy pulpal tissue and resembled periodontal membrane connective tissue with relatively more cells and fibers. The odontoblast layer and predentin were absent from the affected dentinal walls, which were populated by large, multinucleated odontoclasts occupying resorption lacunae. The odontoclasts showed evidence of active resorption (see Fig. 16-11). They were accompanied by mononuclear cells, believed to be odontoclast precursors, which populated the connective tissue adjacent to the site of resorption. Both types of cell displayed tartarate-resistant acid phosphatase (TRAP) activity.

Interestingly, the root canal wall was incompletely lined with a mineralized tissue resembling bone or cementum in all of the examined teeth.[118] Furthermore, islands of calcified tissue of a similar nature occupied the pulpal space in three of the cases. Islands of mineralized tissue occupying the root canal space are the defining feature of internal replacement resorption.[9] The authors suggested that deposition of this mineralized tissue is likely to be part of a coupling process, at the

end of a period of resorption, in which osteoblasts are attracted to the affected site and participate in bone formation.[118]

Clinical Features

The clinical features of IRR largely depend on the histologic status of the affected pulp, the extent of the hard tissue destruction caused by the resorptive process, and the position of the resorptive cavity in the root canal space. In the active stages of resorption, bacterial contamination of vital pulpal tissue may cause an acute inflammatory response, leading to clinical symptoms of pulpitis. With the onset of pulpal necrosis and an established bacterial colonization of the root canal space, clinical signs and symptoms associated with acute or chronic apical periodontitis may develop. Sinus tract(s) may occur and may be associated with suppuration in the periapical tissues or possibly at the site of a perforation of the root canal wall caused by the hard tissue destruction. Extensive resorption of the coronal pulp may result in a pink or red discoloration visible through the crown of the affected tooth; this is caused by granulomatous tissue extending into and occupying the resorptive defect.[75] Although often reported as a common clinical indicator of the process, these pink spots actually are rare in cases of IRR. They may occur relatively more frequently in cases of ECR, but they are not very common with that resorption type, either. Often the affected tooth is asymptomatic, and clinical signs are absent.

Radiographic Features and Diagnosis

The diagnosis of any type of root resorption depends on radiographic demonstration of its presence. The two-dimensional nature of conventional radiographic imaging makes the detection and differentiation of the various types of resorption challenging. This is especially true when attempts are made to differentiate between IRR and ECR, which may have similar radiographic features.[57,90,91,105] Much has been reported in the literature about the "typical" radiographic features of IRR. Gartner et al.[51] reported that lesions of IRR present radiographically as radiolucencies of uniform density that have a smooth outline and are symmetrically distributed over the root of the affected tooth (Fig. 16-12). The authors further reported that the outline of the root canal wall should not be traceable through the resorption defect because the root canal wall balloons out. Other authors have described IRR lesions as oval, circumscribed radiolucencies in continuity with the root canal wall.[82] Although certain cases of IRR may have some or all of these radiographic features, many do not; each case should be assessed individually before a diagnosis is made.

IRR can occur at any location in the root canal system and may manifest radiographically as a radiolucency with variable shape, radiodensity, outline, and symmetry in relation to the root canal. Internal inflammatory root resorption lesions are more likely be uniformly radiolucent, whereas in internal replacement (metaplastic) root resorption, the defect has a somewhat mottled or clouded appearance as a result of the radiopaque nature of the calcified material occupying the lesion (Fig. 16-13).[91] ECR lesions may contain predominantly granulomatous tissue, predominantly calcified tissue, or a mixture of the two; therefore, they may have a radiodensity similar to either type of internal resorption, which complicates the clinical differentiation of the disease process. As noted by Gartner et al.,[51] the best practice is to trace the outline of the root canal wall as it approaches and leaves the resorption defect. An IRR cavity is continuous with the normal root canal walls because it is essentially an extension of them. As such, in teeth with single canals affected by IRR, the canal walls should not be traceable through the defect. This is in contrast to ECR, in which the lesion lies buccal or palatal/lingual to the defect and is consequently superimposed on the canal system when viewed on conventional radiographs. In this situation the canal walls should maintain their normal course as they pass through the resorption defect, allowing them to be traced through it. However, it should be noted that in teeth with multiple canals, a canal that has been unaffected by IRR may be superimposed onto the IRR resorption defect on conventional radiographs. When the guidelines outlined by Gartner et al.[51] are used, this may lead to misdiagnosis.

Parallax radiographs must always be used to obtain further information about the resorptive process. In addition to a paralleled periapical radiograph, a radiograph should be taken with a shift in the horizontal angulation of the x-ray tube in relation to the image receptor. IRR lesions maintain their position relative to the root canal system on the angled view. ECR lesions move in the same direction as the x-ray tube shift if they are lingually/palatally positioned and move in the opposite direction if they are buccally located.[51] This diagnostic technique, coupled with tracing of the root canal/pulp chamber outline through the lesion, has been the most reliable aid in the differential diagnosis of IRR when conventional radiography is used. However, as discussed previously, the amount of information available from conventional radiographic imaging is limited. This can lead to misdiagnoses and incorrect treatment in the management of IRR and invasive cervical resorption.

The use of CBCT as a diagnostic and treatment planning tool in the management of IRR has been reported in the literature.[21] Information such as the position, extent, and dimensions of an IRR lesion, in addition to the presence of any associated perforation, can be obtained from a CBCT scan. The same scan can differentiate between ECR and IRR, removing any doubt about the diagnosis that may have arisen with the conventional radiographic examination.

Management

Once a diagnosis of IRR has been made, the extent of the hard tissue destruction must be assessed and a clinical decision must be made about the prognosis of the affected tooth. If the affected tooth is salvageable and has a reasonable prognosis, root canal treatment is necessary. As with any infected tooth, the main purpose of the root canal treatment is to remove the intraradicular bacteria and disinfect the root canal space. If the resorptive process is still active, the treatment serves an adjunctive purpose, which is to eliminate the vital apical tissue that is sustaining and stimulating the resorbing cells.

The nature of the resorptive process in cases of IRR presents the endodontist with unique operative challenges. In teeth with active resorption, profuse bleeding from the granulomatous and inflamed pulpal tissues may impair visibility in the initial stages of treatment and may provide a stubborn source of mild hemorrhage when attempts are made to dry the canal after chemomechanical preparation. Furthermore, the irregularly concave nature of resorption defects makes them inaccessible to direct mechanical debridement.

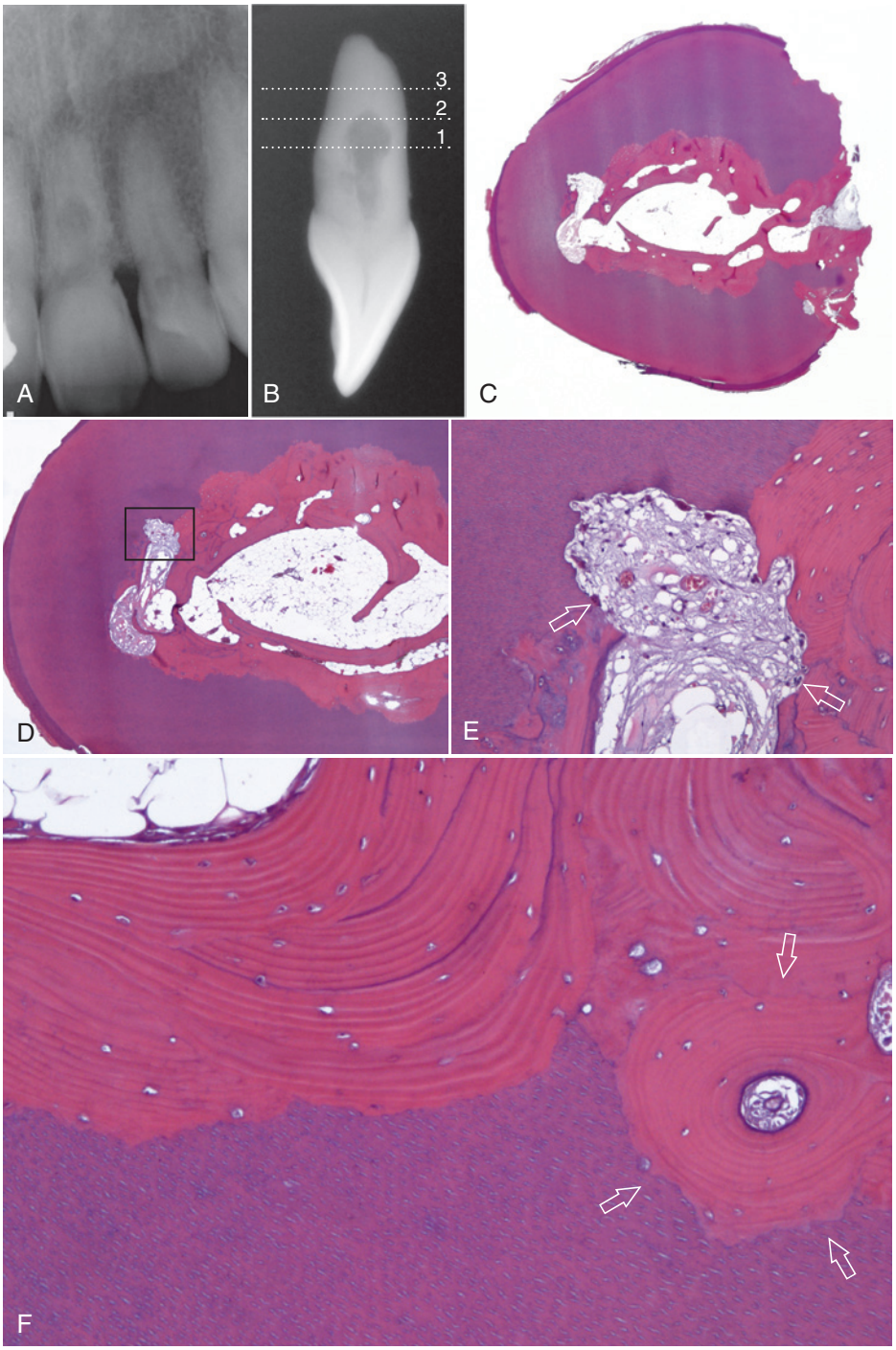

FIG. 16-12 Light microscopy images of a tooth with internal (root canal) replacement resorption. The tooth belonged to a 44-year-old male patient who was referred to the first author for management of a perforated root. The tooth was asymptomatic on examination, but there was a history of previous trauma. **A,** Radiograph of a maxillary central incisor with a radiolucent lesion in the middle third of the root canal. The radiolucent lesion appears to be mottled, which suggests internal root resorption with metaplasia. **B,** Clinical radiograph of the tooth after extraction, taken at a 90-degree angle, showing the continuity of the resorptive lesion with the canal space. **C,** Cross section taken approximately at the level of line 1 in Fig. 16-12, *B.* The low-magnification overview shows that the dentin around the root canal had been replaced by an ingrowth of bone tissue, and the root appears to have been perforated on the distopalatal aspect. (H&E stain; ×8.) **D,** Higher magnification of Fig. 16-12, *C.* (H&E stain; ×16.) **E,** High magnification of the area demarcated by the rectangle in Fig. 16-12, *D.* The intraradicular dentin has been resorbed. (H&E stain; ×100.) **F,** High-magnification view taken from the right part of Fig. 16-12, *C,* showing that the resorbed dentin has been replaced by lamellar bone. Osteocytes are present in lacunae between the lamellae. A characteristic cross section of an osteon can be seen on the right *(open arrows),* with concentric lamellae surrounding a vascular structure. (H&E stain; ×100.)

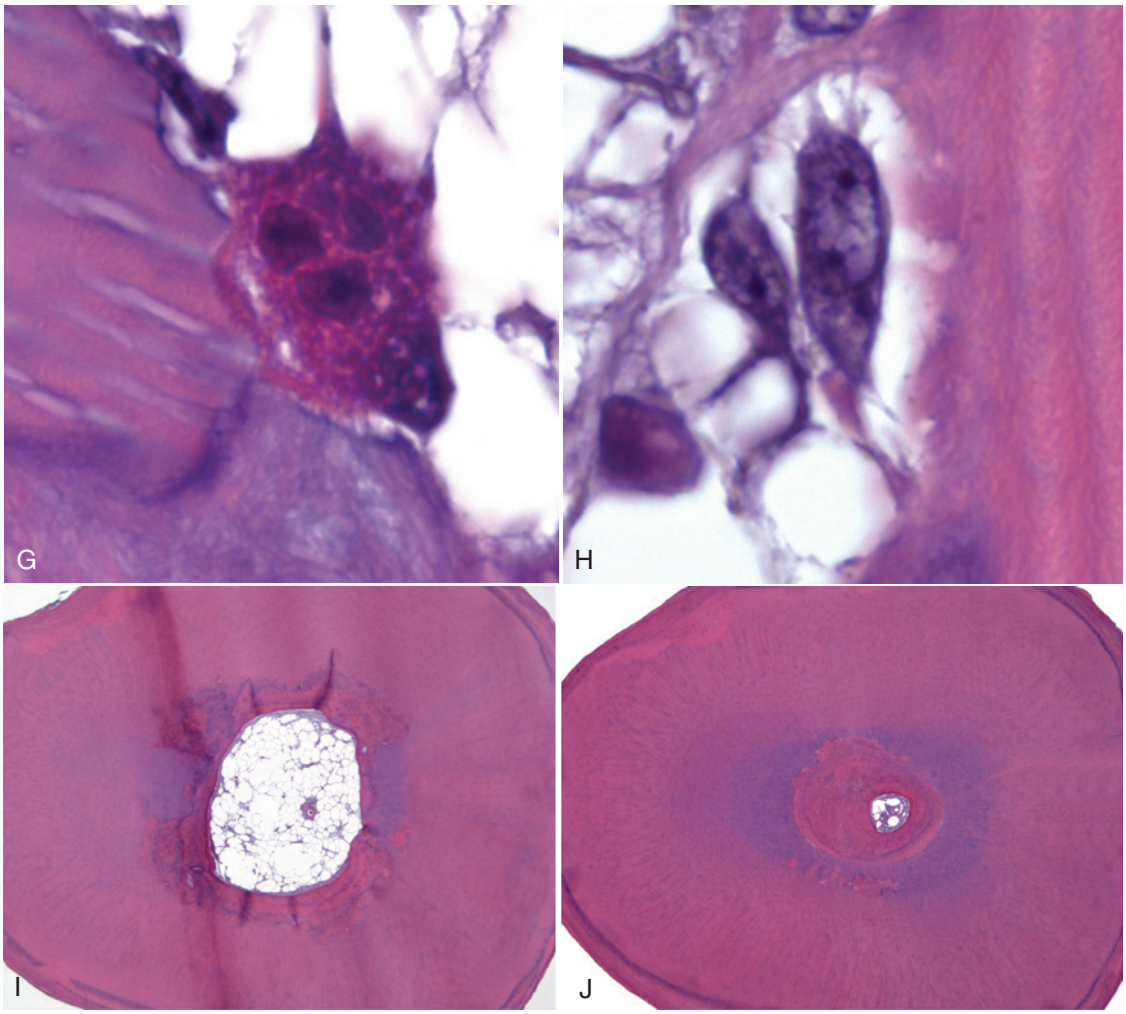

FIG. 16-12, cont'd **G,** High-magnification view of the area indicated by the left open arrow in Fig. 16-12, *E*. A multinucleated resorbing cell (odontoclast) can be seen in a dentinal lacuna, indicating active resorption of the dentinal wall. (H&E stain; ×1000.) **H,** High-magnification view of the bone surface indicated by the right arrow in Fig. 16-12, *E*. The large cells are osteoblast-like cells. Once they produced mineralized tissue, they were embedded in the bone lacunae, assuming the characteristics of osteocytes. (H&E stain; ×1000.) **I,** Cross section taken approximately at the level of line 2 in Fig. 16-12, *B*. The root canal is still large at this level and is surrounded by a relatively thin layer of newly formed bone. (H&E stain; ×16.) **J,** Cross section taken approximately at the level of line 3 in Fig. 16-12, *B*. At this level the root canal appears consistently narrowed by a dense layer of newly formed bone. (H&E stain; ×16.) (From Patel S, Ricucci D, Durack C, Tay F: Internal root resorption: a review, *J Endod* 36:1107, 2010.)

Chemomechanical Debridement of the Root Canal

The complex anatomic and morphologic features of root canal systems provide unique recesses that may harbor microorganisms in infected teeth. Endodontic instruments and passively delivered irrigants fail to penetrate into these secluded spaces and niches.[80,97,99] The use of ultrasonic instruments to aid the penetration of endodontic irrigants has been shown to improve the removal of organic debris and biofilms from the root canal space.[26] Given the inaccessibility of IRR defects to normal instrumentation and passive irrigation, ultrasonic activation of irrigants should be considered an essential step in the treatment of these cases (see Fig. 16-11). However, even when this adjunctive measure is used, microbes may persist in confined areas after chemomechanical debridement.[26] As such, an intracanal antibacterial medicament should be used to further reduce the microbial load and improve the disinfection of the root canal space.[99] Calcium hydroxide is an antibacterial, interappointment, endodontic medicament that has been shown to eradicate bacteria persisting in the root canal space after root canal treatment.[27,100] Also, when used in conjunction with sodium hypochlorite, it potentiates the effect of that irrigant in the removal of organic debris from the root canal system.[2,108] Based on this evidence the authors advocate the use of calcium hydroxide as an intracanal, antibacterial medicament to supplement the conventional chemomechanical debridement of the root canal system.[91]

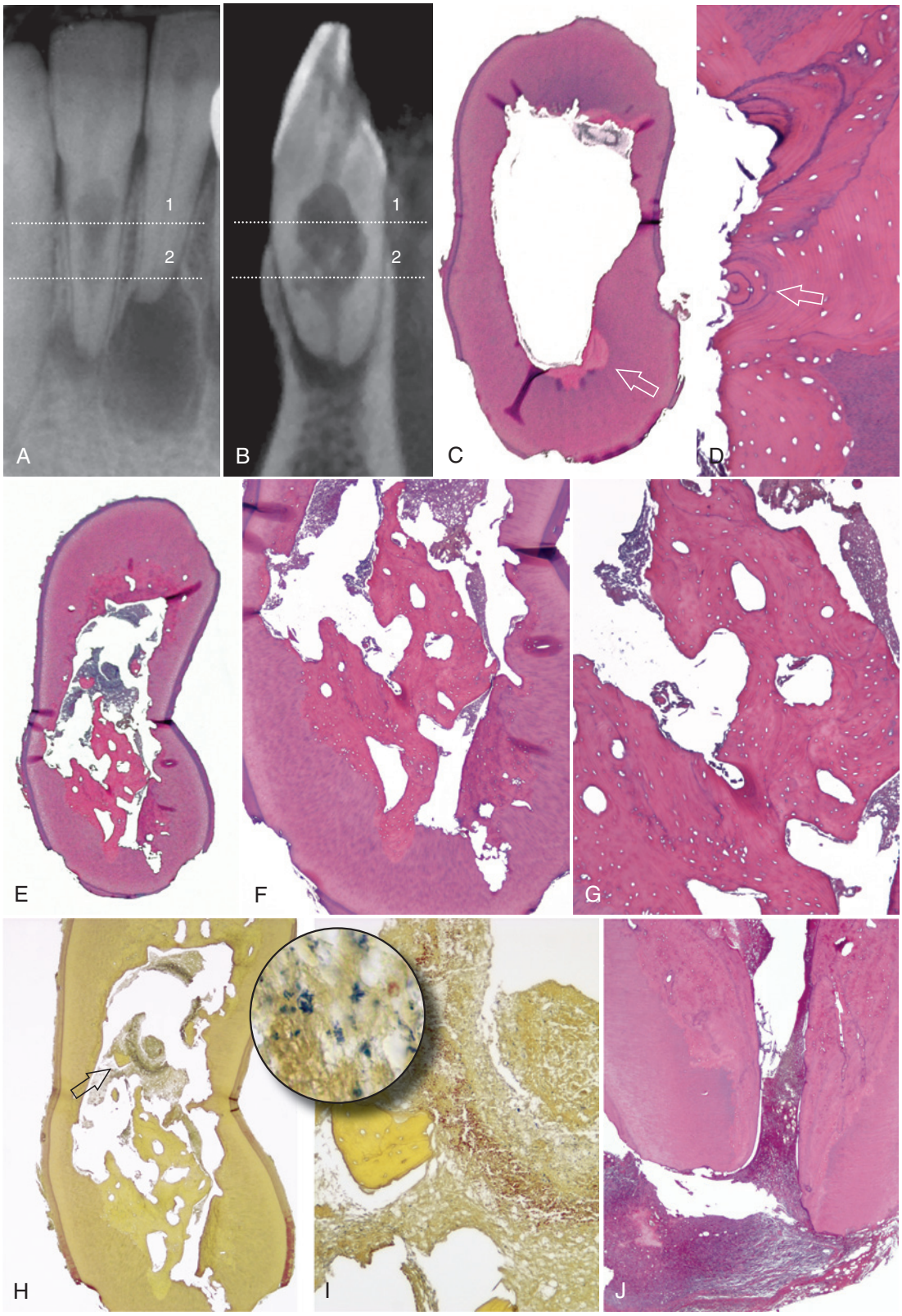

FIG. 16-13 Light microscopy images of a variant of internal (root canal) replacement resorption with tunneling resorption. The lower right lateral incisor belonged to a 39-year-old former boxer who had suffered a jaw fracture in a boxing match in his early twenties and was placed in intermaxillary fixation. The patient developed symptoms 20 years later and complained of pain associated with his lower incisors. **A,** Radiograph of the mandibular right incisors. The lower right central incisor had asymptomatic apical periodontitis associated with a necrotic and infected pulp. The lower right lateral incisor showed a large area of internal root resorption. The tooth did not respond to sensitivity tests. **B,** Sagittal CBCT slice shows some calcified tissue in the resorptive defect. **C,** Cross section taken at the level of line 1 in Fig. 16-13, *A* and *B*. The overview shows that the canal was apparently empty at this level. (H&E stain; ×6.) **D,** High magnification of the area indicated by the arrow in Fig. 16-13, *C*. Lamellar bone filling an area of previous resorption. Note the osteon structure *(arrow)*. (H&E stain; ×100.) **E,** Cross section taken at the level of line 2 in Fig. 16-13, *A* and *B*. Overview shows that the canal lumen was partly occupied by necrotic remnants, partly by bonelike tissue. (H&E stain; ×8.) **F,** High magnification of the lower part in Fig. 16-13, *E*. (H&E stain; ×50.) **G,** Higher magnification of Fig. 16-13, *F*. Bone trabeculae are surrounded by necrotic debris. (H&E stain; ×100.) **H,** Cross section taken from the same area as that in Fig. 16-13, *E*. (Taylor's modified Brown & Brenn [TBB] stain; ×16.) **I,** High magnification of the area indicated by the arrow in Fig. 16-13, *H*. A fragment of bonelike tissue can be seen surrounded by bacteria-colonized necrotic tissues. (TBB stain; ×100; inset ×1000.) **J,** Longitudinal section passing approximately through the center of the root apex. Dentin walls have been resorbed and replaced by a bonelike tissue. (H&E stain; ×16.) (From Patel S, Ricucci D, Durack C, Tay F: Internal root resorption: a review, *J Endod* 36:1107, 2010.)

Obturation

One of the fundamental objectives of endodontic treatment is to fill the disinfected root canal space completely with an appropriate material. In IRR, the hard tissue defects caused by the resorptive process are challenging to fill adequately. To obturate the resorptive defect, the root-filling material must be able to flow. Gutta-percha is widely regarded as the gold standard filling material in endodontics. It can plasticize when pressure is applied, and it becomes flowable with the application of heat. Gencoglu et al.[52] examined the ability of different obturation systems and techniques to fill the defects in artificially created internal resorptive cavities ex vivo. They found that Obtura II (ObII) and Microseal (MS) thermoplastic gutta-percha systems produced significantly better fills in simulated resorptive cavities than did Thermafill, soft core systems (SCS), and cold lateral condensation (CLC). In a similar study Goldberg et al.[55] demonstrated that the Obtura II system filled simulated resorptive defects statistically better than CLC, Thermafill, and a hybrid technique. Gencoglu et al.[52] reported that the Obtura II and Microseal systems produced fills with greater gutta-percha–to-sealer (GP/sealer) ratios than did the other techniques examined. These findings were corroborated in the study by Goldberg et al.[55]

Because root canal sealers shrink on setting[120] and dissolve and degrade to varying degrees in the presence of moisture,[78] fillings with higher GP/sealer ratios reduce the risk of void formation and leakage of contaminants into the root canal system, with potentially positive benefits for the treatment outcome.

Stamos and Stamos[103] and Wilson and Barnes[121] reported on cases of internal resorption in which the Obtura system was successfully used to obturate the canal. In light of the evidence presented, the Obtura and Microseal systems apparently can be expected to produce the best technical results for obturating canals with IRR.

When choosing the appropriate materials and methods for filling resorptive defects of IRR, the clinician first must establish the presence and extent of any perforations in the wall of the affected root. This information can be readily obtained from an appropriate CBCT scan. If perforation has occurred, mineral trioxide aggregate (MTA) should be considered the material of choice to repair the root wall. MTA is biocompatible,[79] has superior sealing properties when used as a retrograde filling material,[17] and has proved effective in the repair of lateral and furcal root perforations in animal studies.[76] Furthermore, the material is well tolerated in the periapical tissues, and when used as a root-end filling material in the absence of infection, it supports almost complete regeneration of the adjacent periodontium.[94] These are desirable properties in the context of perforation repair because of the very real possibility of unintentional extrusion of the material when a perforating internal resorptive defect is repaired in an orthograde manner. However,, the flow properties of MTA are significantly poorer than those of heated GP. Its use as an effective filling material in IRR depends on adequate ultrasonic activation of the material to disperse it into the recesses of the defect.[21] Use of a dental operating microscope and the correct equipment to deliver the material are essential.

A hybrid technique to obturate canals affected by perforating internal resorption also may be used. In these cases the canal apical to the resorption defect is filled with GP. The GP can then be used as a barrier against which the MTA can be packed. Hsiang-Chi Hsien et al.[63] successfully used this technique to treat a perforating IRR defect in a maxillary incisor. Jacobowitz and de Lima[66] described a case in which a maxillary central incisor with a large, perforating internal resorption defect had a poor prognosis and initially was designated for extraction. The tooth subsequently was successfully treated in an orthograde manner with white MTA and gutta-percha.[66]

Clinical situations arise in which a perforating resorptive defect causes extensive dental hard tissue destruction that fails to respond to or is not amenable to repair with an orthograde approach. Surgical treatment may be needed in these cases. For the reasons discussed already, MTA would be the material of choice to repair these perforations. In cases that have not already been treated in a nonsurgical manner, the operator first must access the root canal as for orthograde treatment. A well-fitting, tapered GP point or an appropriately sized finger spreader is then positioned in the canal to occlude it and to provide a barrier against which the MTA can be packed once surgical access to the defect has been gained. The barrier also prevents inadvertent deposition of the MTA into the apical third of the canal. The perforation is then exposed surgically and repaired with the MTA. The canal can be shaped, disinfected, and obturated with thermoplasticized GP once the MTA has set.

If the resorptive process has caused sufficient tissue destruction to render the tooth unrestorable, extraction is the most appropriate treatment option. If the tooth has been weakened by the disease process to the extent that fracture of the tooth root is likely, the patient may elect to have the tooth extracted. The presence of a perforating resorptive defect is certainly not a contraindication to treatment, but a perforation of significant size will have a bearing on the decision to surgically treat or extract the tooth. CBCT is an invaluable component of the clinician's armamentarium in the treatment of IRR.[42] A CBCT scan gives the clinician a 3D view of the tooth, the resorptive defect, and the adjacent anatomy. The clinician thus has the information necessary to determine a prognosis for the tooth and/or its amenability to surgical repair. If extraction of the affected tooth is indicated, the scan may be used as a diagnostic and treatment planning tool for provision of a dental implant–retained prosthesis. Bhuva et al.[21] described a case in which CBCT was used as a treatment planning tool in the successful treatment of a case of IRR. As mentioned previously, CBCT has been found to be highly accurate in revealing the nature of the resorptive lesion, and this leads to the selection of the most suitable treatment plan.

SUMMARY

- The prevalence, etiology, and pathogenesis of various types of root resorption are not fully understood, and more research is required in these areas.
- More clinical data are required on the presenting features of ECR because this particular type of resorption may vary significantly in its presentation.
- Early detection of root resorption is essential for successful management and favorable outcomes.
- CBCT is an excellent diagnostic tool for confirming the presence of ECR and IRR and also for appreciating the true nature of these conditions and managing them.

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CHAPTER 17

Diagnosis of Nonodontogenic Toothache

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CHAPTER OUTLINE

Review of Neuroanatomy
Somatic Structures
Neural Structures
Autonomic Nervous System
Review of Neurophysiology
Peripheral Sensitization
Central Sensitization
Terminology
Clinical Entities That Can Present as Toothache
Sources of Odontogenic Toothache
Sources of Nonodontogenic Toothache
Taking a Patient's History
Patient Examination
Additional Tests
Case Studies
Summary

An unthinking dentist is a bad dentist. Perfect technique misapplied is at least as unconscionable as sloppy work.

—Marjorie Jeffcoat

A nonodontogenic toothache is, of course, an oxymoron. How can one have a toothache that is not odontogenic in etiology? The answer lies in the differentiation of people's perceptions of where they sense their pain, termed the *site of the pain,* from the location of a pathophysiologic process giving rise to the pain that may or may not be in the same region, termed the *source of the pain.* This concept of the attribution of pain to an anatomic region that is different from the location of the etiologic process is generically known as the *referred pain phenomenon* and can occur in multiple areas of the body. Thus, a nonodontogenic toothache has a source of pain that is not the tooth the patient has indicated, clearly demonstrating the diagnostic challenge (Fig. 17-1).

Pain is common. It causes human suffering and has significant socioeconomic effects. Pain is a motivator that provokes individuals to seek care. But protracted chronic pain debilitates and can significantly impair the quality and productivity of a person's life. One survey revealed that 66% of respondents reported experiencing pain or discomfort over a 6-month period. Significantly, 40% of respondents reported that this pain affected them to a "high degree."[19] A study published in 2003 estimated the lost productive work time attributed to common pain conditions among active workers to cost $61.2 billion per year.[127] One investigator reported that over a 6-month period, 22% of Americans experienced at least one of five types of facial pain. Of these pains, the most common type (12.2%) was toothache.[81]

Although toothache is the most common pain entity occurring in the facial region,[81] many other types of pain can occur in the same general area. A primary responsibility of a dental practitioner is to diagnose pathologic entities associated with the oral cavity and masticatory apparatus. Many of these pathologic entities have pain as a primary component of their presentation. Because dental practitioners are sought out daily for the alleviation of odontogenic pain, it is imperative for them to have a basic working knowledge of other types of facial pain in order to make an accurate diagnosis and properly select care for patients. It is paramount to realize that not all pain entities presenting as toothache are of odontogenic origin. The presenting toothache may be a heterotopic symptom of another disorder. A heterotopic symptom is perceived to originate from a site that is different from the tissue that is actually the source of the pain. This is in contrast to primary pain, in which the perceived site of pain is the actual tissue from which the pain originates. Before discussing pain entities that mimic toothache, it is helpful to understand the neurobiologic mechanisms of orofacial pain.

REVIEW OF NEUROANATOMY

Somatic Structures

To understand the pathways by which orofacial pain occurs, one must first gain a basic understanding of the structures involved in its transmission to higher brain centers (see also Chapter 4). Structures of the orofacial region can be divided

FIG. 17-1 Pantomogram of a patient who has undergone several endodontic procedures without resolution of her chief complaint. (Courtesy Dr. Jeffrey Okeson, Lexington, Kentucky.)

into two broad categories: somatic and neural structures. Somatic structures are those that make up the different nonneural tissues and organs. The somatic structures can be further anatomically divided into superficial and deep structures. Superficial structures include the skin, mucosa, and gingiva; pain that arises from these superficial structures is usually well localized (e.g., a sharp explorer penetrating the gingiva results in well-localized pain). Deep structures include musculoskeletal and visceral tissues. Pains from these deep structures are typically poorly localized and diffuse in nature.

Neural Structures

Neural structures involved in the perception of pain include the afferent (toward the brain) and efferent (away from the brain) regulation of somatic structures. Nerve impulses are transmitted from orofacial structures to the brain via the peripheral nervous system, whereas modulation and interpretation of these impulses into what we feel as pain occurs in the central nervous system. Pain can arise solely from either central or peripheral nervous tissue but heterotopic pain, which is often involved with nonodontogenic toothache, likely requires central modulation to occur.

Peripheral Nervous System

Pain arises as a result of tissue damage, or the potential for tissue damage, and is transmitted via terminal nerve fibers known as primary afferent nerve fibers. Two major classes of nociceptive (or pain-sensing) primary afferent nerve fibers can detect potentially damaging noxious stimuli: the A-delta and C fibers. Both fiber types have a wide distribution throughout the skin, oral mucosa, and tooth pulp. In addition, separate classes of nerve fibers exist that are involved in detecting nonnoxious stimuli such as vibration and in proprioception. These fibers can be found in the periodontal ligament, skin, and oral mucosa and include the A-beta fibers.

Primary Afferent Neurons

Primarily the trigeminal, or fifth cranial, nerve detects and encodes noxious stimuli for the orofacial region. The majority of cell bodies of the trigeminal sensory fibers are in the trigeminal ganglion located on the floor of the middle cranial fossa. The peripheral axons of the trigeminal ganglion run in three divisions—the ophthalmic (V1), maxillary (V2), and mandibular (V3)—which innervate most of the oral mucosa, the temporomandibular joint, the anterior two thirds of the tongue, the dura of the anterior and middle cranial fossae, the tooth pulp, the gingiva, and the periodontal membrane.

In the peripheral nervous system, these neurons or nerves are referred to as primary afferent (i.e., sensory) fibers. The primary afferent fibers can broadly be divided into A-beta fibers, which transmit light touch or proprioceptive information, and A-delta and C fibers, which encode pain. The tooth is densely innervated by afferent nerve fibers, which are believed to transmit mainly pain in response to thermal, mechanical, or chemical stimuli. The majority of dental nerves are C fibers that innervate the central pulp, most of which terminate beneath the odontoblasts.[23]

A-beta Fibers

The rapidly conducting myelinated neurons that respond to light touch are called A-beta fibers. Under normal conditions, activation of the A-beta fibers by high-intensity stimulation results in low-frequency output in the central nervous system. Activation of A-beta fibers normally is interpreted as nonpainful mechanical stimulation[133] or, under certain conditions, can be perceived as a "prepain" sensation.[23] A-beta fibers also have been shown to undergo phenotypic changes that allow them to encode painful stimuli under certain inflammatory conditions.[98]

A-delta Fibers

The A-delta fibers are lightly myelinated, have a faster conduction velocity than C fibers, and are believed to transmit a sharp or pricking sensation. A-delta fibers respond primarily to noxious mechanical stimuli rather than to chemical or thermal stimuli. Other A-delta fibers may be polymodal (responding to mechanical, chemical, and thermal stimuli)[13] or respond only to cold/mechanical[78] or hot/mechanical noxious stimuli.[39]

In the tooth pulp, A-delta fibers traverse the odontoblastic layer and terminate in the dentinal tubules.[25] Because of their location and their sensitivity to mechanical stimulation, A-delta fibers are believed to respond to stimuli that result in

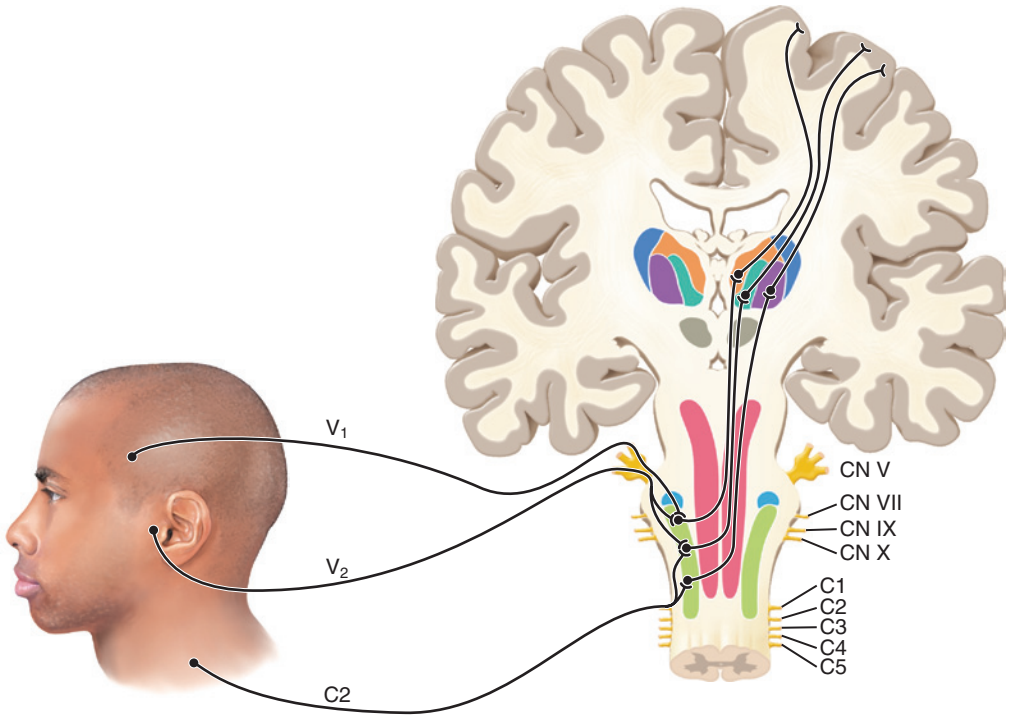


FIG. 17-2 A graphic depiction of the trigeminal nerve entering the brainstem. The primary afferent neuron synapses with a second-order neuron in the trigeminal nucleus. The second-order neuron carries pain information to the thalamus, from which it is sent to the cerebral cortex for interpretation. (Redrawn from Okeson JP: *Bell's orofacial pains,* ed 5, Chicago, 1995, Quintessence Publishing.)

movement of fluid within the dentinal tubules (e.g., osmotic, mechanical probing, or thermal stimuli applied to the external surface of the tooth).[18] Consistent with the hypothesized mechanism of dentinal pain is the fact that the stimuli that cause dentinal fluid movement result in a sharp pain associated with A-delta fiber activation.[95] When intense noxious stimuli activate the A-delta fibers, the input to the central nervous system consists of high-frequency action potentials.

C Fibers

The C fibers are unmyelinated, have slower conduction velocity, and are associated with a dull, aching, or burning sensation. Most C fibers are polymodal, responding to mechanical, thermal, and chemical stimuli. Because of the difference in conduction velocities, A-delta fibers are believed to transmit early, shooting pain, whereas C fibers would transmit late, dull pain. Noxious stimuli that exceed the receptor threshold of these nociceptive primary afferent terminals result in action potentials that travel centrally, signaling tissue damage. In the pulp tissue, the more centrally located C fibers respond to thermal, mechanical, and chemical stimuli and are believed to be sensitized by inflammation.[39] All visceral structures are innervated primarily by afferent fibers conducting nociceptive information such as that carried by A-delta and C fibers.

Central Nervous System

The primary afferent fibers are responsible for the transduction and transmission of sensory information to higher brain centers, and they do so by synapsing on neurons located within the trigeminal nucleus, which spans the midbrain and cervical spinal cord. This point marks the beginning of the central nervous system and is the point at which processing of pain information begins (Fig. 17-2).

Just as there are different types of sensory neurons in the periphery, in the trigeminal nucleus there are also different types of neurons that receive nociceptive input from the periphery. The ascending neurons located in the trigeminal nucleus are known collectively as *second-order* or *projection neurons* and can be subdivided into three distinct groups of neurons based on the type of information they receive: (1) low-threshold mechanoreceptors, (2) nociceptive-specific, and (3) wide dynamic range neurons.

The primary central site of termination for nociceptive fibers is the subnucleus caudalis, located in the most caudal region of the trigeminal nucleus,[39,57,144] which anatomically and functionally resembles the dorsal horn of the spinal cord and has been referred to as the medullary dorsal horn.[57] Four major components of nociceptive processing are located in the dorsal horn of the subnucleus caudalis: central terminals of afferents, local circuit neurons (interneurons), projection neurons, and descending neurons.[71] Within the subnucleus caudalis, the A-delta and C fibers terminate primarily in the outer laminae (I and IIa) and lamina V. Local circuit neurons are composed of islet cells (which are thought to be inhibitory) and stalked cells (which are believed to be excitatory).[38] Combined, the local circuit neurons may modulate nociceptive transmission from the primary afferents to the projection neurons.

The fourth component of the dorsal horn are the terminal endings of descending neurons. The descending neurons originate in the nucleus raphe magnus (NRM), the medullary reticular nuclei, and the locus ceruleus (LC). Descending brainstem neurons release serotonin (from the NRM) or

norepinephrine (from the LC), which may inhibit the activity of projection neurons directly or by activating local opioid interneurons. These neurons are responsible for the endogenous abatement of pain; blockade of their activity increases pain transmission and reduces pain thresholds.

Second-Order Neurons

Projection neurons have axons that cross to the contralateral medulla to ascend in the trigeminothalamic tract and project to the ventral posterior medial and intralaminar nuclei of the thalamus, where additional neurons project to the cortex. Projection neurons involved in the transmission of painful stimuli can be divided into two classes: wide dynamic range and nociceptive-specific neurons. Wide dynamic range neurons receive input from mechanoreceptors, thermoreceptors, and nociceptors, whereas nociceptive-specific neurons are excited solely by nociceptors. These two types of projection neurons may be responsible for signaling the severity and location of pain, respectively.[79]

Multiple primary afferent neurons may synapse on a single projection (i.e., convergence). This occurs to a much greater degree in deep tissues as opposed to cutaneous tissues. Primary afferent fibers of nontrigeminal origin such as those derived from vagus, glossopharyngeal, facial, and cervical spinal ganglia have been shown to converge and synapse onto trigeminal projection neurons located as far caudal as spinal level C4.[74] This phenomenon of convergence may result in the clinical finding of pain that radiates beyond an area of tissue injury. Convergence may also explain why pain appears to be associated with a site other than the injured area. Interestingly, when projection neurons receive input from superficial and deep structures, the more superficial inputs usually predominate.[121] Thus, pain originating from deep structures would typically be referred to superficial areas (e.g., pain originating from the jaw muscles would typically be referred to the face rather than deeper structures).

Autonomic Nervous System

The stellate ganglia supply the entire sympathetic innervation of the orofacial region, which is located bilaterally at the level of the seventh cervical vertebra. Under normal conditions, sympathetic stimulation has no influence on sensory function. However, afferent sympathetic fibers in an area of trauma may become involved in the response to pain and may also play a role in chronic pain states. Specifically, C fibers in the area of partial nerve injury may become responsive to sympathetic nerve stimulation. The modulation of nociception by the sympathetic nervous system has been shown such that release of pain neurotransmitters may be altered in the presence of sympathetic agonists and by blockade of the sympathetic nervous system, using antagonists.[70] Whether the effects of sympathetic nerve fibers on pain transmission are direct (via homeostatic regulation) or indirect remains unclear. The parasympathetic division of the autonomic nervous system has not been shown to be involved in the development or modulation of pain.

REVIEW OF NEUROPHYSIOLOGY

Peripheral Sensitization

After tissue insult there is an inflammatory reaction that often produces pain. The severity of pain that follows is related to several aspects of the injury, such as the type, extent, and location; the innervation of the tissue; and the phase of the inflammation. In the nociceptive system, tissue injury can manifest itself as increased responsiveness or reduced thresholds to a noxious stimulus, referred to as hyperalgesia. Hyperalgesia can be partially accounted for by sensitization of nociceptors (primary hyperalgesia) and by central nervous system mechanisms (secondary hyperalgesia).

In the absence of tissue damage, activation of C or A-delta fibers produces a transient pain. This pain is believed to serve as a physiologic warning. When there is tissue injury, afferent fibers may be activated by lower intensity stimuli than usual, and the quality of pain may be more persistent and intense. This phenomenon is due, in part, to the sensitization of nociceptors, including an increase in spontaneous activity.

At the site of tissue injury, there are a number of inflammatory mediators that can directly or indirectly sensitize primary afferent nociceptors (see Chapter 12 for more details). These inflammatory mediators may be released from the local tissue cells, circulating and resident immune cells, vasculature and endothelial smooth muscle cells, and peripheral nervous system cells.

Central Sensitization

After peripheral tissue injury there is an afferent barrage from C fibers resulting from peripheral tissue inflammation, decreased afferent thresholds, and spontaneous firing of afferent fibers. When a second-order neuron receives a prolonged barrage of nociceptive input, the second-order neuron may also become sensitized. This results in a phenomenon referred to as central sensitization.[17] The result of central sensitization is enhanced processing (i.e., amplification) of neural impulses that are being transmitted to higher brain centers. Two effects of central sensitization are secondary hyperalgesia and referred pain.

Secondary hyperalgesia is an increased response to painful stimulation at the site of pain resulting from central nervous system changes. This is in contrast to primary hyperalgesia, which is a lowered pain threshold resulting from sensitization of peripheral neurons. Secondary hyperalgesia might be felt in superficial (e.g., gingiva or skin) or deep structures (e.g., muscles or teeth).

Terminology

In general, as research progresses and uncovers new ways for us to look at pain, the terminology changes. This can introduce some confusion, especially when older terms are used. Therefore, it may be helpful to present contemporary definitions of some of the basic terms and review some of the previously mentioned terms (Box 17-1).

CLINICAL ENTITIES THAT CAN PRESENT AS TOOTHACHE

Sources of Odontogenic Toothache

Before considering heterotopic pains that may present as toothache, it is important to fully understand odontogenic pain as a primary source for toothache. Only two structures serve as sources for primary odontogenic pain: the pulp–dentin complex and the periradicular tissues. The innervation of the pulp is similar to that of other deep visceral tissues, and in various states of pathosis will have pain characteristics similar to deep

BOX 17-1

Types of Pain

Pain
An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.[86]

Nociceptive Pain
Pain arising from activation of nociceptors.[86]

Neuropathic Pain
Pain arising as a direct consequence of a lesion or disease affecting the somatosensory system.[86,135]

Peripheral Sensitization
Increased responsiveness and reduced thresholds of nociceptors to stimulation of their receptive fields.[86]

Central Sensitization
Increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input.[86]

Heterotopic Pain
Any pain that is felt in an area other than its true source is heterotopic pain. There are three types of heterotopic pain: referred, central, and projected.[105,106] Referred pain is pain felt in an area innervated by a nerve different from the one that mediates the primary pain. Referred pain cannot be provoked by stimulation of the area where the pain is felt; rather, it is brought on by manipulation of the primary source of pain (Fig. 17-3). In addition, referred pain cannot be arrested unless the primary source of pain is anesthetized. The referral of pain tends to occur in a laminated fashion (Fig. 17-4). This is because peripheral nociceptors enter the spinal trigeminal tract in a laminated fashion. As a result there are general referral patterns in the face. In addition, the referral of pain is usually in a cephalad or upward direction. This is evidenced clinically in that pain from mandibular molars typically is referred to maxillary molars, as opposed to premolars or incisors.

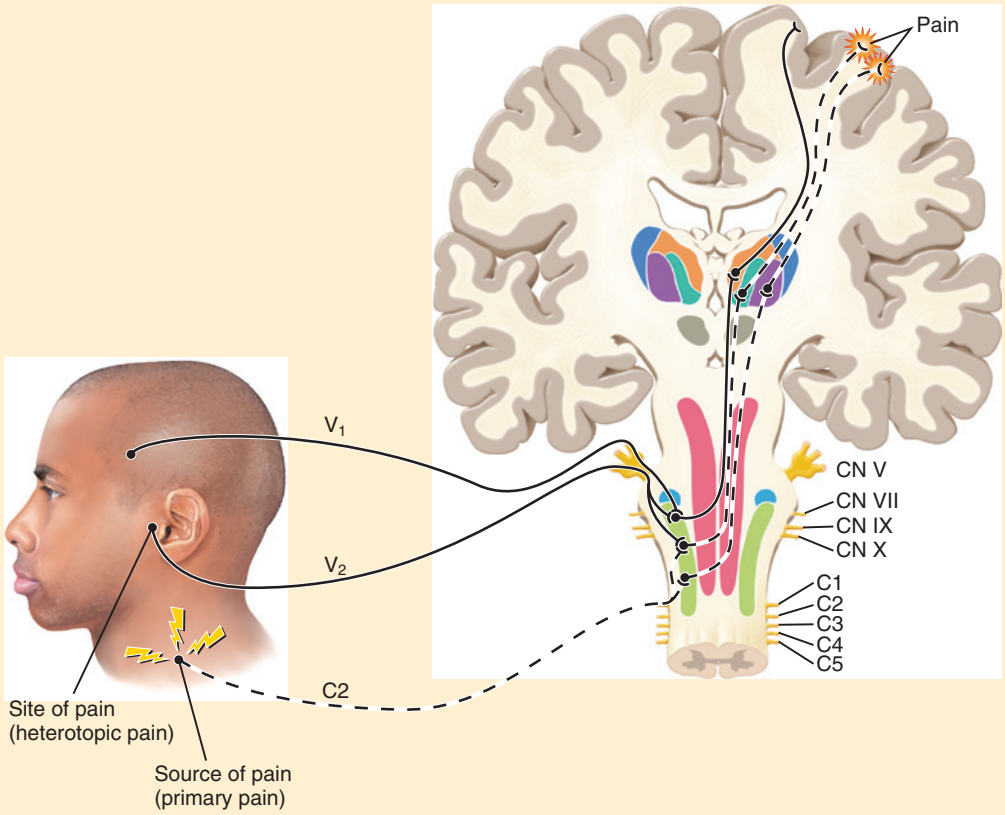


FIG. 17-3 Illustration of pain that is referred from an area innervated by one nerve *(C2)* to an area innervated by a different nerve *(V_2).* Note that this phenomenon occurs secondary to the convergence of different neurons onto the same second-order neuron in the trigeminal nucleus. The sensory cortex perceives two locations of pain. One area is the trapezius region that represents the source of pain. The second area of perceived pain is felt in the temporomandibular joint area, which is only a site of pain, not a source of pain. This pain is heterotopic (referred). (Redrawn from Okeson JP: *Bell's orofacial pains,* ed 5, Chicago, 1995, Quintessence Publishing.)

BOX 17-1

Types of Pain—cont'd

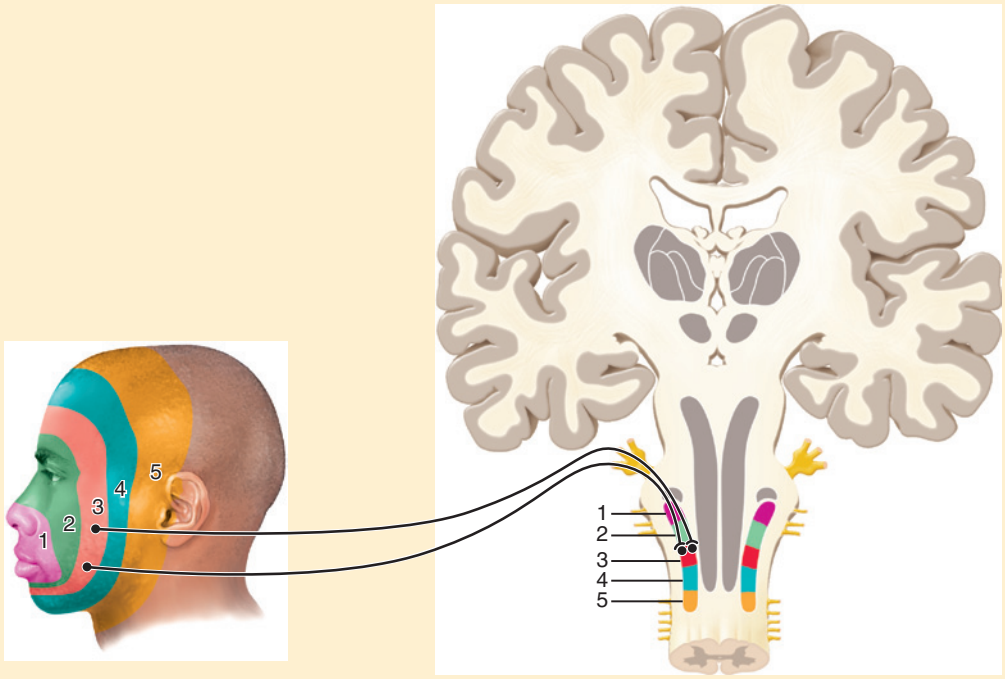


FIG. 17-4 Illustration of the laminated pattern of innervation from orofacial structures into the trigeminal nucleus. These laminated patterns commonly reflect the patterns of referred pains felt in the orofacial structures. (Redrawn from Okeson JP: *Bell's orofacial pains,* ed 5, Chicago, 1995, Quintessence Publishing.)

visceral tissues. The primary nociceptors of the pulp that respond to inflammation are the slow-conducting, high-threshold C fibers. Because their threshold is high and they rarely terminate in dentinal tubules, C fibers do not respond to normal or nonpathologic dentinal stimulation. C fibers typically conduct pain that is associated with tissue damage. In addition, C fibers respond in a threshold manner that can be termed "all or nothing." For example, a slightly cold stimulus that is below the C fiber threshold will fail to produce any sensation. Only when a stimulus is intense enough to reach the threshold will the C fiber fire, resulting in the sensation of pain.

Pulpal pain is mediated by C fibers and is dull, aching, or throbbing in nature. This is in contrast to the quick, short, sharp sensation produced by A-delta fibers that mediate dentinal pain. Therefore, when pulp testing, it is meaningful to note not only whether the patient perceived the stimulus but also the nature of the stimulus perceived. A simple notation would be to use an "s" (short) to indicate a response more typical of an A-delta fiber (dentinal pain) or a "p" (prolonged) to indicate the response was more indicative of a C fiber response (pulpal pain).

Tissue inflammation can result in sensitization of nerve fibers. When peripheral nociceptors (e.g., pulpal C fibers) are sensitized, the threshold of firing in response to a given stimulus (e.g., temperature and pressure) is lowered. In states of sensitization these nociceptors can be provoked with a less intense stimulus. The threshold for excitation is still "all or nothing" but the required level of stimulation has decreased. These fibers can become so sensitized that they may fire at as low a temperature threshold as body temperature,[95] normally not sufficient to stimulate a C fiber. In fact, they can become so sensitized that they will fire in response to the normal pulse pressure of cardiac contraction, eliciting a complaint of "I can feel my heartbeat in my tooth" or "my tooth is throbbing." Sensitized C fibers can even fire without provocation, resulting in spontaneous pain.

Similar to deep visceral tissues, the pulpal nociceptors demonstrate a high degree of convergence in the central nervous system. In a study of cat brain, 74% of the neurons tested in the subnucleus caudalis showed convergence from multiple tooth pulps.[22] In addition, the dental pulp has little to no proprioceptive neurons. The high degree of convergence from pulp tissue and the lack of proprioceptive information provided are the key factors in why purely pulpal pain can be so difficult for patients to localize. In addition to reducing localization of pain, convergence increases the referral of pain to tissues not actually affected by the inflammation. The fact that neurons from the pulps of mandibular teeth converge with those of maxillary teeth can result in pain from a mandibular pulpitis being referred to the maxillary arch. Because the patient may poorly localize pulpal pain, it is important for the clinician to localize the source of the pain. This is often accomplished through the use of tests that are employed in an attempt either to reproduce the eliciting stimulus of a patient's pain or to eliminate the pain. For example, pulpal pain should be aggravated with hot or cold stimulation and should be eliminated or significantly reduced with local anesthetic.

Unlike pulpal pain, pain of periradicular origin is easier to localize. Mechanoreceptors are numerous in the periodontal ligament (PDL) and are most densely concentrated in the apical one third.[87] Once inflammation from pulpal disease extends into the periodontal ligament, patients are able to locate the source of the pain much more readily. As a musculoskeletal structure, the PDL responds to noxious stimulation in a graded fashion—that is, the degree of discomfort a patient feels in relation to periradicular pain depends on the degree of peripheral sensitization and the amount of provocation to this structure. A sensitized PDL will be uncomfortable to a patient if percussed lightly but more uncomfortable if percussed heavily. This is known as a graded response. It is, therefore, appropriate to record periradicular testing such as percussion and palpation in terms of degrees of tenderness (versus "all or nothing"). As with pulpal pain, pain of periradicular origin should also have an identifiable etiology. Periradicular pain tends to be dull, aching, or throbbing and should resolve completely with local anesthesia. If pain of suspected periradicular origin is nonresponsive to local anesthetic, it is a strong indication that the pain may be nonodontogenic in origin.

The tooth is unique in the human body in that it has a visceral-like component, the pulp, and a musculoskeletal component, the periodontal ligament. Therefore, odontogenic pain can have a wide variety of presentations. Tooth pain can be diffuse or well localized, mild or intense, or spontaneous or provoked with various stimuli applied at various intensities. The quality can vary between sharp and dull and aching or throbbing. This potential for extreme variability makes it possible for toothaches to mimic or be mimicked by many other types of pain that occur in the head and neck. In addition, because both the pulp tissue and periodontal ligament can be categorized as deep somatic tissue, continued nociceptive input from odontogenic pain has a great propensity to produce central excitatory effects such as secondary hyperalgesia, referred pain, secondary co-contraction of muscles, myofascial trigger points, and autonomic changes. These effects add to the complexity of diagnosing odontogenic pain and differentiating tooth pain from other sources in the region.

Sources of Nonodontogenic Toothache

This chapter provides information that will help the dental clinician to identify toothaches with a nonodontogenic etiology. The clinician must have a thorough knowledge of all possible causes of orofacial pain, which includes both odontogenic and nonodontogenic conditions. This knowledge prevents misdiagnosis and allows for proper treatment selection and referral if necessary. For information about treatment of these disorders, other references should be used.

Consensus on the exact taxonomy with diagnostic criteria and their interrelationships among various orofacial pain disorders has not been established. Various health care professions involved in the diagnosis and treatment of such pains have used different terms in the literature. This, of course, can and has led to confusion, especially within what we refer to as neuropathic pain. The terms used in the literature are diverse, and they overlap in meaning to an unknown degree; for example, *phantom tooth pain* and *atypical odontalgia* are used interchangeably. At other times the literature uses the same terms to describe seemingly different disorders; for example, trigeminal neuralgia has the connotation of an idiopathic pain disorder characterized either as intense, intermittent lightning bolt–type pain within one or more distributions of the trigeminal nerve, or as continuous pain that is often mild to moderate in intensity that arises in association with injury to a specific branch of the trigeminal nerve. Efforts have resulted in a working diagnostic framework for neuropathic pains.[135] Our classification scheme uses this framework to enhance the clarity of communication and follows the American Academy of Orofacial Pain's guidelines for assessment, diagnosis, and management for orofacial pain,[30] even though the application of these criteria to pains that present in the orofacial region is known to be associated with misclassification.[35]

Overall, one can classify the nonodontogenic reasons for toothache into five broad groups of pain disorders:

1. Musculoskeletal and other nonprogressive pains arising from somatic structures
2. Neurovascular pain, otherwise known as headache disorders
3. Neuropathic pains
4. Pain of purely psychologic origin, otherwise known as psychogenic toothache
5. Pain associated with a pathologic process

Musculoskeletal and Somatic Pain

Myofascial Pain

Although any deep somatic tissue type in the head and neck has the propensity to induce central excitatory effects and therefore cause referral of pains to teeth, pains of muscular origin appear to be the most common.[46] Myofascial pain (MFP) emanates from small foci of hyperexcitable muscle tissue. Clinically these areas feel like taut bands or knots and are termed *trigger points*.[134] Typically the pain is described as a diffuse, constant, dull, aching sensation; this may lead the clinician to a misdiagnosis of pulpal pain. Another potentially misleading characteristic of masticatory muscle pain is that patients may report pain when chewing. This feature is similar to pain that is periradicular, not pulpal, in origin. On further investigation, it should become clear that the pain is triggered by contraction of masticatory muscles rather than loading of periodontal ligaments. Palpation of the muscles of mastication should reproduce the pain, whereas percussion of the teeth should not. The intensity of the pain will increase and can be perceived in a distant site. Myofascial pain that is perceived to emanate from a tooth is a referred type of heterotopic pain—that is, the pain is felt in an area other than the nerve branch that innervates the trigger point. Typically muscles that refer pain to teeth are the masseter, temporalis, and lateral pterygoid; muscles of the neck and nonmuscular deep structures of the face can also be a source for this type of pain.[134,142]

Although the definitive pathogenesis of MFP is unknown, authors have theorized that muscles may become disturbed through injury or sustained contraction such as clenching.[45,105] Clinically this muscular contraction might occur as a parafunctional habit or as a protective response by localized muscle to an ongoing deep noxious input such as dental pain. Considering this theory and what is witnessed clinically, trigger points appear to be induced or aggravated by toothache. It also appears that trigger points can persist after the toothache has been resolved. This can be confusing for the clinician and frustrating for the patient. It is important to realize the relationship of these two entities. MFP can mimic toothache, and toothaches may induce the development of MFP.

Toothaches of myofascial origin may arise with or without evidence of pulpal or periapical pathosis. Definitive diagnosis is based on lack of symptoms after pulp testing and percussion or palpation sensitivity, or failure to resolve symptoms with local anesthetic blockade. In contrast, jaw function and palpation of the masticatory muscle(s) elicit toothaches of myofascial origin. Typically, local anesthetic infiltration into the trigger point(s) will resolve symptoms.

Common therapeutic modalities used to treat myofascial pain include deep massage, relaxation techniques, "spray and stretch," muscle relaxants, and trigger point injections. Deep massage and relaxation techniques have the advantage of being noninvasive and easily administered. Spray and stretch involves an application of a vapor coolant spray to the skin overlying the trigger point, followed by a gentle stretching of the muscle. Trigger point injections are used for both the diagnosis and treatment of myofascial pain. Specifically, if the pain complaint is diminished on injection of the trigger point(s), then the source of the pain has been confirmed. The therapeutic efficacy of a trigger point injection varies. Some patients might experience lasting relief with one injection or several, whereas others may not. See the Additional Tests section for further information about trigger point injections.

Pain of Sinus or Nasal Mucosal Origin

Sinus/nasal mucosal pain is another source of pain that can mimic toothache.[1,2,28,138] Sinus pain can exhibit symptoms of fullness or pressure below the eyes but is generally not particularly painful unless the nasal mucosa is also affected.[37] Pain from the nasal mucosa tends to be dull and aching and can also have a burning quality typical of visceral mucosal pain. In general, these pains are of viral, bacterial, or allergic etiology. Other symptoms consistent with these types of disease (e.g., congestion or nasal drainage) should be noted in the patient history.

Typical of deep visceral-like tissues, sinus/nasal mucosal pain can induce central excitatory effects such as secondary hyperalgesia, referral of pain, and autonomic changes. It is this tendency that gives sinus/nasal pain the ability to masquerade as toothache. Secondary hyperalgesia, seen clinically as a concentric spread of pain beyond the area of tissue injury, results in tenderness of the mucosa in the area of the maxillary sinuses as well as tenderness to percussion of several maxillary teeth. Teeth tender to percussion and palpation suggest periradicular inflammation. Autonomic sequelae might present as edema or erythema in the area, which could suggest a dental abscess. However, when an etiology for pulpal and therefore periradicular pathosis is absent, sinus/nasal mucosal disease should be suspected. The three cardinal symptoms of acute rhinosinusitis are (1) purulent nasal discharge, (2) nasal obstruction, and (3) facial pain-pressure-fullness.[118] Other symptoms of sinus disease include sensitivity to palpation of structures overlying sinuses (i.e., paranasal tenderness) and a throbbing or increased pain sensation when the head is placed lower than the heart. Dental local anesthetic blockade will not abate sinus/nasal mucosal pain, although topical nasal anesthetic will.

Patients with suspected sinus/nasal mucosal disease should be referred to an otolaryngologist for further diagnosis and treatment. Physical examination as well as adjunctive tests may be necessary for a definitive diagnosis. Tests may include nasal cytologic and ultrasound studies and the use of nasal endoscopes, in addition to imaging tests such as radiology and computed tomographic imaging.[36,125] Treatment of sinus/nasal mucosal pain is dependent on the etiology (e.g., bacterial, viral, allergic, or obstructive).

Salivary Gland Pain

Pain referred from one or more of the salivary glands may be perceived as tooth pain; the authors have not encountered this response in clinical practice, but it has been reported to present as a nonodontogenic toothache.[80,115] Because the primary somatosensory innervation of the major salivary glands comes from the mandibular branch, it is conceivable that such a presentation will occur most often in mandibular teeth.

Neurovascular Pain

Neurovascular pains, otherwise and interchangeably referred to as headache disorders, have qualities similar to pulpal pain. These types of pain can be intense, often pulsatile, and are known to occur only in the head. The International Headache Society (Oxford, UK) has developed a classification system that is widely accepted even though validation studies of these criteria have yet to be published. The interested reader should consult the classification system for more details on this topic.[56] Primary neurovascular pain disorders are thought to be a referred pain phenomenon, meaning that intracranial branches of the trigeminal nerve become sensitized via incompletely understood mechanisms and the associated pain and symptoms are perceived in the somatic structures of the head. Most commonly people report pain presenting in the forehead, back of the head, and temples but also in the sinuses, jaws, and teeth.

The current understanding of the pathophysiology of headaches implies that dental disease and treatments are not likely to be a cause of a person developing a headache disorder, but rather, because the same neuroanatomic circuitry is involved, these aspects of dentistry can be thought of as an inciting event, similar to the analogy that exercise producing increased demands on the cardiovascular system can be an inciting event for an acute myocardial infarction. For this reason, dental clinicians should be aware of the diagnostic status of their patients, because patients with headache disorders are likely to experience more peritreatment pain complications that are related to the innate hyperexcitability of the trigeminal nervous system in these people.

Of most interest to the dental clinician are the primary headache disorders, which make up the bulk of the headache disorders that occur within the population and have been reported to present as nonodontogenic toothache. To simplify thinking, these primary headache disorders can be grouped into three major subdivisions: (1) migraine, (2) tension-type headache, and (3) cluster headache and other trigeminal autonomic cephalalgias (TACs).

Migraine is a common headache experienced by about 18% of females and 6% of males.[82,128] It is associated with significant amounts of disability, which is the motivating factor that brings the patient to seek care and the reason why this type of headache is the one most often seen in medical clinics.[131] Migraine has been reported to present as toothache[4,26,34,52,96,103] and is likely the most common neurovascular disorder to do so. In addition, people with migraine headaches are thought of as having increased regional pain sensitivity that has diagnostic and treatment implications for the clinician.[102]

Migraine headaches typically last between 4 and 72 hours. They tend to be unilateral in presentation and pulsatile in quality, with a moderate to severe intensity to the pain. Patients may also experience nausea or vomiting, as well as photophobia or phonophobia, which are different from toothache. The headache is usually aggravated with routine physical activity, such as walking up stairs. Caffeine/ergotamine compounds have been used widely in the past as abortive agents for migraine headaches, but in contemporary times they have been replaced with triptans, such as sumatriptan and rizatriptan.[93] Of note, migraine headaches may partially or fully abate with the use of nonsteroidal anti-inflammatory medications in a similar fashion as toothaches.

Tension-type headache is the most frequent headache disorder experienced, with a wide range of reported prevalence (41% to 96%).[117,123] The concept of tension-type headache pain presenting as toothache has not been reported in the literature to our knowledge, likely because the construct of what a tension-type headache is has not been clearly defined. Some research supports the notion that a tension-type headache has a significant musculoskeletal component to the pain,[129] whereas other research suggests otherwise. Tension-type headaches are likely a heterogeneous group of similarly presenting head pains that have overlapping pathophysiologic mechanisms, which has led some researchers to consider aspects of tension-type headache to be the same as musculoskeletal orofacial pain, otherwise known as temporomandibular disorders (TMDs).[55] This has further been supported by data from a TMD validation study to derive criteria for such headaches that are of TMD origin.[6,122]

Cluster headaches and other TACs are rare neurovascular disorders that are strictly unilateral pains defined by the concurrent presentation of at least one ipsilateral autonomic symptom—such as nasal congestion, rhinorrhea, lacrimation, eyelid edema, periorbital swelling, facial erythema, ptosis, or miosis—that occurs with the pain. The major distinguishing features between these headache disorders are the duration and frequency of the pain episodes, as well as the gender most often afflicted. Cluster headache is the most common of the group, occurring in men three to four times more often than in women, with pain episodes lasting between 15 minutes and 2 hours that occur at a frequency of eight episodes per day to one every other day. These headaches come in clusters, with active periods of 2 weeks to 3 months,[56] thus the name. Elimination of pain after 10 minutes with inhalation of 100% oxygen is diagnostic for cluster headache,[49] whereas sublingual ergotamine and sumatriptan are also effective acute treatments for cluster headache.[42] Paroxysmal hemicrania, which has a 3:1 female predilection, presents with characteristics similar to those of cluster headache but with a frequency of more than five per day and a duration lasting 2 to 30 minutes.[56] This headache disorder has a 100% response to indomethacin but is refractory to other treatments,[65] thus underscoring the need for obtaining an accurate diagnosis from an experienced clinician.

From a nonodontogenic perspective, cluster headache[4,14,21,51] and almost all the other TACs have been reported in the literature to present as nonodontogenic toothache.[4,11,12,31,92,110,120] The concurrent autonomic features, such as discoloration or swelling in the anterior maxilla, might compound the diagnostic problem by suggesting tooth abscess. It is important to note that neurovascular headaches tend to be episodic with complete remission between episodes, whereas toothache pain usually has at least some background pain that stays between any exacerbations. Provocation of the tooth should not result in a clear increase in pain but cause a slight alteration because this tissue has become hypersensitized. Local anesthetic is unpredictable in these cases and can mislead the clinician. Management by the typical clinician is to determine that the pain is not of odontogenic origin and then to refer the patient to an appropriate care provider. Other neurovascular disorders not classified as primary headaches have been reported to present as nonodontogenic toothache, such as cough headache.[91] One would not expect a dental clinician who does not have a specific focus on orofacial pain to arrive at such a specific diagnosis but rather to be aware of and sensitive to the fact that more obscure headache disorders exist and should be considered in the differential diagnosis of a nonodontogenic toothache that is not easily classified.

Neuropathic Pain

All previously described pain entities can be classified as somatic pain. That is, they are a result of noxious stimulation of somatic structures. These impulses are transmitted by normal neural structures, and their clinical characteristics are related to stimulation of normal neural structures. Neuropathic pain actually arises from abnormalities in the neural structures themselves, specifically the somatosensory system. The clinical examination generally reveals no somatic tissue damage, and the response to stimulation of the tissue is disproportionate to the stimulus. For this reason, neuropathic pains can be misdiagnosed as psychogenic pain simply because a local cause cannot be readily identified. There are many ways to categorize neuropathic pain in the orofacial region. For the purposes of this chapter and ease of discussion, neuropathic pain is divided into four subcategories: neuralgia, neuroma, neuritis, and neuropathy. It should be acknowledged that these subcategories are arbitrary and are not mutually exclusive.

Neuralgia

As alluded to previously, not all uses of the term *neuralgia* refer to what is often thought of as the classic trigeminal neuralgia or tic douloureux. Sometimes the term *neuralgia* is used to describe pain felt along a specific peripheral nerve distribution, such as with postherpetic neuralgia and occipital neuralgia, as opposed to a focus of pain disorders that have similar characteristics and are thought to have common underlying pathophysiologic mechanisms. When used in the generic sense to describe pains that present intraorally, the term can lead to a great deal of confusion.

Although deviations are not uncommon, trigeminal neuralgia is characteristically an intense, sharp shooting pain that is most often unilateral. Ipsilateral to the perceived location of the symptoms is an area that, on stimulation such as light touch, elicits sharp shooting pain. The area that elicits the pain is referred to as a trigger zone, and it can be in the distribution of the resultant pain or in a different distribution—but is always ipsilateral. Although most patients present with a characteristic trigger zone, not all patients will present with this finding. An important characteristic of trigger zones is that the response to the stimulus is not proportional to the intensity of the stimulus—that is, slight pressure on a trigger zone results in severe pain. In addition, once triggered, pain typically subsides within a few minutes until triggered again. This is in

contrast to odontogenic pain, which may come and go but does not do so in such a predictable and repeatable manner. Finally, the trigger for odontogenic pain is an area that has no sensory abnormalities (e.g., dysesthesia or paresthesia).

Trigger zones for trigeminal neuralgia tend to be related to areas of dense somatosensory innervation, such as the lips and teeth. For this reason, triggers that elicit this type of pain may include chewing and may lead both the patient and clinician to think of a diagnosis of odontogenic pain. In addition, because the trigger involves peripheral input, anesthetizing the trigger zone may diminish symptoms. This can be very misleading to the clinician if the assumption is that local anesthetic blocks only odontogenic pain.

Because symptoms can be quite severe, patients may consent to or even insist on treatment even though the clinical findings do not definitively support an odontogenic etiology. The possibly misleading symptoms, along with the willingness of the patient to consent to what may seem to be desperate measures, emphasize the importance of a thorough history and clinical evaluation. The absence of a dental etiology for the symptoms (e.g., large restorations, dental trauma, or recent dental treatment) in the presence of the characteristic sharp shooting pain should alert the clinician to consider trigeminal neuralgia in the differential diagnosis. In general, these individuals should be referred to a neurologist or orofacial pain/oral medicine clinician for a complete diagnostic workup and treatment, because case series have suggested 15% to 30% of patients have secondary reasons for their pain,[58,143] such as brain tumors and multiple sclerosis.

Trigeminal neuralgia typically presents in individuals older than 50 years of age. The etiology is thought to be irritation/compression of the root of the trigeminal nerve, prior to the gasserian ganglion, possibly as a result of carotid artery pressure. Individuals with multiple sclerosis develop trigeminal neuralgia more frequently than the general population. For this reason, a person younger than 40 years of age who develops trigeminal neuralgia should also be screened for multiple sclerosis[147] or other intracranial pathosis.[58]

The two general treatment options for trigeminal neuralgia are pharmacologic and surgical procedures. Because of the possible complications associated with surgery, this form of treatment is usually considered only after attempting pharmacologic therapies. Several medications, including carbamazepine, baclofen, gabapentin, and more recently pregabalin and oxcarbazepine, have been used to treat trigeminal neuralgia. Drugs aimed at relieving nociception, such as nonsteroidal antiinflammatory agents, have no significant benefit in these patients, nor do opioid-based analgesics. Clinical trials support carbamazepine as a first-line drug for treating trigeminal neuralgia.[8] In patients who experience pain relief from carbamazepine, the effect is usually rapid; most will report a decrease in severity of symptoms within the first couple of days.

What is thought to be a variation of trigeminal neuralgia, and may also mimic toothache, is pretrigeminal neuralgia. Pretrigeminal neuralgia, as the name suggests, has been described as symptoms that are different from those of classic trigeminal neuralgia but that respond to pharmacotherapy like classic trigeminal neuralgia and, over time (usually weeks to 3 years), take on the classic characteristics of trigeminal neuralgia. The definitive features include the presence of a dull aching or burning pain that is less paroxysmal in nature but still triggered by a light touch within the orofacial region, with variable periods of remission.[48] The subsequent onset of true neuralgic pain may be sudden or may appear several years later,[105] which emphasizes the need for long-term follow-up of these patients to obtain an accurate final diagnosis.

Neuroma

The term *neuroma* has been around for many years and is often overused in an attempt to describe other types of neuropathic pain. A traumatic neuroma, also known as an amputation neuroma, is a proliferative mass of disorganized neural tissue at the site of a traumatically or surgically transected nerve. A part of the diagnosis, therefore, is confirmation of a significant event that would account for the damage to the nerve. Symptoms will not develop until the neural tissue on the proximal stump has had time to proliferate, typically about 10 days after the event. Tapping over the area of a neuroma elicits volleys of sharp electrical pain (i.e., Tinel sign) similar to trigeminal neuralgia. In contrast to trigeminal neuralgia, there should be a zone of anesthesia peripheral to the area of the neuroma[111] that can be identified by checking for loss of pinprick sensibility, such as with the use of an explorer.

Treatment of a neuroma involves pharmacologic management, often via local measures, and may involve surgical coaptation of the nerve with prognosis being variable and dependent on adequate distal nerve tissue and the time interval between injury and reconstruction.[148] Therefore, early recognition and referral are of key importance to prevent significant distal nerve degeneration.[76] Although neuromas most commonly develop in the area of the mental foramen, lower lip, and tongue, there is some evidence that they can also form in extraction sites and after pulpal extirpation. Neuromas were found to form in extraction sites between 4 and 6 months after removal of the tooth in an experimental animal model.[69] Although not all neuromas that form are painful, this could be a potential explanation for ongoing pain in extraction sites after healing has appeared to occur.[111] It is interesting to ponder the possibility of neuroma formation in deafferentation injuries such as pulpectomy and the implications this might have on continued PDL sensitivity after adequate root canal treatment. For treatment of neuromas that are not amenable to surgical correction, see the Neuropathy section of this chapter.

Neuritis

Neuritis is a condition caused by inflammation of a nerve or nerves secondary to injury or infection of viral or bacterial etiology. In general, pain from a virally induced neuritis, such as recurrent herpes simplex or herpes zoster, is associated with skin or mucosal lesions (Fig. 17-5). This presentation does not result in much of a diagnostic challenge, but pain can precede the vesicular outbreak by many days or even weeks.[47] Because neuritic disorders are caused by reactivation of a virus that has been dormant in the trigeminal ganglion, they are considered projected pain with distribution within the dermatomes innervated by the affected peripheral nerves. The nerves affected by the virus may solely supply deeper tissues and therefore may not produce any cutaneous lesions. In the absence of skin or mucosal lesions, a viral neuritis can be difficult to diagnose[47,60,67] and should be considered in the differential diagnosis of a patient with a history of primary herpes zoster infection. Bacterial infection of the sinuses or dental abscess can also cause neural inflammation that may result in pain. This pain occurs simultaneously with pain of the infected tissues and usually

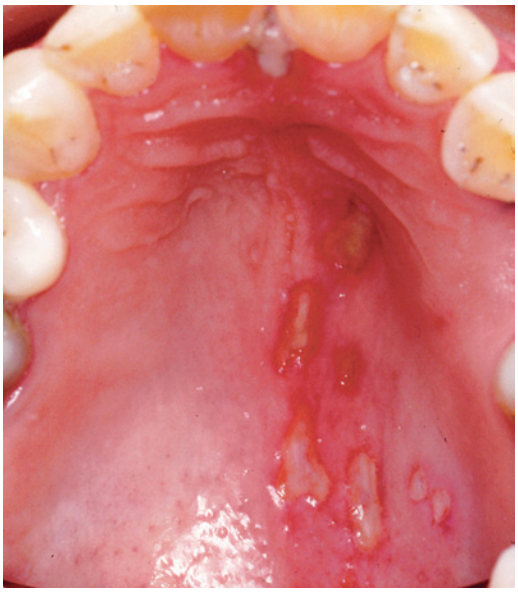


FIG. 17-5 Herpes zoster involving the maxillary division of the left trigeminal nerve of the palate of a 45-year-old male. He complained of a deep, diffuse dull ache of his maxillary left quadrant for 1 week before this vesicular outbreak.

FIG. 17-6 Extrusion of the filling material from the distal canal of tooth #30 of a 36-year-old female. Her complaint was of extreme pain after completion of the root canal treatment followed by sharp, burning continuous pain that could be made worse by a light touch of the tooth.

dissipates once the etiology is addressed. In susceptible individuals, virally or bacterially induced neuritis may produce a postinfection neuropathy of the infected nerve. The pain is fairly constant and can be dull, aching, and burning. Also, the pain may be accompanied by allodynia, a painful response to normally non-noxious stimulation such as light brushing of the skin. Oral acyclovir has become the most common treatment for acute herpetic outbreaks and has been shown to be efficacious in decreasing the duration and severity of pain after herpes zoster infection. Efficacy is based only on administration in the prevesicular, not the vesicular, stage. The addition of prednisolone to acyclovir produces only slight benefits over acyclovir alone. Neither acyclovir alone nor its combination with prednisolone appears to reduce the frequency of postherpetic neuralgia.[140]

Localized traumatic injury can also induce neuritis. This injury can be chemical, thermal, or mechanical in nature. A classic endodontic example of a chemical injury to a nerve is the overextension of a highly neurotoxic paraformaldehyde-containing paste (e.g., Sargenti paste) onto the inferior alveolar nerve canal. Chemical trauma can be due to certain toxic components of the endodontic filling materials (such as eugenol), irrigating solutions (such as sodium hypochlorite), or intracanal medicaments (such as formocresol) (Fig. 17-6).[94] Mechanical compression, in addition to thermal trauma, may be a factor when thermoplasticized material is overextended, using an injectable[50] or carrier-based technique. Mechanical nerve trauma is more commonly associated with oral surgical procedures, such as orthognathic surgery, and third molar extraction.

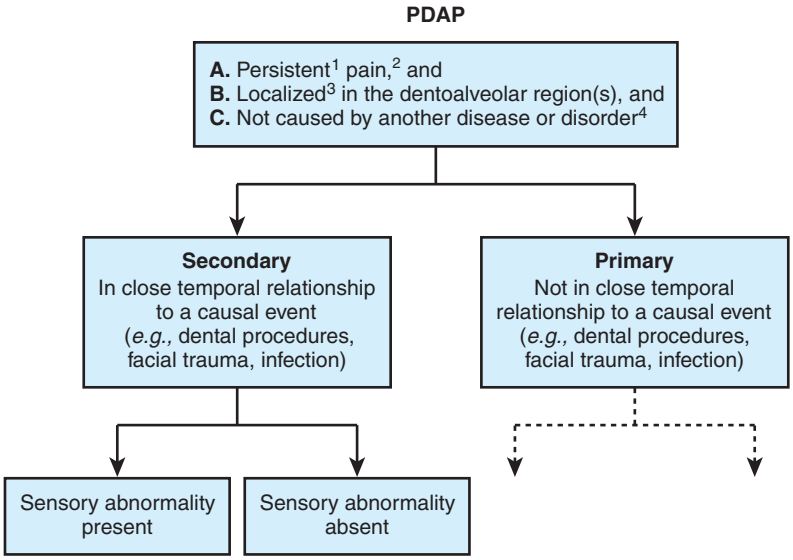
Neuritic complications have also been documented after mandibular implant surgery at a rate of 5% to 15%, with permanent neuropathies, which are discussed later, resulting in approximately 8% of these cases.[66] It is unfortunate that traumatic neuritis is often misdiagnosed as a posttreatment chronic infection and that the area is reentered and debrided. Additional surgical insult further traumatizes the nerve, prolonging the already present nociceptive barrage, which puts the patient at an increased risk of developing central hyperalgesia. Undiagnosed and mistreated cases of acute neuritis not only lead to unnecessary dental procedures but may also aggravate the neuritis and, therefore, the neuritic pain has a greater chance of becoming chronic, something that is often referred to as neuropathic pain.

Neuritic pain typically is a persistent, nonpulsatile burning often associated with sensory aberrations such as paresthesia, dysesthesia, or anesthesia. The pain can vary in intensity, but when stimulated, the pain provoked is disproportionate to the stimulus.

Treatment of acute neuritis is based on its etiology. In instances of chemical trauma (e.g., Sargenti paste) where an obvious irritant is present, surgical debridement of the nerve to remove any substance that can continue to irritate the nerve is an important aspect of treatment. With neuritis secondary to mechanical compression (e.g., implant placement) of a nerve, nerve decompression by removal of the implant fixture is indicated. Such localized, acute, traumatically induced neuritis is inflammatory in nature and, therefore, can also benefit from supportive pharmacotherapies such as steroids. For management of neuritis that is not responsive to the previously cited treatments, medications used to treat neuropathic pain may be used (see Neuropathy). For neuritis occurring secondary to an infection, such as one of odontogenic or viral etiology, treatment is directed at eliminating the offending pathogen and minimizing injury to the afferent nerves.

Neuropathy

In this chapter we use the word *neuropathy* as the preferred term for localized, sustained nonepisodic pain secondary to an injury or change in a neural structure. Historically, other terms have been used including *atypical facial pain*. This term suggests pain that is felt in a branch of the trigeminal nerve and that does not fit any other pain category. Pain of an unknown source that is perceived in a tooth may be labeled *atypical odontalgia*. Pain that persists after the tooth has been extracted



Criteria
[1] Persistent, meaning pain present at least 8 hours/day≥15 days or more per month for ≥3 months
[2] Pain is defined as per IASP criteria (includes dysesthesia)
[3] Localized, meaning the maximum pain defined within an anatomic area
[4] Extent of evaluation non-specified (dental, neurologic exam ± imaging, such as intraoral, CT and/or MRI)

FIG. 17-7 Diagnostic criteria for persistent dentoalveolar pain disorder (PDAP).

is referred to as *phantom tooth pain*. The major limitation in the use of all these terms is that they merely suggest an area where a pain of unknown etiology exists and completely lack any information regarding the pathophysiology. Although each of these terms has been extensively described in the literature,[88,89] probably none actually represents one discrete condition but rather a collection of various conditions. Based on these thoughts, a consensus process resulted in proposing new terminology, persistent dentoalveolar pain disorder (PDAP), and diagnostic criteria (Fig. 17-7).[99]

Once a nerve has been sensitized via injury or disease it may remain so and present as a peripherally sensitized nerve. This peripheral sensitization and the ongoing pain (nociceptive barrage) that accompanies it can induce changes in the central nervous system. *Peripheral* sensitization and *central* sensitization can potentially impact the clinical presentation of a neuropathy. A typical clinical course of someone with an undiagnosed neuropathy might consist of treatment for a toothache. When the pain does not resolve with nonsurgical root canal treatment, it might then be followed by apical surgery and then perhaps an extraction. The extraction site might then be explored and debrided in a misguided attempt to remove any potential source of the patient's ongoing pain. After each treatment, there tends to be a reduction of the pain for a short time and then a return to its original, or even increased, level of pain intensity. It is likely that this is a result of a new neural injury consisting of reorganization and resprouting that increases the inhibition of nerve firing for a time. Surgical approaches to neuropathies are not effective because they do not desensitize the nerve. On the contrary, surgical intervention may aggravate the situation by inflicting an additional neural injury in the periphery and contributing to the already present nociceptive input. This intervention therefore puts the patient at increased risk of developing persistent pain, which is supported by a couple of long-term observational studies[3,113] and further supported by the observation that patients with pain following root canal therapy did not uniformly experience elimination of this pain with apicoectomy surgery.[108]

A diagnosis of neuropathy is based primarily on history and examination with the use of selected diagnostic tests to rule out other potential etiologies. The history should reveal some inflammation-inducing event (see the earlier sections Neuritis and Neuroma), although the nature of the initial insult is not always identified, as seemingly spontaneous development of such pains has been reported.[99] Typically, the examination is grossly unremarkable with no evidence of local tissue damage, leaving the clinician to rely mainly on the patient's report of symptoms. Although quality of pain is no longer thought to be capable of distinguishing neuropathic pains from others, patients repeatedly reported several features that may be key for identification purposes (Box 17-2).[40] Regarding examination features, the area where the pain is perceived may be hyperalgesic or allodynic—that is, noxious stimulation to the area will be perceived as more painful or non-noxious stimulation will now be perceived as painful. This phenomenon is documented by reports where standardized applications of stimuli to the affected tissue are performed and demonstrate exaggerated responses.[85,90,146] Besides gain in sensory function, loss of function has also been observed,[85] which is more in line with a general definition for neuropathic pain.[135] Furthermore, maintenance of pain following local anesthesia to the affected region[83] and a lack of pain reduction with fentanyl and ketamine[7] all suggest the role of a central pain-related mechanism.

Research related to the diagnostic imaging of cases that may be PDAP suggests two roles: first, to identify pathosis contributing to the pain presentation and, second, as a means of

BOX 17-2

Recurrent Patient-Reported Themes Regarding Their Persistent Dentoalveolar Pain Disorder (PDAP)

- Difficult for patients to respond to history taking because their words do not adequately describe what they feel; therefore time may be needed to obtain the necessary information
- Well localized to a region within the dentoalveolar structures
- Pain is perceived to be deep in the tissues, rather than on the surface
- Continuous pain, one that never stops and seems to always be there
- Pain has the sensation of feeling pressure with a dull ache quality
- Complex and confounding descriptors, such as itching, tingling, or pricking, are sometimes present

obtaining a positive finding for this enigmatic chronic pain disorder. As for the first role, diagnostic imaging is recommended to assess for odontogenic-related pathosis and other regionally presenting disease, as most pain in the dentoalveolar region is tooth-related. For patients suspected of having PDAP, the diagnostic yield of cone-beam computed tomography (CT) was reported to be superior to PA radiograph, but the findings were of questionable significance.[112] For patients without local pathosis and PDAP, magnetic resonance imaging (MRI) of the brain revealed several cases of intracranial findings thought to be related to the pain presentation (e.g., cysts, tumors, infarcts).[104] This is consistent with the clinical experience of one of the authors, which has led to brain imaging being routine prior to a diagnosis of PDAP being rendered. As for the second role of diagnostic imaging, conventional dental radiographic techniques have consistently been found not to be able to identify patients with PDAP, thus prompting pilot investigation into other imaging techniques. Results suggest high levels of sensitivity and specificity can be reached using thermography[64] revealing a "cold" image profile.[63] On the contrary, results suggest that a technetium-99 bone scan had low sensitivity and specificity for detecting dentoalveolar regions with chronic pain,[32] and MRI techniques do not seem to have been studied.

Neuropathic disorders have a predilection for women but can affect both genders. These patients are usually older than 30 years of age and may have a history of migraine.[126] In the orofacial region, neuropathies are most commonly seen in the maxillary premolar area and molar region.[61,108]

Neuropathies can be classified on the basis of their clinical presentation and response to therapies. *Peripheral neuropathy* may develop after sensitization of a peripheral nerve and presents clinically as described previously. Diagnosis of peripheral neuropathy is based on its favorable response to peripheral neural blockade. Treatment is directed at decreasing the sensitization of peripheral nerves and reducing ectopic neuronal firing. Topical as well as systemic medications can be used to treat cutaneous peripheral neuropathies. Topical medications include topical anesthetics, capsaicin-containing compounds, and anticonvulsants, as well as nonsteroidal anti-inflammatory drugs (NSAIDs), sympathomimetic agents, and *N*-methyl-D-aspartate (NMDA) receptor-blocking agents[109] with encouraging results.[72]

The clinical presentation of a *central neuropathy* is similar to that of a peripheral neuropathy. After sensitization of peripheral nerves and the accompanying nociceptive barrage, the pain is nonremitting and lacks evidence of tissue insult. Unlike its peripheral counterpart, allodynia and secondary hyperalgesia are clearly present—that is, the area of pain is significantly larger than the initial site of injury. The most telling sign that a neuropathy has taken on a more central component is that local anesthetics are no longer effective. Therefore, the treatment must be directed toward the central processing of pain. This is done with medications such as NMDA receptor agonists (ketamine), gabapentin, tricyclic antidepressants, and opioids. The prognosis for a central neuropathy is not as good as for a peripheral neuropathy, as central neuropathic pain tends to become more refractory with time. Treatment is often based on the management of pain, rather than its cure, and sometimes is best performed in a multidisciplinary chronic pain clinic facility.

The last variation of neuropathic pain is sympathetically enhanced or maintained pain. In cases of sympathetically maintained pain (SMP), peripheral nerve fibers upregulate the expression of adrenergic receptors, making them responsive and sensitive to sympathetic input. SMP may also have a central component, whereby the constant sympathetic drive alters neuronal excitability. Neuronal injury may induce sprouting of sympathetic axons into the trigeminal spinal nucleus because basket-like formations of sympathetic fibers have been reported around the cell bodies of sensory neurons in the dorsal root ganglia.[141] Increases in sympathetic drive, such as with stress and fever, may aggravate SMP. Diagnosis of sympathetically maintained pain is based on blocking sympathetic outflow to the affected region via sympathetic nerve blocks. In the orofacial region this would require a stellate ganglion block. The block is considered diagnostic for SMP if it effectively decreases the patient's pain. Multiple blocks can also be used as a form of therapy. Other therapies include drugs that target peripheral α_2-adrenoceptors (agonists) or α_1-adrenoceptors (antagonists), such as guanethidine, phentolamine, and clonidine. SMP presenting in the orofacial region is extremely rare[54] and therefore makes clinicians prone to deriving a falsely positive diagnosis of this condition.[101] Furthermore, researchers have failed to produce SMP-type pain in animals,[10] something that is presumed to be due to the fact that the efferent nerve fibers in the head and neck region run with the blood vessels as opposed to the afferent nerves, as they do elsewhere in the human body. For these reasons, the likelihood of this type of pain presenting as "toothache" is extremely low and thus does not require much discussion here.

Psychogenic Toothache

Psychogenic toothache falls within a group of mental disorders known as somatoform disorders. The name is derived from the fact that the patient has somatic complaints yet lacks a physical cause. Because these patients lack a physical cause for pain, they also present without local tissue changes. Patients with somatoform disorders are not fabricating the symptoms, nor are they consciously seeking benefit. It is important to make a distinction between somatoform disorders and factitious pain or malingering behavior.[5] In factitious pain there are physical or psychological symptoms that are produced by the individual and are under voluntary control. Malingering is similar to factitious pain with the added characteristic that the symptoms are

presented for obvious and recognizable benefit. This category poses a significant diagnostic challenge. Lacking evidence of local tissue damage is typical of heterotopic pain entities previously discussed in this chapter. It is important to emphasize that psychogenic pain is rare. When arriving at this diagnosis it is critical that all other potential diagnoses have been ruled out.

The diagnosis of psychogenic toothache is one of exclusion and is based on the clinician's awareness of other heterotopic pain characteristics and behavior. Of particular note are centrally emanating pains, cardiac pain, neurovascular pain, and neuropathic pain. Adding to the diagnostic difficulty is that comorbid psychological disorders are commonly present with chronic intraoral pain disorders, including those erroneously presenting as toothache.[84,130] This has led to the current thinking that psychological disorders (i.e., depression, anxiety, somatization) may not be related to the initiation or perpetuation of chronic pain disorders but rather are a consequence of living with chronic pain.

Psychogenic pain is known to be precipitated by severe psychologic stress. These pains present a general departure from the characteristics of any other pain condition—that is, they may not fit normal anatomic distributions or physiologically plausible patterns. The pain may be felt in multiple teeth and may jump around from one tooth to another. The intensity of pain reported tends to be more severe than is reflected by the patient's level of concern about the condition. Patients' responses to therapy are variable and include a lack of response or an unusual or unexpected response. Early identification of psychogenic pain and referral to a psychologist or psychiatrist is necessary to avoid irreversible and unnecessary dental treatment.

Toothache Referred from a Distant Organic Source

A variety of pathologies that seem to be unrelated have been reported to present as nonodontogenic toothache.[107,115] The only common link that can be identified is that branches of cranial nerves innervate the involved tissues, and hence the trigeminal nucleus processes nociceptive input. Therefore, conceivably, any somatic structure with cranial nerve innervation has the potential to cause pain that the patient perceives as a toothache. For this reason, once dentoalveolar etiologies for such pain have been ruled out, all possible sources of nonodontogenic pain including distant pathology should be considered in the differential diagnosis. Several of these types of organic pathologies that have been reported to present as toothache are described in the following sections.

Cardiac and Thoracic Structures

Cardiac pain has been cited as the cause of nonodontogenic toothache in a number of case reports.[9,41,62,77,97,136] Classically, cardiac pain presents as a crushing substernal pain that most commonly radiates to the left arm, shoulder, neck, and face. Although not as common, anginal pain may present solely as dental pain, generally felt in the lower left jaw.[16] Similar to pain of pulpal origin, cardiac pain can be spontaneous and diffuse, with a cyclic pattern that fluctuates in intensity from mild to severe. The pain can also be intermittent and the patient may be completely asymptomatic at times. The quality of cardiac pain when referred to the mandible is chiefly aching and sometimes pulsatile. Cardiac pain may be spontaneous or increased with physical exertion, emotional upset, or even the ingestion of food.[9] Cardiac pain cannot be aggravated by local provocation of teeth. Anesthetizing the lower jaw or providing dental treatment will not reduce the pain. It can be decreased with rest or a dose of sublingual nitroglycerin. Diagnosis of cardiac pain, along with immediate referral, is mandatory to avoid impending myocardial infarction.

Besides pain of cardiac origin, other chest structures have been reported to produce nonodontogenic toothache pain. Various cancerous lesions of the lungs have been described to present a mandibular pain, with the pain being both ipsilateral and contralateral to the side where the tumor is present.[24,59] Furthermore, diaphragmatic pain is mediated via the phrenic nerve and may present as nonodontogenic tooth pain.[15]

Intracranial Structures

Space-occupying lesions within and around the brain are known to impinge on structures innervated with somatosensory fibers, such as the dural and perivascular tissues, causing pain. These pains are highly variable, with a common complaint being headache or head pain. Just as intracranially derived pain may be referred to the face and jaws in neurovascular disorders, it may also present as a toothache.[137] To outline the vast differences in clinical features of such pain, intracranial lesions have also been reported to cause trigeminal neuralgic pain in response to treatment of what was first thought to be toothache.[29] This extreme variability has been observed by one of the authors, which leads to the recommendation that if local etiologic factors are not readily identified in a patient with toothache symptoms, magnetic resonance brain imaging should be considered.

Throat and Neck Structures

Nonodontogenic toothache has been reported to arise from various structures of the neck, but these reports are sparse and hence it is not possible to draw conclusions regarding how patients with these pain-provoking disorders may present. Squamous cell carcinoma of the lateral pharyngeal surface presenting as ipsilateral mandibular molar pain has been observed by one of the authors. This finding is consistent with previous reports of nonodontogenic pain being associated with smooth muscle tumors of a similar location.[139] Vascular structures of the neck have also been implicated in the production of toothache symptoms, with a report of a patient initially presenting for dental care when pain was from the result of a life-threatening carotid artery dissection.[119]

Craniofacial Structures

Clinically, pain from other craniofacial structures has been observed as being the most common reason for organic pathologies presenting as nonodontogenic toothache, likely because these structures are innervated by branches of the trigeminal nerve. Tumors in the maxillary sinus[27,43,145] and jaw,[132] as well as metastatic disease, particularly within the mandible,[33,53,114,124] have been reported. The clinical presentation of symptoms is highly variable, but a common feature is sensory loss along the distribution of the nerve, the result of pain arising from nerve impingement. This underscores the need for regional imaging techniques, such as pantomography or computed tomography (CT) (as opposed to just periapical radiographs). This is especially true in patients who have a history of cancer. One must also not forget that nerve impingement anywhere along the distribution of the trigeminal nerve,

even within the cranial vault itself,[20] can elicit nonodontogenic tooth pain.

Vascular structures within the craniofacial region have also been reported to present as nonodontogenic toothache, with arteritis being the pain-provoking pathology.[68,73] These pains have been described as a continuous dull pain that can sometimes be made worse with jaw function. The stereotypical presentation includes a history of eyesight changes, such as blurred vision, and the examination feature of pulseless, indurated temporal arteries that are painful to palpation. A laboratory finding of an elevated erythrocyte sedimentation rate (ESR) is suggestive of the disorder, and diagnosis is confirmed by temporal artery biopsy. Treatment includes administration of corticosteroids; therefore, because permanent blindness is a possible sequela if cranial arteries are left unmanaged, immediate referral to the appropriate medical colleague is indicated.

Frequency of Nonodontogenic Toothache

The prevalence of nonodontogenic toothache in the population is unknown, as is the prevalence of such a presentation for dental care. Specific to endodontics, pain is thought to be present in 5.3% of patients 6 or more months following treatment,[100] with about half of those individuals being estimated to have nonodontogenic etiologies accounting for their complaint of that pain.[100] At present, the proportion of such patients having the aforementioned nonodontogenic pain diagnosis is unknown. It remains unclear how this nonodontogenic pain is related to the endodontic diagnosis and subsequent treatment.

TAKING A PATIENT'S HISTORY

Pain diagnosis is largely based on the patient's subjective history; however, patients rarely give all pertinent diagnostic information about their pain of their own accord. Often it is necessary to carefully extract the details of the patient's pain complaint through systematic and thorough questioning. This is known as "taking a history," and it involves both careful listening and astute questioning. Fig. 17-8 is an example of a basic diagnostic workup for odontogenic pain. It can be easily used to obtain histories of typical odontogenic pain by circling all descriptors that apply and then filling in the remaining blanks. As the details of a patient's pain complaint are gathered, the clinician should be mentally progressing through an algorithm of possible diagnoses, as each detail should lend itself to one type of pain over another. After completing a thorough and accurate history of the complaint(s) (Fig. 17-9), often the diagnosis has already been narrowed down to one particular pain entity. This is particularly true with odontogenic pain. The only question that will remain is "which tooth is it?" It is critical to keep in mind that whereas patients will provide information about the perceived site of pain, it is the clinician's examination that will reveal the true source of their pain. With more complicated pain complaints, the clinician may have a list of possible diagnoses. This is known as a differential diagnosis. This differential will guide the examination and testing in an effort to confirm one diagnosis while ruling out all others. If after completing the subjective examination all items on the differential are outside the clinician's scope of practice, then the clinician should continue the examination until he or she has a firm idea of the possible diagnosis so that a proper referral can be made. In addition, it is paramount that all odontogenic sources have been ruled out and that this information is communicated to the health care provider to which the patient is referred. If no differential can be formulated after the history has been

Sufficient for pain of odontogenic origin.

Subjective

Pain: (Circle all appropriate) Level (0-10) _____

Well-localized	Diffuse	Intermittent
Spontaneous	Elicited (cold, hot, chewing)	Throbbing
Constant	Fluctuant	
Dull ache	Sharp shooting	

Onset _____

Progression (F/I/D) _____

Aggravating factors _____

Relieving factors _____

FIG. 17-8 Example of a form to evaluate odontogenic pain.

Chief complaint
Prioritize complaints _____
Specify location _____
VAS 0-10 _____
Initial onset
When did you first notice this? _____
Progression
Frequency _____
Intensity _____
Duration _____
Previous similar complaints
Have you ever had this type of pain before? _____
Characterize complaint
Daily, not daily _____
Constant, fluctuant, intermittent _____
Duration _____
Temporal pattern _____
Quality _____
Aggravating factors
What makes this pain worse? Be specific! _____
Alleviating factors
What makes this pain better? _____
How much better? _____
Associated factors
Swelling _____
Discoloration _____
Numbness _____
Relationship to other complaints
Would your jaw hurt if your tooth didn't hurt? _____
Prior consults/treatment
Who? _____
When? _____
What was the diagnosis? _____
What was done? _____
How did it affect the pain? _____

FIG. 17-9 Example of a form to evaluate pain history.

taken, then the history should be redirected to the patient to confirm that the information is complete and accurate. If the patient is unable to provide sufficient information regarding the pain complaint, then it may be helpful to have the patient keep a pain history, detailing the aspects of the pain on a daily basis. Of utmost importance is to avoid treatment when the diagnosis is uncertain. Diagnostic therapy (i.e., "let's do a root canal treatment and see if it helps") may result in costly treatment that does not improve the patient's condition and could be a factor in aggravating and perpetuating a patient's pain. Treatment should always specifically address a diagnosis.

A complete medical history along with current medications and drug allergies should always be ascertained. It is also important to make note of demographic information, as patients of certain genders and ages are more at risk for some disorders compared with others.

Recording a patient's *chief complaint* in the patient's own words is a medical legal necessity but falls short of constituting a thorough pain history. A complete history begins with a patient's general pain complaint—for example, "My tooth hurts." Patients may have more than one pain complaint—for example, "My tooth hurts and it is starting to make my jaw hurt." All pain complaints should be noted and investigated separately. Understanding the specific components of the complaints makes it possible to discern the relationship between them—that is, either the complaints are wholly separate and two types of pathosis are present, or one source of pain is merely creating a heterotopic pain that is wholly secondary to the first.

Begin with determining the *location* at which the patient perceives the pain. Aspects of the location involve localization and migration. Pain should be definable as either well localized or diffuse and either superficial or deep. Easily localized superficial pain tends to be cutaneous or neurogenic. Musculoskeletal pain is felt deeply and is more localizable once it is provoked. Deep, diffuse pain is suggestive of deep somatic pain, be it visceral or musculoskeletal. Both tissue types are involved in a high degree of nociceptor convergence in the trigeminal nucleus and, therefore, much more likely to be involved in creating heterotopic pain. Typical referral patterns of deep somatic pain tend to follow peripheral dermatomes that reflect the laminations in the trigeminal nucleus. Referred pain also tends to occur in a cephalad direction. Therefore, referred pain from a deep somatic tissue such as tooth pulp, cardiac tissue, or skeletal muscle will respect this pattern. Pain that spreads distally along a nerve branch is much more indicative of a projected type of heterotopic pain. Projected pains imply a neurogenic source and possibly one that is secondary to impingement from intracranial pathosis. Recall that superficial sources of pain are not likely to be involved in referral, so if a patient is indicating that the pain is superficial and spreading, this is highly suggestive of a neurogenic rather than a cutaneous source.

Assessment of the *intensity* of pain is easily accomplished using a verbal analog scale. This question is best phrased, "On a scale of one to ten, zero being no pain and ten being the worst pain you can imagine, how bad is your pain?" Not only can intensity provide insight about pain type; it can also help guide posttreatment pain management as well as provide a baseline for response to therapies.

Identifying the *onset* of pain may provide information regarding etiology. Question if the onset followed a particular event such as a dental appointment or a traumatic injury. Beware of these relationships, as they can be misleading. Having a temporal correlation does not necessarily ensure a cause-and-effect relationship. The onset of pain may be either gradual or sudden. Severe pain of sudden onset can signal a more serious problem. Pain that has been present over a protracted period of time, particularly if the pain has been unchanging, is highly suggestive of a nonodontogenic pain source.

Other *temporal* aspects of pain include frequency and duration. The clinician should ask the patient, "How often does the pain occur and how long does it last?" These temporal aspects may establish patterns that point more clearly to one condition over another.

Progression of the patient's pain over time should be noted. Whether pain is better, worse, or unchanging since its onset should be broken down into three factors: frequency, intensity, and duration. Static pain that does not change over time is typically not odontogenic in origin.

The *quality* of pain—that is, "what it feels like"—is a critical aspect of a pain history. Knowledge of pain characteristics as they relate to tissue types is essential. Pain quality can be difficult for patients to describe, and it is often necessary to provide them with a list of descriptors from which to choose. In instances of odontogenic pain, the list is fairly short. The deep visceral and musculoskeletal components of a tooth limit true odontogenic pain to having qualities that are dull, aching, or throbbing. If there is an aspect of sharpness to the pain, it is helpful to understand whether the sharpness is stabbing in nature, which would indicate A-delta fiber–mediated dentinal pain, or whether it is electrical in nature, which would indicate neuralgia. Common examples of pain descriptors and their respective pain types are listed in Table 17-1.

Factors that precipitate or aggravate the patient's pain complaint are of key importance in diagnosis. Not only do aggravating factors suggest the tissue types that may be involved, but they also aid in directing the objective tests. When gathering information, it is important to be specific. If a patient reports pain while eating, keep in mind that many structures are stimulated during mastication, such as muscles, temporomandibular joints (TMJs), mucosa, PDLs, and, potentially, pulps. Be specific as to the aggravating factor. The lack of any aggravating factors indicates that the pain is not of odontogenic origin.

Alleviating factors can provide insight as to the nature of the pain. If a medication relieves the pain, it is critical to know the specific medication, its dosage, and the degree to which the pain was attenuated. It is equally important to know what has no effect on the intensity of the pain. For example, if a pain of midlevel intensity is completely unresponsive to anti-inflammatory drugs, then it is probably not inflammatory in origin.

TABLE 17-1
Examples of Pain Descriptors

Origin	Quality of Pain
Muscular	Dull, aching
Neurogenic	Shocking, burning
Vascular	Throbbing, pulsatile

Associated factors such as swelling, discoloration, and numbness must be ascertained, as well as their correlation with symptoms. Swelling of acute onset suggests an infection, and its concurrent pain would be of inflammatory origin. Swelling that comes and goes with the intensity of pain suggests an autonomic component. The same can be said for discoloration, such as redness. Numbness or any other type of sensory aberrations should be recorded. If the altered sensation is a major component of the pain complaint, then it should be investigated separately and its relationship to the pain determined. Pains that occur with sensory aberrations tend to have a strong neurogenic component.

If the patient complains of more than one pain, an effort should be made during the subjective history to determine the *relationship of the complaints*. One pain might serve as an aggravating factor to the other. There may be a correlation as to the onset, intensity, or progression of the complaints. Also, keep in mind that patients may actually have more than one type of pathosis occurring concurrently and there may be no relationship whatsoever. Ask whether there have been any *previous similar complaints* and, if so, what happened. Recurrence of similar pains might reveal a pattern that lends itself to a particular pain diagnosis.

It is critical to gain knowledge of any prior consultations that have occurred. Details regarding the type of clinician, actual workup performed, and diagnosis rendered will help to narrow down a differential. Any treatment that was performed should be ascertained along with its effect on the chief complaint.

PATIENT EXAMINATION

As stated previously, the purpose of the history is to gather information about a patient's pain complaint in order to formulate a list of possible diagnoses based on specific pain characteristics. Poor or improper symptom analysis will lead to a false differential, and any testing will therefore have limited meaning. Performing a general examination including an extraoral, intraoral, and hard and soft tissue assessment is requisite to confirm the health of various structures and to identify possible pain-producing etiologies. When a patient presents with a toothache, the pain is usually of odontogenic origin. Diagnostic procedures are often limited to confirming a suspect tooth rather than identifying a nonodontogenic source of pain. Standard pulpal and periradicular tests serve to aid in both ruling in odontogenic pain and therefore ruling out nonodontogenic pain as a diagnosis. Remember that the site of pain is determined by patient history, but the true source of the pain should be revealed with testing. If the chief complaint cannot be reproduced with standard tests then additional tests may be necessary to narrow down a differential diagnosis. For details on general examinations and standard tests, please refer to Chapter 1.

Additional Tests

Further tests should be chosen with forethought in an effort to develop a workable differential that can guide the clinician toward a meaningful consultation or an appropriate referral for the patient. These tests may consist of palpation or provocation of various structures, sensory testing, or diagnostic blocks. The application of these tests is not covered in detail in this chapter. For more information about the application and interpretation of these tests, please consult other sources.

Palpation and percussion are common tests to differentiate odontogenic pain from pain of sinus origin. Palpation of the sinuses consists of firm pressure placed over the involved sinus (usually maxillary). In addition, pain of sinus origin may be provoked with a lowering of the patient's head.

If pain of muscular origin is suspected, then an attempt to reproduce this pain can be done by palpation of the muscles of mastication or provocation via functional manipulation. The temporalis, deep and superficial masseter, medial pterygoid, and digastric muscles should be palpated in an effort to discover tenderness or trigger points that reproduce the pain complaint. The medial pterygoid is only partially accessible to palpation and may need to be functionally tested by stretching the muscle (opening wide) or contracting the muscle (biting firmly). The lateral pterygoid may be difficult if not impossible to palpate intraorally and therefore is more appropriately assessed by functional manipulation. Pain emanating from this muscle may be increased by protruding the jaw against resistance. Exacerbating the chief complaint by muscular function provides a strong indication of a myofascial source of pain.

Because of the complexity of innervation and the occurrence of heterotopic pain in the orofacial region, it may be difficult to definitively determine the origin of pain by testing alone. It cannot be stressed enough that primary pain should not only be provoked by local manipulation but also be relieved by anesthetic blocking. In diagnostic anesthesia the relief of pain has a typical onset and duration, depending on the particular anesthetic used. In addition, the pain should be completely diminished or else suspicion of a central component or a coexisting disorder should arise. The use of diagnostic anesthesia may be necessary and useful in augmenting the diagnostic workup (Fig. 17-10). Topical anesthetic can be helpful in the investigation of cutaneous pain and peripheral neuropathies. Anesthetic injection including peripheral nerve blocks can be used to determine whether the etiology of the pathosis is peripheral to the area of the block. Pain that persists after the onset of usual signs of anesthesia suggests a central component. The patient's history and general examination are of key importance in differentiating between the pain of a central neuropathy and the central pain emanating from an intracranial mass.

Pain that is primarily muscular, as suggested by trigger points discovered on examination, can be further investigated by local anesthetic injection into the trigger point. Trigger point injections are typically performed with either a 27- or a 25-gauge needle and a minimally myotoxic anesthetic such as 2% lidocaine or 3% mepivacaine without a vasoconstrictor. Myofascial trigger point injections may temporarily relieve pain at the trigger point as well as at the site of referral.

Sympathetic efferent activity can play a role in the enhancement or maintenance of chronic pain. In the head and neck, sympathetic activity flows through the stellate ganglion located bilaterally near the first rib. When there is suspicion of a sympathetic component to a patient's pain, a stellate ganglion block can be used to provide diagnostic insight. A trained anesthesiologist usually performs this procedure. An effective block delivered to the stellate ganglion will interrupt sympathetic outflow to the ipsilateral side of the face, resulting in a partial Horner syndrome. This is evidenced by flushing, congestion, lacrimation, miosis of the pupil, ptosis, and anhidrosis.[75] A sympathetic blockade that diminishes or eliminates a pain state may guide future treatment such as repeated blocks or systemic

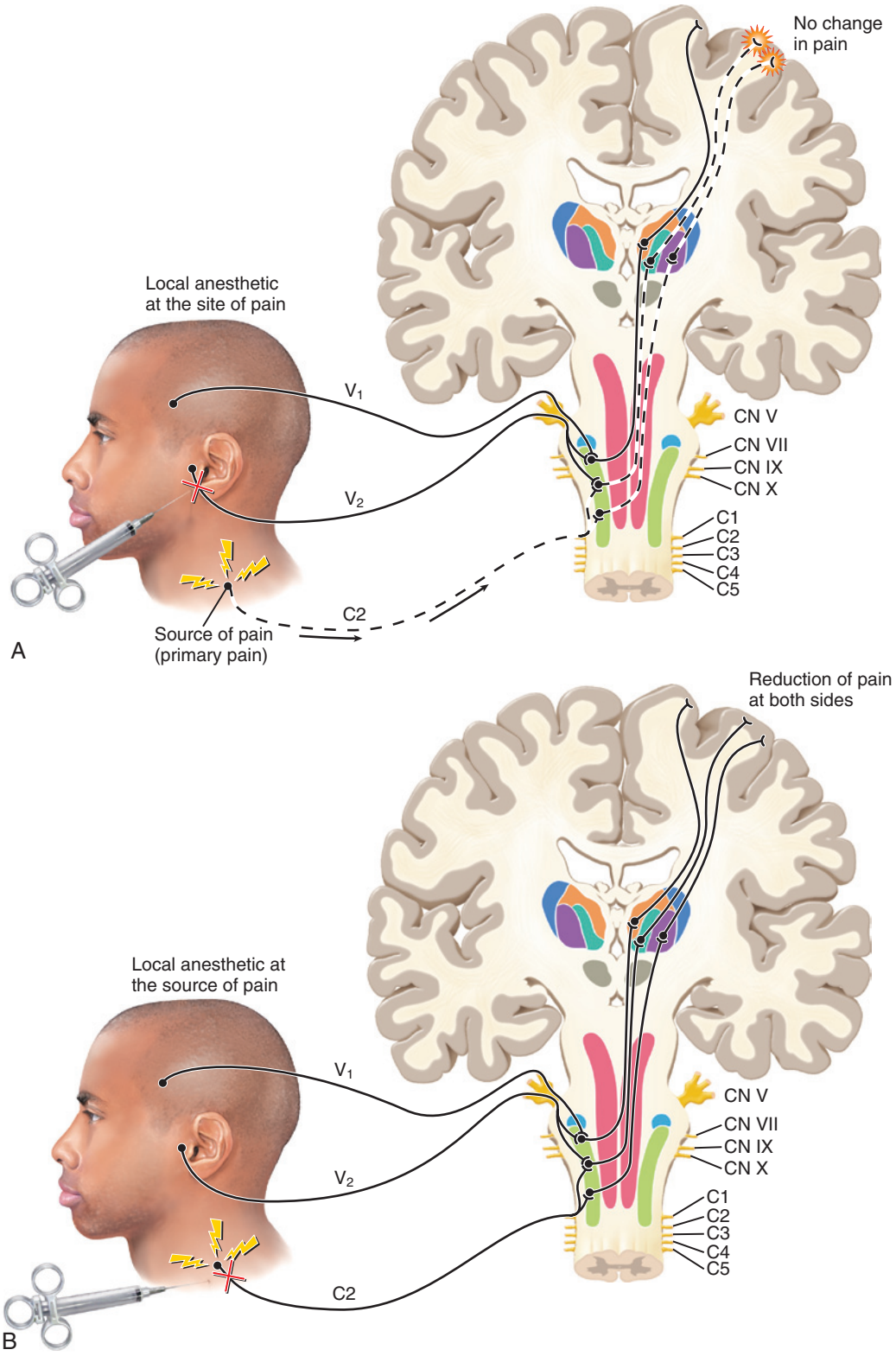


FIG. 17-10 A, Local anesthetic at the site of pain fails to reduce the pain. B, Local anesthetic at the source of pain reduces the pain at the source as well as at the site. (Redrawn from Okeson JP: *Bell's orofacial pains,* ed 5, Chicago, 1995, Quintessence Publishing.)

treatment with sympathetically active drugs (e.g., clonidine and prazosin).[116]

Neurologic conditions, both peripheral and central, can present as pain in the orofacial region. A role of the clinician is to help rule out gross neurologic conditions secondary to intracranial pathosis. Systemic complaints such as nausea, dizziness, or changes in one of the special senses should raise suspicion of intracranial pathosis. A neurologic screening examination including a gross sensory and motor evaluation of cranial nerves II through XII should be performed. For details on cranial nerve examination, please refer to other sources.[44] Investigation of sharp/dull differentiation as well as light touch discrimination between the different branches of the trigeminal nerve can provide insight as to the location and etiology of the pathosis.

Case Studies

Case 1

A 56-year-old male presents with a chief complaint of "This tooth still hurts and it's getting worse. It even hurts when I smile." He has a history of angina secondary to a 70% occlusion of his right coronary artery. He also reports a history of hypercholesterolemia. He has no history of myocardial infarction and denies any other significant medical history. The patient is taking lovastatin (Mevacor, 400 mg/day), nifedipine (Procardia, 60 mg once per day), and atenolol (50 mg once per day). He has no known drug allergy.

The patient was referred by a periodontist for evaluation of continued pain associated with tooth #3. He had been receiving periodontal maintenance therapy for generalized moderate adult periodontitis for more than 5 years. He had root canal treatment and a mesial buccal root amputation of tooth #3 as treatment for a localized area of advanced periodontitis 6 months ago.

Subjective History

After careful questioning, it becomes apparent that the patient is experiencing pain of two different qualities: an intermittent ache associated with tooth #3 and an intermittent sharp shooting pain also associated with tooth #3. The intermittent dull ache was of gradual onset 9 months ago. This pain was unaffected by the nonsurgical root canal treatment and root amputation. This pain has increased in frequency, intensity, and duration over the past 3 months. There is no temporal component. The dull ache is aggravated by biting and by the occurrence of the sharp shooting pain. The sharp shooting pain had a sudden onset 6 months ago. It has also increased in frequency, intensity, and duration without a temporal component. It can occur spontaneously or when the patient is "smiling big." The patient reports that the sharp shooting pain can also be aggravated by pressing lightly on his face in the area overlying #3, but not by pressing intraorally on #3.

Examination

The coronal portion of the amputated mesiobuccal root had been restored with IRM (Intermediate Restorative Material; DENTSPLY Caulk, Milford, DE). No cracks, fractures, sinus tracts, or swelling is detected. There are generalized probing depths of 4 mm throughout the upper right sextant. Tooth #3 has an 8-mm-broad probing defect mesially with bleeding on probing. For the results of clinical testing, see Table 17-2.

TABLE 17-2
Case 1: Clinical Results of Testing

	Tooth		
Test	#2	#3	#4
Endo Ice*	+ (s)†	–	+ (s)
Percussion	–	+	–
Palpation	–	–	–

*Endo Ice (Coltène/Whaledent, Cuyahoga Falls, Ohio) is used to detect pulp vitality.
†s, pain of short duration.

FIG. 17-11 Periapical radiograph showing prior nonsurgical root canal treatment and mesiobuccal root amputation of tooth #3.

A periapical radiograph (Fig. 17-11) shows evidence of prior nonsurgical root canal treatment and mesiobuccal root amputation of tooth #3. Mild to moderate horizontal bone loss is evident in the quadrant. No radiographic evidence of caries or apical radiolucencies is noted.

Additional Tests

In the absence of a clear etiology, a more extensive extraoral examination is performed. Cranial nerves II through XII are intact. The sharp shooting pain is predictably produced with light brushing of the skin over the area of tooth #3. This examination increases the patient's subjective complaint of a dull ache associated with tooth #3. With the likelihood of two possible sources of pain existing, a diagnostic anesthetic block of tooth #3 is performed: buccal infiltration of tooth #3 with 27 mg of 3% mepivacaine without epinephrine. After 3 minutes the patient no longer reports a dull ache at tooth #3 and he is nontender to percussion. His sharp shooting pain can still be initiated with light brushing of the skin over the area of tooth #3 and continues to cause a dull ache in the area of tooth #3. Diagnoses of trigeminal neuralgia and advanced localized adult periodontitis of tooth #3 are made. The patient is referred to a neurologist for evaluation and treatment. The diagnosis of trigeminal neuralgia is confirmed and he is placed on carbamazepine at 100 mg/day.

Case 2

A 28-year-old male presents with a chief complaint of "My teeth on the right side hurt." His medical history is not significant. He denies any systemic disease and has no known drug

allergies. He is currently taking 600 mg of ibuprofen as needed for pain. He is taking no other medications. The patient was referred by his general clinician for evaluation of pain associated with his teeth on the right side.

Subjective History

After careful questioning, it is determined that the patient is experiencing pain of two different types. The most distressing pain to the patient is a diffuse, right-sided, constant low-grade dull ache (3/10 on a verbal analog scale [VAS]). The onset was gradual, beginning 2 years ago. The pain has recently increased in intensity and duration. This pain is aggravated by opening wide and increases in intensity after the occurrence of a sharp pain that is induced by biting down. There is no notable temporal component, and the patient has made no attempts to obtund the pain.

His other pain type had a sudden onset approximately 4 months ago. This pain is localized to the area of the right first molars. It is an intermittent sharp shooting pain (8/10 on VAS) that occurs when biting.

Examination

Tooth #3 has an occlusal amalgam with cracks evident on the mesial marginal ridge and the buccal groove. Tooth #30 has an occlusal amalgam, and cracks are noted on the mesial and distal marginal ridges of the tooth. There are no swellings or sinus tracts and no probing greater than 4 mm on the right side. A periapical radiograph demonstrates no evidence of caries or apical radiolucencies. The patient's sharp pain is reproduced with a bite test applied to the mesial lingual cusp of tooth #30. After the bite test, the patient reports that his dull ache has intensified. For the results of clinical testing see Table 17-3. Thirty seconds after pulp testing has been completed, the patient again reports that his dull ache has intensified.

Additional Tests

In consideration of an uncertain diagnosis, a more extensive examination is performed. Palpation and provocation tests of the muscles of mastication reveal a trigger point in the patient's right deep masseter. Palpation of this trigger point immediately intensifies his "toothache." A trigger point injection of 3% mepivacaine without epinephrine is done in an effort to clarify a diagnosis. After a trigger point injection, all tests are repeated. Palpation of the trigger point no longer produces pain. The bite test and cold test still produce a short sharp pain but are no longer followed by a dull ache.

Diagnoses of reversible pulpitis secondary to a cracked tooth #31 and myofascial pain of the right masseter are made. The patient is given home care instructions for treatment of his myofascial pain, and he is referred to his general clinician for cuspal coverage of both teeth #3 and #30.

TABLE 17-3

Case 2: Clinical Results of Testing

	Tooth					
Test	#2	#3	#4	#31	#30	#29
Endo Ice	+ (s)	+ (s)	+ (s)	+ (s)	+ (s)	+ (s)
Percussion	–	–	–	–	–	–
Palpation	–	–	–	–	–	–

SUMMARY

As clinicians who are frequently called on to diagnose and treat complaints of orofacial pain, it is important to have a thorough knowledge of the odontogenic and nonodontogenic causes. The basis for this knowledge begins with an understanding of the anatomy and physiology of the pain system, and how alterations in this system can result in pain that is poorly localized and therefore misdiagnosed. A realization that pain does not always originate in the structures in which they are felt, along with an understanding of the neurobiologic basis of heterotopic pain, is necessary to ensure accurate diagnosis of orofacial pain.

There are several indicators that a toothache may be nonodontogenic in origin. Red flags for nonodontogenic pain include toothaches that have no apparent etiology for pulpal or periradicular pathosis; pain that is spontaneous, poorly localized, or migratory; and pain that is constant and nonvariable. In addition, pain that is described as burning, pricking, or "shocklike" is less likely to be pulpal or periradicular in origin.

A thorough pain history and an examination of dental and nondental structures are essential to differentiate between odontogenic and nonodontogenic sources of pain. Examples of key components of the pain history and examination are included in this chapter for reference. In addition, the chapter has focused on the more common nonodontogenic sources of orofacial pain. As stated previously, the role of the dental clinician is to diagnose and treat disorders of the oral cavity and masticatory structures. In the event that a nondental pathosis is suspected, a differential diagnosis of probable disorders is essential as part of a referral to a more appropriate health care provider. In addition, an understanding of any potential role or interaction of dental structures in the patient's pain complaint should be communicated as part of the referral.

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CHAPTER 18

Management of Endodontic Emergencies

SAMUEL O. DORN | GARY SHUN-PAN CHEUNG

CHAPTER OUTLINE

Emergency Classifications
Emergency Endodontic Management
Teeth with Vital Pulps
Reversible Pulpitis
Irreversible Pulpitis
Pulpal Necrosis with Acute Apical Abscess
Fascial Space Infections
Management of Abscesses and Cellulitis
Incision for Drainage
Symptomatic Teeth with Previous Endodontic Treatment
Leaving Teeth Open
Systemic Antibiotics for Endodontic Infections
Analgesics
Laboratory Diagnostic Adjuncts
Flare-Ups
Cracked and Fractured Teeth
Summary

EMERGENCY CLASSIFICATIONS

The proper diagnosis and effective management of acute dental pain are possibly the most rewarding and satisfying aspects of providing dental care. An endodontic emergency is defined as pain or swelling caused by various stages of inflammation or infection of the pulpal or periapical tissues. The cause of dental pain is typically from caries, deep or defective restorations, or trauma. Sometimes occlusion-related pain can also mimic acute dental pain (Fig. 18-1). Bender[8] stated that patients who manifest severe or referred pain almost always had a previous history of pain with the offending tooth. Approximately 85% of all dental emergencies arise as a result of pulpal or periapical disease, which would necessitate either extraction or endodontic treatment to relieve the symptoms.[38,68] It has also been estimated that about 12% of the U.S. population experienced a toothache in the preceding 6 months.[65]

Determining a definitive diagnosis can sometimes be challenging and even frustrating for the clinician; but a methodical, objective, and subjective evaluation, as described in Chapter 1, is imperative before developing a proper treatment plan. Unfortunately, on the basis of the diagnosis, there are conflicting opinions on how to best clinically manage various endodontic emergencies. According to surveys of board certified endodontists by Dorn and associates in 1977[22,23] and 1990[31] and by Lee in 2009,[63] there are seven clinical presentations that are considered endodontic emergencies:

1. Irreversible pulpitis with normal periapex
2. Irreversible pulpitis with symptomatic apical periodontitis
3. Necrotic pulp with symptomatic apical periodontitis, with no swelling
4. Necrotic pulp, fluctuant swelling, with drainage through the canal
5. Necrotic pulp, fluctuant swelling, with no drainage through the canal
6. Necrotic pulp, diffuse facial swelling, with drainage through the canal
7. Necrotic pulp, diffuse facial swelling, with no drainage through the canal

There are other endodontic emergencies that were not discussed in these surveys. These emergencies pertain to traumatic dental injuries, as discussed in Chapter 20, to teeth that have had previous endodontic treatment, as discussed in Chapters 8 and 19, and endodontic flare-ups that may occur between treatment sessions. Of course, there are also many types of facial pain that have a nonodontogenic origin; these are described in detail in Chapter 17.

In the decades between the previously cited surveys, there have been several changes pertaining to the preferred clinical management of endodontic emergencies. Many of these treatment modifications have occurred because of the more contemporary armamentarium and materials as well as new evidence-based research and the presumption of empirical clinical success.

EMERGENCY ENDODONTIC MANAGEMENT

Because pain is both a psychological and biologic entity, as discussed in Chapters 4 and online Chapter 28, the management of acute dental pain must take into consideration both the physical symptoms and the emotional status of the

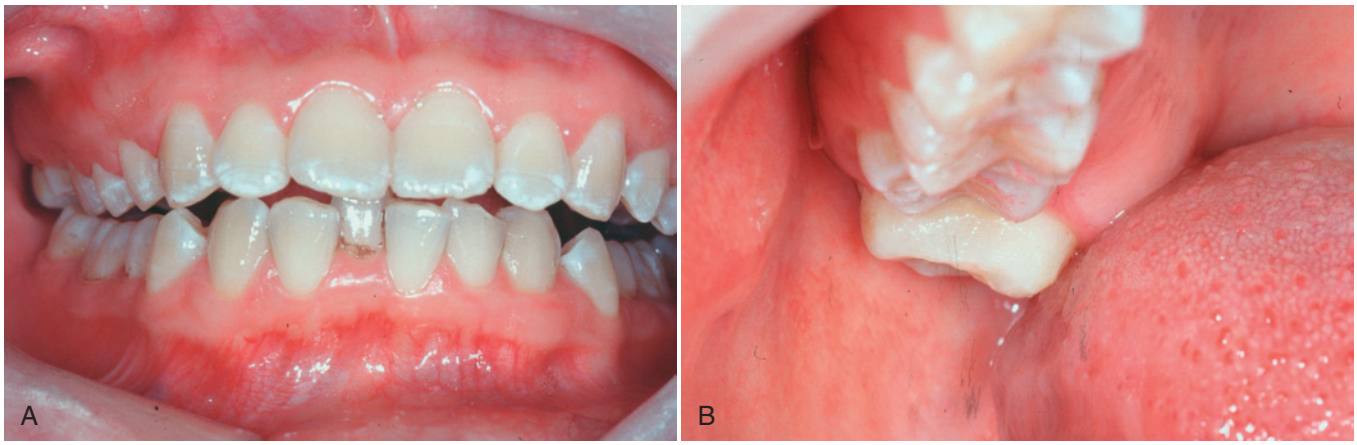

FIG. 18-1 A, Patient complained of acute pain on biting at the lower right molar. B, The pain was resolved after removal of an overerupted upper right wisdom tooth. Notice the presence of wear facet on the mesial marginal ridge and surface of this tooth before extraction.

patient. The patient's needs, fears, and coping mechanisms must be compassionately understood. This assessment and the clinician's ability to build rapport with the patient are key factors in the comprehensive success of the patient's management.[8,30,48,91]

The methodical steps for determining an accurate diagnosis, based on evaluation of the patient's chief complaint, review of the medical history, and the protocols used for an objective and subjective diagnosis, are described in detail in Chapter 1. Once it has been determined that endodontic treatment is necessary, it is incumbent on the clinician to take the proper steps necessary to manage the acute dental emergency.

As described in Chapters 3 and 29, the clinician has a responsibility to inform the patient of the recommended treatment plan and to advise the patient of the treatment alternatives, the risks and benefits that pertain, and the expected prognosis under the present circumstances. Given this information, the patient may elect extraction over endodontics, or possibly request a second opinion. The treatment plan should never be forced on a patient. The informed course of treatment is made jointly between the patient and the clinician.

In the event of an endodontic emergency, the clinician must determine the optimal mode of endodontic treatment pursuant to the diagnosis. Treatment may vary depending on the pulpal or periapical status, the intensity and duration of pain, and whether there is diffuse or fluctuant swelling. Paradoxically, as discussed later, the mode of therapy that we tend to choose has been directed more from surveys of practicing endodontists rather than from controlled clinical studies or research investigations.

Teeth with Vital Pulps

As described in Chapter 1, teeth with vital pulps can have one of the following presentations:

- *Normal.* The teeth are asymptomatic with no objective pathoses.
- *Reversible pulpitis.* There is a reversible sensitivity to cold or osmotic changes (i.e., sweet, salty, and sour).
- *Irreversible pulpitis.* The sensitivity to temperature changes is more intense and with a longer duration.

Reversible Pulpitis

Reversible pulpitis can be induced by caries, exposed dentin, recent dental treatment, and defective restorations. Conservative removal of caries, protection of dentin, and a proper restoration will typically resolve the symptoms. However, the symptoms from exposed dentin, specifically from gingival recession and cervically exposed roots, can often be difficult to alleviate. Topical applications of desensitizing agents and the use of certain dentifrices have been helpful in the management of dentin hypersensitivity; the etiology, physiology, and management of this are discussed in Chapter 12.

Irreversible Pulpitis

The diagnosis of irreversible pulpitis can be subcategorized as asymptomatic or symptomatic. *Asymptomatic* irreversible pulpitis pertains to a tooth that has no symptoms, but has deep caries or tooth structure loss that, if left untreated, will cause the tooth to become symptomatic or nonvital. On the other hand, the pain from *symptomatic* irreversible pulpitis is often an emergency condition that requires immediate treatment. These teeth exhibit intermittent or spontaneous pain, whereby exposure to extreme temperatures, especially cold, will elicit intense and prolonged episodes of pain, even after the source of the stimulus is removed.

In 1977,[22,23] 187 board-certified endodontists responded to a survey to determine how they would manage various endodontic emergencies. Ten years later, 314 board-certified endodontists responded to the same questionnaire in order to determine whether there had been any changes in how these emergencies were managed.[31] The clinical management of emergency treatment of a tooth with irreversible pulpitis with or without a normal periapex seemed to be fairly similar by removing the inflamed pulp tissue either by pulpotomy or complete instrumentation.[75] In a similar survey conducted in 2009,[63] most respondents stated that they cleaned to the level of the "apex," as confirmed with an electronic apex locator; this suggests a change in the management of endodontic cases based on the advent of a more contemporary armamentarium. In general, the most current survey indicates that there is a trend toward more cleaning and shaping of the canal when irreversible pulpitis presents with a normal periapex,

compared with performing just pulpectomies as described in the 1977 survey. None of the individuals surveyed in the 1990 or 2009 poll stated that they would manage these emergencies by establishing any type of drainage by trephinating the apex, making an incision, or leaving the tooth open for an extended period of time.

In addition, for vital teeth, the 1977 survey did not even broach the concept of completing the endodontics in one visit, whereas in the 1988 study about one third of the respondents indicated that they would complete these vital cases in a single visit and the response rose to 79% in the most recent survey. Since the early 1980s, there seems to have been an increase in the acceptability of providing endodontic therapy in one visit, especially in cases of vital pulps, with most studies revealing an equal number, or fewer, flare-ups after single-visit endodontic treatment.[24,78,83,88,90,98] However, this has not come without controversy, with some studies showing otherwise,[111] contending that there is more posttreatment pain after single-visit endodontics, and possibly a lower long-term success rate. Unfortunately, time constraints at the emergency visit often make the single-visit treatment option not practical.[4]

If root canal therapy is completed at a later date, medicating the canal with calcium hydroxide has been suggested to reduce the chances of bacterial growth in the canal between appointments in most studies,[17] but not all.[13,17] One randomized clinical study showed that a dry cotton pellet was as effective in relieving pain as a pellet moistened with camphorated monochlorophenol (CMCP), metacresylacetate (Cresatin), eugenol, or saline.[40] Sources of infection, such as caries and defective restorations, should be completely removed to prevent recontamination of the root canal system between appointments.[40] The concept of single- versus multiple-visit endodontics is described in greater detail in Chapter 11.

For emergency management of vital teeth that are not initially sensitive to percussion, occlusal reduction has not been shown to be beneficial.[19,31] However, the clinician should be cognizant of the possibility of occlusal interferences and prematurities that might cause tooth fracture under heavy mastication. In vital teeth in which the inflammation has extended periapically, which will present with pretreatment pain to percussion, occlusal reduction has been reported to reduce posttreatment pain.[31,74,89]

Antibiotics are not recommended for the emergency management of irreversible pulpitis[53,99] (see Chapters 11 and 14), as placebo-controlled clinical trials have demonstrated that antibiotics have no effect on pain levels in patients with irreversible pulpitis.[72]

Most endodontists and endodontic textbooks recommend the emergency management of *symptomatic* irreversible pulpitis to involve the initiation of root canal treatment,[17,31,39,63,103] with complete pulp removal and total cleaning of the root canal system. Unfortunately, in an emergency situation, the allotted time necessary for this treatment is often an issue. Given the potential time constraints and inevitable differences in skill level between clinicians, it may not be feasible to complete the total canal cleaning at the initial emergency visit. Subsequently, especially with multirooted teeth, a pulpotomy (removal of the coronal pulp or tissue from the widest canal) has been advocated for emergency treatment of irreversible pulpitis.[15,39,103] In a clinical study of various emergency procedures, it has been demonstrated that this treatment is highly effective for alleviating acute dental pain due to irreversible pulpitis.[15]

To assist the clinician in assessing the level of difficulty of a given endodontic case, the American Association of Endodontists (Chicago, IL) has developed the "AAE Endodontic Case Difficulty Assessment Form and Guidelines" (Fig. 18-2). This form is intended to make case selection more efficient, more consistent, and easier to document, as well as to provide a more objective ability to determine when it may be necessary to refer the patient to another clinician who may be better able to manage the complexities of the case.

Pulpal Necrosis with Acute Apical Abscess

No Swelling

Over the years, the proper methodology for the emergency endodontic management of necrotic teeth has been controversial. In a 1977 survey of board-certified endodontists,[22,23] it was reported that, in the absence of swelling, most respondents would completely instrument the canals, keeping the file short of the radiographic apex. However, when swelling was present, the majority of those polled in 1977 preferred to leave the tooth open, with instrumentation extending beyond the apex to help facilitate drainage through the canals. Years later and again validated in a 2009 study, most respondents favored complete instrumentation regardless of the presence of swelling. Also, it was the decision of 25.2% to 38.5% of the clinicians to leave these teeth open in the event of diffuse swelling; 17.5% to 31.5% left the teeth open in the presence of a fluctuant swelling. However, as discussed later, there is currently a trend toward not leaving teeth open for drainage. There is also another trend: when treatment is done in more than one visit, most endodontists will use calcium hydroxide as an intracanal medicament.[63]

Care should be taken not to push necrotic debris beyond the apex during root canal instrumentation, as this has been shown to promote more posttreatment discomfort.[13,31,87,96] Crown-down instrumentation techniques have been shown to remove most of the debris coronally rather than pushing it out the apex. The use of positive-pressure irrigation methods, such as needle-and-syringe irrigation, also poses a risk of expressing debris or solution out of the apex.[10,20] Improvements in technology, such as electronic apex locators, have facilitated increased accuracy in determining working length measurements, which in turn may allow for a more thorough canal debridement and less apical extrusion. These devices are now used by an increased number of clinicians.[56,63]

Trephination

In the absence of swelling, trephination is the surgical perforation of the alveolar cortical plate to release, from between the cortical plates, the accumulated inflammatory and infective tissue exudate that causes pain. Its use has been historically advocated to provide pain relief in patients with severe and recalcitrant periradicular pain.[22,23] The technique involves an engine-driven perforator entering through the cortical bone and into the cancellous bone, often without the need for an incision,[16] in order to provide a pathway for drainage from the periradicular tissues. Although more recent studies have failed to show the benefit of trephination in patients with irreversible pulpitis with symptomatic apical periodontitis[69] or necrotic teeth with symptomatic apical periodontitis,[74] there remain some advocates who recommend trephination for managing acute and intractable periapical pain.[45] The clinician should

understand that local anesthesia may be difficult for cases with acute inflammation or infection.[49] Extreme care must be taken when carrying out a trephination procedure to guard against inadvertent and possibly irreversible injury to the tooth root or surrounding structures, such as the mental foramen, intra-alveolar nerve, or maxillary sinus.

Necrosis and Single-Visit Endodontics

Although single-visit endodontic treatment for teeth diagnosed with irreversible pulpitis is not contraindicated,[2,83,85,90,112] performing single-visit endodontics on necrotic and previously treated teeth is not without controversy. In cases of necrotic teeth, although research[24] has indicated that there may be no difference in posttreatment pain if the canals are filled at the time of the emergency versus a later date, some studies[97,104] have questioned the long-term prognosis of such treatment, especially in cases of symptomatic apical periodontitis. Several studies,[25,60] including a CONSORT (Consolidated Standards of Reporting Trials) meta-analysis,[84] have shown no difference in outcome between single-visit and two-visit treatments. The concept of single- versus multivisit endodontics is further discussed in Chapters 3 and 11.

AAE Endodontic Case Difficulty Assessment Form and Guidelines

PATIENT INFORMATION

Name_____

Address_____

City/State/Zip_____

Phone_____

DISPOSITION

Treat in Office: Yes ☐ No ☐

Refer Patient to:

_____

Date:_____

Guidelines for Using the AAE Endodontic Case Difficulty Assessment Form

The AAE designed the Endodontic Case Difficulty Assessment Form for use in endodontic curricula. The Assessment Form makes case selection more efficient, more consistent and easier to document. Dentists may also choose to use the Assessment Form to help with referral decision making and record keeping.

Conditions listed in this form should be considered potential risk factors that may complicate treatment and adversely affect the outcome. Levels of difficulty are sets of conditions that may not be controllable by the dentist. Risk factors can influence the ability to provide care at a consistently predictable level and impact the appropriate provision of care and quality assurance.

The Assessment Form enables a practitioner to assign a level of difficulty to a particular case.

LEVELS OF DIFFICULTY

MINIMAL DIFFICULTY	Preoperative condition indicates routine complexity (uncomplicated). These types of cases would exhibit only those factors listed in the MINIMAL DIFFICULTY category. Achieving a predictable treatment outcome should be attainable by a competent practitioner with limited experience.
MODERATE DIFFICULTY	Preoperative condition is complicated, exhibiting one or more patient or treatment factors listed in the MODERATE DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for a competent, experienced practitioner.
HIGH DIFFICULTY	Preoperative condition is exceptionally complicated, exhibiting several factors listed in the MODERATE DIFFICULTY category or at least one in the HIGH DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for even the most experienced practitioner with an extensive history of favorable outcomes.

Review your assessment of each case to determine the level of difficulty. If the level of difficulty exceeds your experience and comfort, you might consider referral to an endodontist.

The AAE Endodontic Case Difficulty Assessment Form is designed to aid the practitioner in determining appropriate case disposition. The American Association of Endodontists neither expressly nor implicitly warrants any positive results associated with the use of this form. This form may be reproduced but may not be amended or altered in any way.

© American Association of Endodontists, 211 E. Chicago Ave., Suite 1100, Chicago, IL 60611-2691; Phone: 800/872-3636 or 312/266-7255; Fax: 866/451-9020 or 312/266-9867; E-mail: info@aae.org; Web site: *www.aae.org*

FIG. 18-2 The American Association of Endodontists (AAE) Endodontic Case Difficulty Assessment Form and Guidelines, developed to assist the clinician in assessing the level of difficulty of a given endodontic case and to help determine when referral may be necessary. *Continued*

AAE Endodontic Case Difficulty Assessment Form

Criteria and Subcriteria	Minimal Difficulty	Moderate Difficulty	High Difficulty
A. PATIENT CONSIDERATIONS			
Medical history	☐ No medical problem (ASA Class 1*)	☐ One or more medical problems (ASA Class 2*)	☐ Complex medical history/serious illness/disability (ASA Classes 3-5*)
Anesthesia	☐ No history of anesthesia problems	☐ Vasoconstrictor intolerance	☐ Difficulty achieving anesthesia
Patient disposition	☐ Cooperative and compliant	☐ Anxious but cooperative	☐ Uncooperative
Ability to open mouth	☐ No limitation	☐ Slight limitation in opening	☐ Significant limitation in opening
Gag reflex	☐ None	☐ Gags occasionally with radiographs/treatment	☐ Extreme gag reflex which has compromised past dental care
Emergency condition	☐ Minimum pain or swelling	☐ Moderate pain or swelling	☐ Severe pain or swelling
B. DIAGNOSTIC AND TREATMENT CONSIDERATIONS			
Diagnosis	☐ Signs and symptoms consistent with recognized pulpal and periapical conditions	☐ Extensive differential diagnosis of usual signs and symptoms required	☐ Confusing and complex signs and symptoms: difficult diagnosis ☐ History of chronic oral/facial pain
Radiographic difficulties	☐ Minimal difficulty obtaining/interpreting radiographs	☐ Moderate difficulty obtaining/interpreting radiographs (*e.g.*, high floor of mouth, narrow or low palatal vault, presence of tori)	☐ Extreme difficulty obtaining/interpreting radiographs (*e.g.*, superimposed anatomical structures)
Position in the arch	☐ Anterior/premolar ☐ Slight inclination (<10°) ☐ Slight rotation (<10°)	☐ 1st molar ☐ Moderate inclination (10-30°) ☐ Moderate rotation (10-30°)	☐ 2nd or 3rd molar ☐ Extreme inclination (>30°) ☐ Extreme rotation (>30°)
Tooth isolation	☐ Routine rubber dam placement	☐ Simple pretreatment modification required for rubber dam isolation	☐ Extensive pretreatment modification required for rubber dam isolation
Morphologic aberrations of crown	☐ Normal original crown morphology	☐ Full coverage restoration ☐ Porcelain restoration ☐ Bridge abutment ☐ Moderate deviation from normal tooth/root form (*e.g.*, taurodontism, microdens) ☐ Teeth with extensive coronal destruction	☐ Restoration does not reflect original anatomy/alignment ☐ Significant deviation from normal tooth/root form (*e.g.*, fusion, dens in dente)
Canal and root morphology	☐ Slight or no curvature (<10°) ☐ Closed apex <1 mm diameter	☐ Moderate curvature (10-30°) ☐ Crown axis differs moderately from root axis. Apical opening 1-1.5 mm in diameter	☐ Extreme curvature (>30°) or S-shaped curve ☐ Mandibular premolar or anterior with 2 roots ☐ Maxillary premolar with 3 roots ☐ Canal divides in the middle or apical third ☐ Very long tooth (>25 mm) ☐ Open apex (>1.5 mm in diameter)
Radiographic appearance of canal(s)	☐ Canal(s) visible and not reduced in size	☐ Canal(s) and chamber visible but reduced in size ☐ Pulp stones	☐ Indistinct canal path ☐ Canal(s) not visible
Resorption	☐ No resorption evident	☐ Minimal apical resorption	☐ Extensive apical resorption ☐ Internal resorption ☐ External resorption
C. ADDITIONAL CONSIDERATIONS			
Trauma history	☐ Uncomplicated crown fracture of mature or immature teeth	☐ Complicated crown fracture of mature teeth ☐ Subluxation	☐ Complicated crown fracture of immature teeth ☐ Horizontal root fracture ☐ Alveolar fracture ☐ Intrusive, extrusive or lateral luxation ☐ Avulsion
Endodontic treatment history	☐ No previous treatment	☐ Previous access without complications	☐ Previous access with complications (*e.g.*, perforation, non-negotiated canal, ledge, separated instrument) ☐ Previous surgical or nonsurgical endodontic treatment completed
Periodontal-endodontic condition	☐ None or mild periodontal disease	☐ Concurrent moderate periodontal disease	☐ Concurrent severe periodontal disease ☐ Cracked teeth with periodontal complications ☐ Combined endodontic/periodontic lesion ☐ Root amputation prior to endodontic treatment

*American Society of Anesthesiologists (ASA) Classification System

Class 1: No systemic illness. Patient healthy.
Class 2: Patient with mild degree of systemic illness, but without functional restrictions, *e.g.*, well-controlled hypertension.
Class 3: Patient with severe degree of systemic illness which limits activities, but does not immobilize the patient.
Class 4: Patient with severe systemic illness that immobilizes and is sometimes life threatening.
Class 5: Patient will not survive more than 24 hours whether or not surgical intervention takes place.

www.asahq.org/clinical/physicalstatus.htm

FIG. 18-2, cont'd

Swelling

Tissue swelling may be associated with an acute periradicular abscess at the time of the initial emergency visit, or it may occur as an inter-appointment flare-up or as a postendodontic complication. Swellings may be localized or diffuse, fluctuant or firm. Localized swellings are confined within the oral cavity, whereas a diffuse swelling, or cellulitis, is more extensive, spreading through adjacent soft tissues and dissecting tissue spaces along fascial planes.[92]

Swelling may be controlled by establishing drainage through the root canal or by incising the fluctuant swelling. As discussed later and in Chapter 14, antibiotics may be recruited as part of the management of swelling, especially when there are systemic manifestations of the infection, such as fever and malaise. The principal modality for managing swelling secondary to endodontic infections is to achieve drainage and remove the source of the infection.[36,92] When the swelling is localized, the preferred avenue is drainage through the root canal (Fig. 18-3). However, it is also possible to achieve drainage with an incision and iodoform gauze drain before entering the canal. In this manner, the canal can be dried and the endodontic treatment completed in one visit. The dentist should see the patient the following day to remove the drain. Complete canal debridement and disinfection[37,106] are paramount

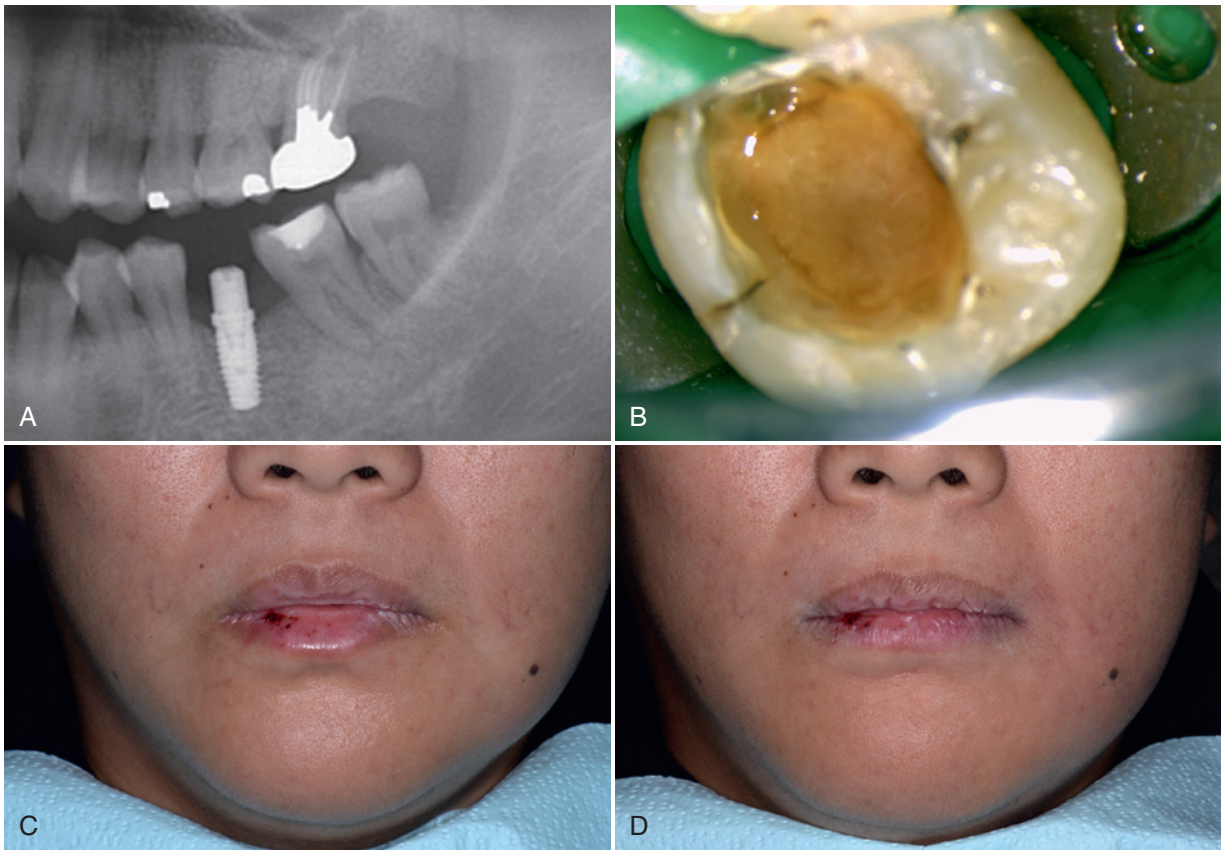

FIG. 18-3 Drainage of pus through the root canal. **A,** Acute apical abscess arising from the lower left first molar with little radiographic radiolucency. **B,** Drainage through the canals. **C,** Extracoronal swelling bear angle of left mandible before drainage. **D,** Reduction in the extent of swelling after drainage.

for success regardless of observable drainage, because the presence of any bacteria remaining within the root canal system will compromise the resolution of the acute infection.[67] In the presence of persistent swelling, gentle finger pressure to the mucosa overlying the swelling may help facilitate drainage through the canal. Once the canals have been cleaned and allowed to dry, the access should be closed.[17,31,39] In these cases, when not completing the treatment in a single visit, there has been a trend to use calcium hydroxide as the intracanal medicament.[63]

FASCIAL SPACE INFECTIONS

If bacteria from the infected root canal gain entry into the periradicular tissues and the immune system is unable to suppress the invasion, an otherwise healthy patient eventually shows signs and symptoms of an acute apical abscess, which can in turn evolve to cellulitis. Clinically, the patient experiences swelling and mild to severe pain. Depending on the relationship of the apices of the involved tooth to the muscular attachments, the swelling may be localized to the vestibule or extend into a fascial space. The patient may also have systemic manifestations, such as fever, chills, lymphadenopathy, headache, and nausea. Because the reaction to the infection may occur quickly, the involved tooth may or may not show radiographic evidence of a widened periodontal ligament space. In most cases, the tooth elicits a positive response to percussion, and the periradicular area is tender to palpation. The tooth now becomes a *focus of infection* because it leads to periradicular infection and secondary spread to the fascial spaces of the head and neck, resulting in cellulitis and systemic signs and symptoms of infection.

In such cases, treatment may involve incision for drainage, root canal treatment, or extraction in order to remove the source of the infection. Antibiotic therapy may be indicated in patients with a compromised host resistance, the presence of systemic symptoms, or fascial space involvement. Fascial space infections of odontogenic origin are infections that have spread into the fascial spaces from the periradicular area of a tooth, the focus of infection. They are *not* examples of the theory of focal infection, which describes the dissemination of bacteria or their products from a distant focus of infection. Rather, this is an example of the local spread of infection from an odontogenic source.

Fascial spaces are potential anatomic areas that exist between the fascia and underlying organs and other tissues. During infection, these spaces are formed as a result of the spread of purulent exudate. The spread of infections of odontogenic origin into the fascial spaces of the head and neck is determined by the location of the root end of the involved tooth in relation to its overlying buccal or lingual cortical plate and the relationship of the apex to the attachment of a muscle (Fig. 18-4, *A*). For example, if the source of the infection is a mandibular molar whose apices lie closer to the lingual cortical plate and *above* the attachment of the mylohyoid muscle of the floor of the mouth, the purulent exudate may break through

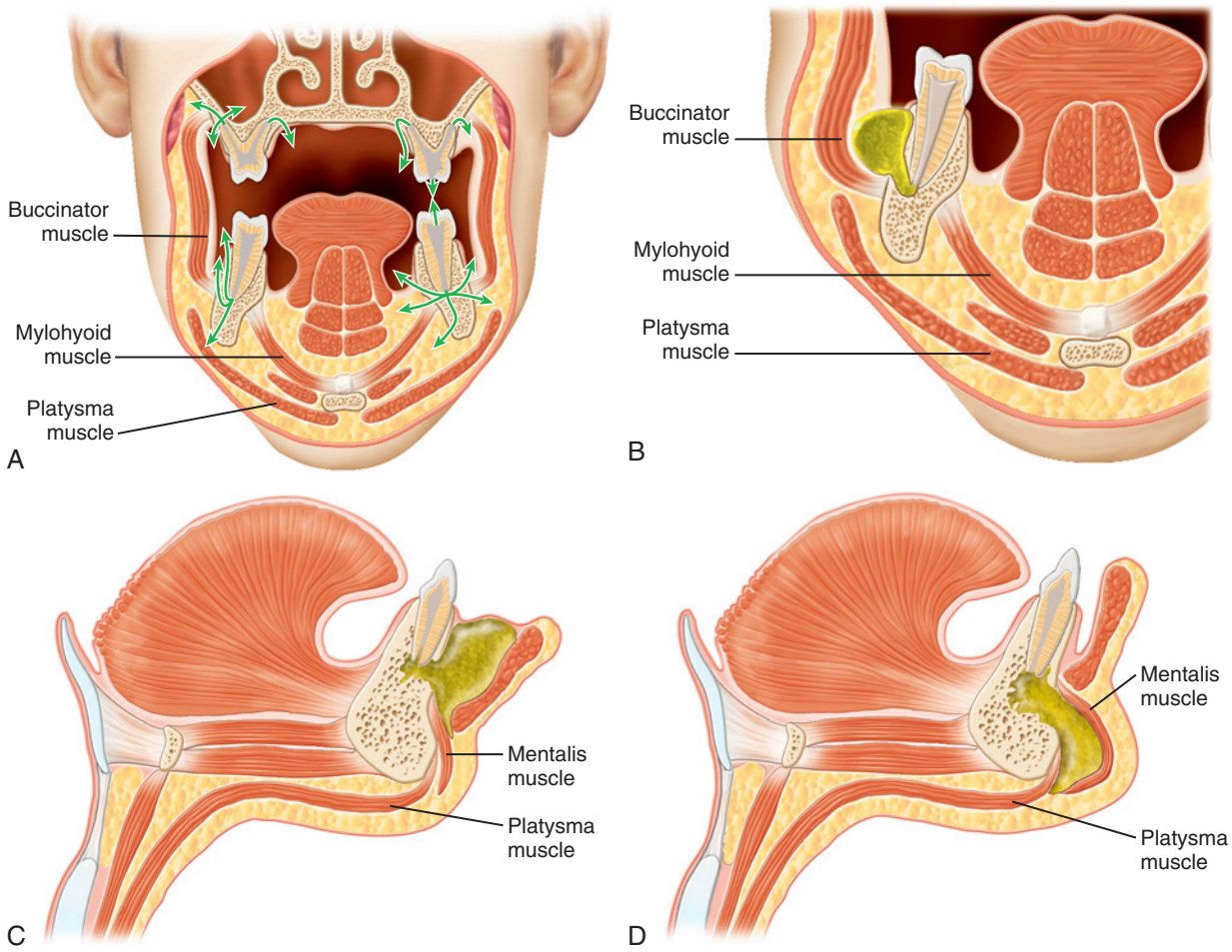

FIG. 18-4 A, Spread of odontogenic infections. B, Mandibular buccal vestibule (posterior tooth). C, Mandibular buccal vestibule (anterior tooth). D, Mental space. *Continued*

the lingual cortical plate and into the *sublingual* space. However, if the apices lie *below* the attachment of the mylohyoid muscle, the infection may spread into the *submandibular* space.

As described by Hohl and colleagues,[47] the fascial spaces of the head and neck can be categorized into four anatomic groups:

- The mandible and below
- The cheek and lateral face
- The pharyngeal and cervical areas
- The midface

Swellings of and below the mandible include six anatomic areas or fascial spaces:

- The buccal vestibule
- The body of the mandible
- The mental space
- The submental space
- The sublingual space
- The submandibular space

The *mandibular buccal vestibule* is the anatomic area amid the buccal cortical plate, the overlying alveolar mucosa, and the buccinator muscle (posterior) or the mentalis muscle (anterior) (Figs. 18-4, *B* and *C*). In this case, the source of the infection is a mandibular posterior or anterior tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex or apices of the involved tooth lie above the attachment of the buccinator or mentalis muscle, respectively.

The *space of the body of the mandible* is the potential anatomic area between the buccal or lingual cortical plate and its overlying periosteum. The source of infection is a mandibular tooth in which the purulent exudate has broken through the overlying cortical plate but not yet perforated the overlying periosteum. Involvement of this space can also occur as a result of a postsurgical infection.

The *mental space* (Fig. 18-4, *D*) is the potential bilateral anatomic area of the chin that lies between the mentalis muscle superiorly and the platysma muscle inferiorly. The source of the infection is an anterior tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies below the attachment of the mentalis muscle.

The *submental space* (Fig. 18-4, *E*) is the potential anatomic area between the mylohyoid muscle superiorly and the platysma muscle inferiorly. The source of the infection is an anterior tooth in which the purulent exudate breaks through the lingual cortical plate, and the apex of the tooth lies below the attachment of the mylohyoid muscle.

The *sublingual space* (Fig. 18-4, *F*) is the potential anatomic area between the oral mucosa of the floor of the mouth superiorly and the mylohyoid muscle inferiorly. The lateral boundaries of the space are the lingual surfaces of the mandible. The

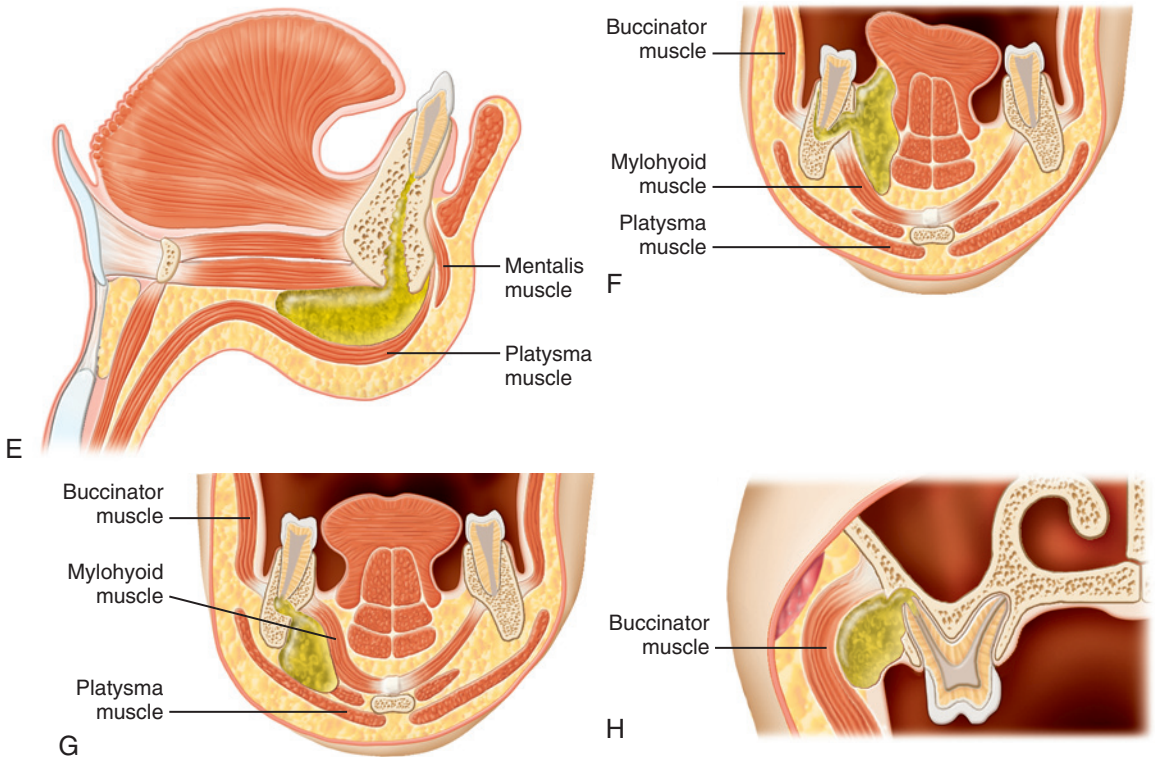

FIG. 18-4, cont'd E, Submental space. F, Sublingual space. G, Submandibular space. H, Maxillary buccal vestibule.

source of infection is any mandibular tooth in which the purulent exudate breaks through the lingual cortical plate, and the apex or apices of the tooth lie above the attachment of the mylohyoid muscle.

The *submandibular space* (Fig. 18-4, *G*) is the potential space between the mylohyoid muscle superiorly and the platysma muscle inferiorly. The source of infection is a posterior tooth, usually a molar, in which the purulent exudate breaks through the lingual cortical plate and the apices of the tooth lie below the attachment of the mylohyoid muscle. If the submental, sublingual, and submandibular spaces are involved at the same time, a diagnosis of Ludwig angina is made. This life-threatening cellulitis can advance into the pharyngeal and cervical spaces, resulting in airway obstruction.

Swellings of the lateral face and cheek include four anatomic areas or fascial spaces:

- The buccal vestibule of the maxilla
- The buccal space
- The submasseteric space
- The temporal space

Anatomically, the *buccal vestibular space* (Fig. 18-4, *H*) is the area between the buccal cortical plate, the overlying mucosa, and the buccinator muscle. The superior extent of the space is the attachment of the buccinator muscle to the zygomatic process. The source of infection is a maxillary posterior tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies below the attachment of the buccinator muscle.

The *buccal space* (Fig. 18-4, *I*) is the potential space between the lateral surface of the buccinator muscle and the medial surface of the skin of the cheek. The superior extent of the space is the attachment of the buccinator muscle to the zygomatic arch, whereas the inferior and posterior boundaries are the attachment of the buccinator to the inferior border of the mandible and the anterior margin of the masseter muscle, respectively. The source of the infection can be either a posterior mandibular or maxillary tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex or apices of the tooth lie above the attachment of the buccinator muscle (i.e., maxilla) or below the attachment of the buccinator muscle (i.e., mandible).

As the name implies, the *submasseteric space* (Fig. 18-4, *J*) is the potential space between the lateral surface of the ramus of the mandible and the medial surface of the masseter muscle. The source of the infection is usually an impacted third molar in which the purulent exudate breaks through the lingual cortical plate, and the apices of the tooth lie very close to or within the space.

The *temporal space* (Fig. 18-4, *K*) is divided into two compartments by the temporalis muscle. The *deep temporal space* is the potential space between the lateral surface of the skull and the medial surface of the temporalis muscle; the *superficial temporal space* lies between the temporalis muscle and its overlying fascia. The deep or superficial temporal spaces are involved indirectly if an infection spreads superiorly from the inferior pterygomandibular or submasseteric spaces, respectively.

Swellings of the pharyngeal and cervical areas include the following fascial spaces:

- The pterygomandibular space
- The parapharyngeal spaces
- The cervical spaces

The *pterygomandibular space* (Fig. 18-4, *L*) is the potential space between the lateral surface of the medial pterygoid muscle and the medial surface of the ramus of the mandible.

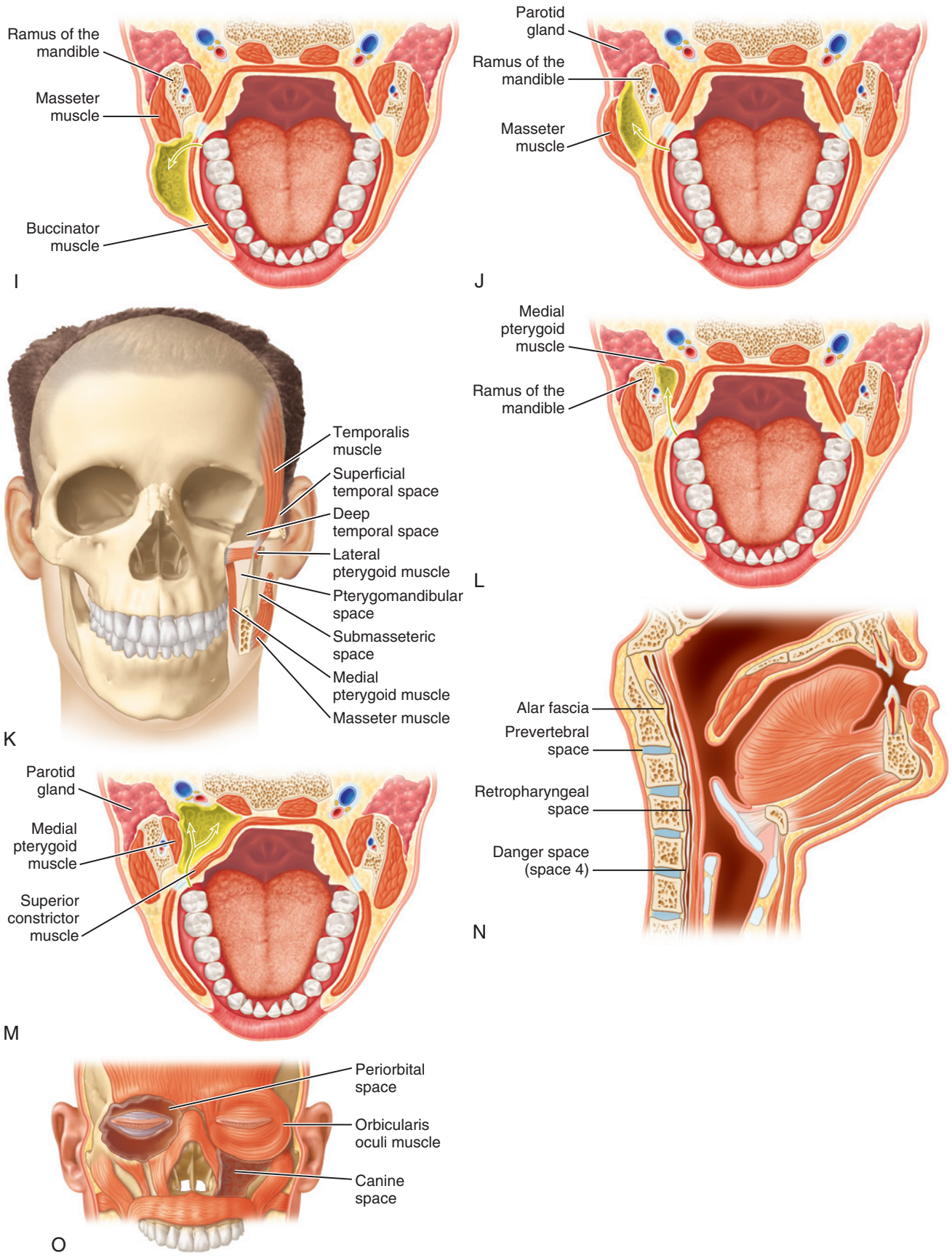

FIG. 18-4, cont'd I, Buccal space. J, Submasseteric space. K, Temporal space. L, Pterygomandibular space. M, Parapharyngeal spaces. N, Cervical spaces. O, Canine (infraorbital) and periorbital space.

The superior extent of the space is the lateral pterygoid muscle. The source of the infection is mandibular second or third molars in which the purulent exudate drains directly into the space. In addition, contaminated inferior alveolar nerve injections can lead to infection of the space.

The *parapharyngeal spaces* comprise the lateral pharyngeal and retropharyngeal spaces (Fig. 18-4, *M*). The *lateral pharyngeal space* is bilateral and lies between the lateral surface of the medial pterygoid muscle and the posterior surface of the superior constrictor muscle. The superior and inferior margins of the space are the base of the skull and the hyoid bone, respectively, and the posterior margin is the *carotid space,* or sheath, which contains the common carotid artery, internal jugular vein, and the vagus nerve. Anatomically, the *retropharyngeal space* lies between the anterior surface of the prevertebral fascia and the posterior surface of the superior constrictor muscle and extends inferiorly into the retroesophageal space, which extends into the posterior compartment of the mediastinum. The pharyngeal spaces usually become involved as a result of the secondary spread of infection from other fascial spaces or directly from a peritonsillar abscess.

The cervical spaces comprise the pretracheal, retrovisceral, danger, and prevertebral spaces (Fig. 18-4, *N*). The *pretracheal space* is the potential space surrounding the trachea. It extends from the thyroid cartilage inferiorly into the superior portion of the anterior compartment of the mediastinum to the level of the aortic arch. Because of its anatomic location, odontogenic infections do not spread to the pretracheal space. The *retrovisceral space* comprises the retropharyngeal space superiorly and the retroesophageal space inferiorly. The space extends from the base of the skull into the posterior compartment of the mediastinum to a level between vertebrae C6 and T4. The *danger space* (i.e., space 4)[26,33] is the potential space between the alar and prevertebral fascia. Because this space is composed of loose connective tissue, it is considered an actual anatomic space extending from the base of the skull into the posterior compartment of the mediastinum to a level corresponding to the diaphragm. It is not unknown for odontogenic infection to spread into this space, if left untreated and undiagnosed.[32] The consequence can be fatal. The *prevertebral space* is the potential space surrounding the vertebral column. As such, it extends from vertebra C1 to the coccyx. A retrospective study showed that 71% of cases in which the mediastinum was involved arose from the spread of infection from the retrovisceral space (21% from the carotid space and 8% from the pretracheal space).[64]

Swellings of the midface consist of four anatomic areas and spaces:

- The palate
- The base of the upper lip
- The canine spaces
- The periorbital spaces

Odontogenic infections can spread into the areas between the palate and its overlying periosteum and mucosa and the base of the upper lip, which lies superior to the orbicularis oris muscle, even though these areas are not considered actual fascial spaces. The source of infection of the palate is any of the maxillary teeth in which the apex of the involved tooth lies close to the palate. The source of infection of the base of the upper lip is a maxillary central incisor in which the apex lies close to the buccal cortical plate and above the attachment of the orbicularis oris muscle.

The *canine,* or *infraorbital, space* (Fig. 18-4, *O*) is the potential space between the levator anguli oris muscle inferiorly and the levator labii superioris muscle superiorly. The source of infection is the maxillary canine or first premolar in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies above the attachment of the levator anguli oris muscle. There is a chance for the infection to spread from the infraorbital space into the cavernous sinus in the cranium via the valveless veins of the face and anterior skull base.[26]

The *periorbital space* (see Fig. 18-4, *O*) is the potential space that lies deep to the orbicularis oculi muscle. This space becomes involved through the spread of infection from the canine or buccal spaces. Infections of the midface can be very dangerous because they can result in *cavernous sinus thrombosis,* a life-threatening infection in which a thrombus formed in the cavernous sinus breaks free, resulting in blockage of an artery or the spread of infection. Under normal conditions, the angular and ophthalmic veins and the pterygoid plexus of veins flow into the facial and external jugular veins. If an infection has spread into the midfacial area, however, edema and the resultant increased pressure from the inflammatory response cause the blood to back up into the cavernous sinus. Once in the sinus, the blood stagnates and clots. The resultant infected thrombi remain in the cavernous sinus or escape into the circulation.[77,115]

MANAGEMENT OF ABSCESSES AND CELLULITIS

The two most important elements of effective patient management for the resolution of an odontogenic infection are correct diagnosis and removal of the cause. When endodontic treatment is possible and preferred, in an otherwise healthy patient, chemomechanical preparation of the infected root canals and incision for drainage of any fluctuant periradicular swelling usually provide prompt improvement of the clinical signs and symptoms. The majority of cases of endodontic infections can be treated effectively without the use of systemic antibiotics. The appropriate treatment is removal of the cause of the inflammatory condition.

Antibiotics are *not* recommended for irreversible pulpitis, symptomatic apical periodontitis, draining sinus tracts, after endodontic surgery, to prevent flare-ups, or after incision for drainage of a localized swelling (without cellulitis, fever, or lymphadenopathy).[28,46,72,86,110] When the ratio of risk to benefit is considered in these situations, antibiotic use may put the patient at risk for side effects of the antimicrobial agent and select for resistant microorganisms. Analgesics (not antibiotics) are indicated for controlling the pain.

Antibiotics in conjunction with appropriate endodontic treatment are recommended for progressive or persistent infections with systemic signs and symptoms such as fever (over 100°F or 37°C), malaise, cellulitis, unexplained trismus, and progressive or persistent swelling (or both). In such cases, antibiotic therapy is indicated as an adjunct to debridement of the root canal system, which is a reservoir of microorganisms. In addition, aggressive incision for drainage is indicated for any infection marked by cellulitis. Incision for drainage is indicated whether the cellulitis is indurated or fluctuant. It is important to provide a pathway of drainage to prevent further spread of the abscess or cellulitis. An incision for drainage

allows decompression of the increased tissue pressure associated with edema and provides significant pain relief. Furthermore, the incision provides a draining pathway not only for bacteria and their products but also for the inflammatory mediators associated with the spread of cellulitis.

A minimum inhibitory concentration of antibiotic may not reach the source of the infection because of decreased blood flow and because the antibiotic must diffuse through the edematous fluid and pus. Drainage of edematous fluid and purulent exudate improves circulation to the tissues associated with an abscess or cellulitis, providing better delivery of the antibiotic to the area. Placement of a drain may not be indicated for localized fluctuant swellings if complete evacuation of the purulent exudate is believed to have occurred.

For effective drainage, a vertically oriented stab incision is made through the mucoperiosteum in the most dependent site of the swelling. The incision must be long enough to allow blunt dissection using a curved hemostat or periosteal elevator under the periosteum for drainage of pockets of inflammatory exudate. A rubber dam drain or a Penrose drain is indicated for any patient with a progressive abscess or cellulitis to maintain an open pathway for drainage. A more detailed description is given later.

Patients with cellulitis should be followed on a daily basis to ensure that the infection is resolving. The best practical guide for determining the duration of antibiotic therapy is clinical improvement of the patient. When clinical evidence indicates that the infection is certain to resolve or is resolved, antibiotics should be administered for no longer than 1 or 2 days more.

Endodontic treatment should be completed as soon as possible after the incision for drainage. The drain can usually be removed 1 or 2 days after improvement. If no significant clinical improvement occurs, the diagnosis and treatment must be reviewed carefully. Consultation with a specialist and referral may be indicated for severe or persistent infections. Likewise, patients requiring extraoral drainage should be referred to a clinician trained in the technique.

INCISION FOR DRAINAGE

Establishing drainage from a localized soft tissue swelling is sometimes necessary. This can be accomplished through the *incision for drainage* of the area.[73] Incision for drainage is indicated whether the cellulitis is indurated or fluctuant,[92] and it is used when a pathway for drainage is needed to prevent further spread of infection. Incision for drainage allows decompression from the increased tissue pressure associated with edema and can provide significant pain relief for the patient; noteworthy is that often pain decreases when the soft tissues swell due to the relief of pressure within the bone. The incision also provides a pathway not only for bacteria and bacterial by-products but also for the inflammatory mediators that are associated with the spread of cellulitis.

The basic principles of incision for drainage are as follows:

- Anesthetize the area.
- Make a vertically oriented incision at the site of greatest fluctuant swelling.
- Dissect gently, through the deeper tissues, and thoroughly explore all parts of the abscess cavity, eventually extending to the offending roots that are responsible for the pathosis. This will allow compartmentalized areas of inflammatory exudates and infection to be disrupted and evacuated.
- To promote drainage, the wound should be kept clean with warm saltwater mouth rinses. Intraoral heat application to infected tissues results in a dilation of small vessels, which subsequently intensifies host defenses through increased vascular flow.[36,92]
- A drain should be placed to prevent the incision from closing too early. The preferable type of drain is ½-inch iodoform gauze, which is more comfortable and less traumatic to the patient.(Fig. 18-5). The patient should be seen the following day to remove the drain.
- In many cases, the endodontic treatment can be completed in one visit after the drain is placed. The drainage allows for the ability to dry the instrumented canal, and completing the endodontic treatment eliminates the source of the infection, enabling the periapical lesion to heal quicker.

A diffuse swelling may develop into a life-threatening medical emergency. Because the spread of infection can traverse between the fascial planes and muscle attachments, vital structures can be compromised and breathing may be impeded. Two examples are Ludwig's angina and cervical fasciitis.[32] It is imperative that the clinician be in constant communication with the patient to ensure that the infection does not worsen and that medical attention is provided as necessary. Antibiotics and analgesics should be prescribed, and the patient should be monitored closely for the next several days or until there is improvement. Individuals who show signs of toxicity, elevated body temperature, lethargy, central nervous system (CNS) changes, or airway compromise should be referred to an oral surgeon or medical facility for immediate care and intervention.

SYMPTOMATIC TEETH WITH PREVIOUS ENDODONTIC TREATMENT

The emergency management of teeth with previous endodontic treatment may be technically challenging and time consuming. This is especially true in the presence of extensive restorations, including posts and cores, crowns, and bridgework. However, the goal remains the same as for the management of necrotic teeth: remove contaminants from the root canal system and establish patency to achieve drainage.[85] Gaining access to the periapical tissues through the root canals may require removal of posts and obturation materials, as well as negotiating blocked or ledged canals. Failure to complete root canal debridement and achieve periapical drainage may result in continued painful symptoms, in which case a trephination procedure or apicoectomy may be indicated. The ability, practicality, and feasibility to adequately retreat the root canal system must be carefully assessed before the initiation of treatment, as conventional retreatment might not be the optimal treatment plan. This is further discussed in Chapter 8.

LEAVING TEETH OPEN

On rare occasions, canal drainage may continue from the periapical spaces (Fig. 18-6). In these cases, the clinician may opt to step away from the patient for some time to allow the drainage to continue and hopefully resolve on the same treatment visit.[103]

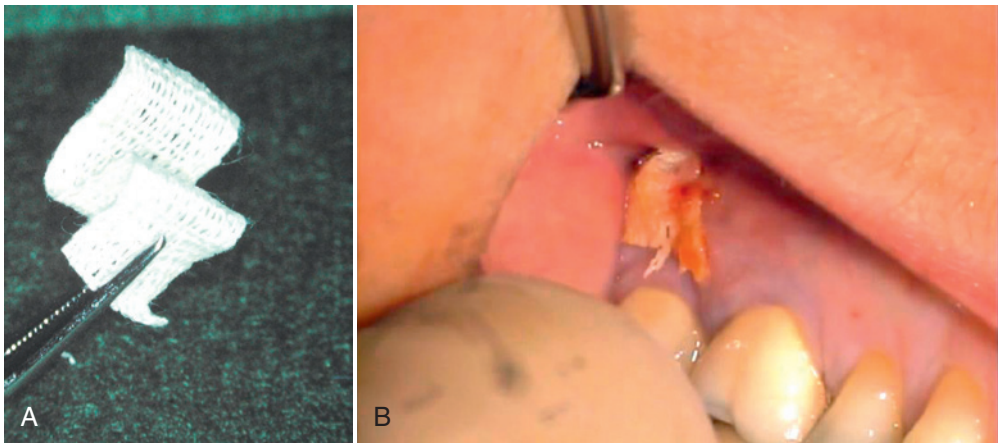

FIG. 18-5 A, Iodoform gauze cut to proper length. B, Iodoform gauze drain after 24 hours.

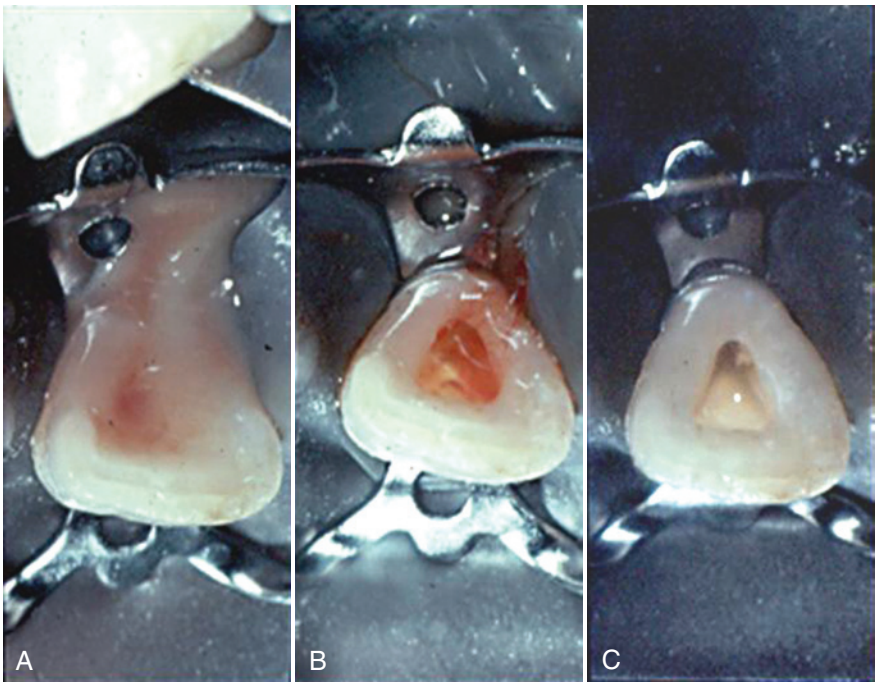

FIG 18-6 Nonvital infected tooth with active drainage from the periapical area through the canal. A, Access opened and draining for 1 minute. B, Drainage after 2 minutes. C, Canal space dried after 3 minutes.

Historically, in the presence of acutely painful necrotic teeth with no swelling or diffuse swelling, 19.4% to 71.2% of surveyed endodontists would leave the tooth open between visits.[22,23] However, the more current literature makes it clear that this form of treatment would impair uneventful resolution and create more complicated treatment.[5,7,113] For this reason, *leaving teeth open between appointments is not recommended*. Foreign objects have been found in teeth left open for drainage (Fig. 18-7). There has even been a documented case report of a foreign object being found to enter the periapical tissues through a tooth that had been left open for drainage.[95] In addition, leaving a tooth open provides an opportunity for oral microorganisms to invade and colonize the root canal system if the tooth is left open for an extended period.

SYSTEMIC ANTIBIOTICS FOR ENDODONTIC INFECTIONS

A hundred years ago, infectious diseases were the major recognized causes of death in the world. The advent of antibiotics resulted in a significant decline in the incidence of life-threatening infections and heralded a new era in the therapy of infectious diseases; but the enthusiasm generated turned out to be premature. Over the years, microbial evolutionary responses to the selective pressure exerted by antibiotics have resulted in microbial species resistant to virtually every known antibiotic agent.[41] The rapid emergence of resistant microbial strains comes as a consequence of the astonishing power of natural selection among microorganisms. If a given member of

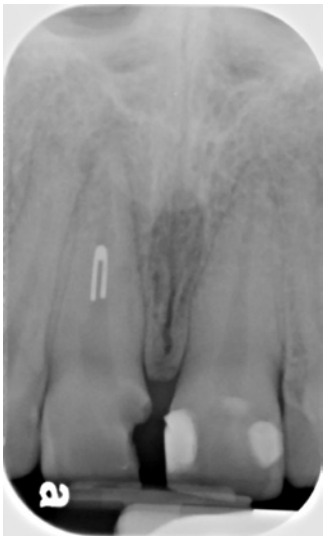

FIG. 18-7 Foreign object in tooth left open to drain. Patient used a sewing needle to clear out food particles that were blocking the canal and broke the needle in the tooth.

a microbial community possesses genes of resistance against a certain antibiotic and the community is persistently exposed to the drug, the resistant microorganism is selected to emerge and multiply to the detriment of the susceptible portion of the community. Passing on the genes responsible for resistance via plasmids and quorum sensing[41] has also been shown to encourage the survival of the microbial community. The emergence of multidrug-resistant strains of several bacterial species capable of causing life-threatening infections has been reported.[41,66,82,100,114] Antibiotic resistance among obligately anaerobic bacteria is increasing, with resistance to penicillins, clindamycin, and cephalosporins noted at community hospitals and major medical centers.[42,43]

Among oral bacteria, there have been reports on emerging resistance to commonly used antibiotics. Resistance has been found in *F. nucleatum* strains for penicillin, amoxicillin, and metronidazole, in *P. intermedia* for tetracycline and amoxicillin, and in *A. actinomycetemcomitans* for amoxicillin and azithromycin.[59,107] Macrolides (erythromycin and azithromycin) have presented decreased activity against *Fusobacterium* and nonpigmented *Prevotella* species.[44,58,59] Production of beta-lactamase by oral bacteria has also been reported, with the most prominent beta-lactamase-producing bacteria belonging to the anaerobic genus *Prevotella*.[9,12,27,34,108] Kuriyama and colleagues[58] revealed that beta-lactamase production was detected in 36% of the black-pigmented *Prevotella* and 32% of the nonpigmented *Prevotella* species isolated from pus samples of oral abscesses. Susceptibility of *Prevotella* strains to several cephalosporins, erythromycin, and azithromycin has been found to correlate with amoxicillin susceptibility; amoxicillin-resistant strains can be similarly resistant to these other antibiotics.[59] This finding suggests that there is little value in the use of oral cephalosporins and macrolides in managing endodontic abscesses, particularly when penicillin-resistant strains are evident. Other enzyme-producing oral anaerobic species include strains of *F. nucleatum, P. acnes, Actinomyces* species, and *Peptostreptococcus* species.[12,27,34,108] Facultative bacteria such as *Capnocytophaga* and *Neisseria* species have also been detected among the beta-lactamase producers.[34] Bacteria that produce beta-lactamases protect not only themselves but also other penicillin-susceptible bacteria present in a mixed community by releasing free beta-lactamase into the environment.[11]

Overuse and misuse of antibiotics has been considered the major cause for the emergence of multidrug-resistant strains. Improper use of antibiotics includes use in cases with no infection, erroneous choice of the agent, dosage or duration of therapy, and excessive use in prophylaxis.[80,81] Antibiotics are used in clinical practice far more often than necessary. Antibiotic therapy is actually warranted in about 20% of the individuals who are seen for clinical infectious disease, but they are prescribed up to 80% of the time. To complicate matters further, in up to 50% of cases, recommended agents, doses, or duration of therapy are incorrect.[66]

The appalling rise in the frequency of multidrug resistance among leading pathogens should cause great concern and incite a commitment to act carefully and responsibly. A single erroneous use of antibiotics can be a significant contribution to the current scenario of increasing microbial resistance. Diseases that were effectively treated in the past with a given antibiotic may now require the use of another drug, usually more expensive and potentially more toxic, to achieve effective antimicrobial treatment. Unfortunately, even the new drug may not be effective.

Antibiotics are defined as naturally occurring substances of microbial origin or similar synthetic (or semisynthetic) substances that have antimicrobial activity in low concentrations and inhibit the growth of or kill selective microorganisms. The purpose of antibiotic therapy is to aid the host defenses in controlling and eliminating microorganisms that temporarily have overwhelmed the host defense mechanisms.[80] Based on the earlier discussion, it becomes clear that the most important decision in antibiotic therapy is not so much *which* antibiotic should be employed but *whether* antibiotics should be used at all.[79] One should bear in mind that antibiotics are very useful drugs classically employed to treat or help treat infectious disease and provide prophylaxis in carefully selected cases.

The majority of infections of endodontic origin are treated without the need for antibiotics. As mentioned, the absence of blood circulation in a necrotic pulp prevents antibiotics from reaching and eliminating microorganisms present in the root canal system; therefore, the source of infection is often unaffected by systemic antibiotic therapy. Antibiotics can, however, help impede the spread of the infection and development of focal infections in medically compromised patients and provide a valuable adjunct for managing selected cases of endodontic infection. In addition to the indications for systemic antibiotics discussed earlier for acute abscesses and cellulitis, antibiotics are also prescribed for prophylaxis in medically compromised patients during routine endodontic therapy, in some cases of persistent exudation not resolved after revision of intracanal procedures, and after the replantation of avulsed teeth.

Selection of antibiotics in clinical practice is either empirical or based on the results of microbial sensitivity tests. For diseases with known microbial causes, empirical therapy may be used. This is especially applicable to infections of endodontic origin, because culture-dependent antimicrobial tests of anaerobic bacteria can take too long to provide results about their susceptibility to antibiotics (7 to 14 days).

Therefore, it is preferable to opt for an antimicrobial agent whose spectrum of action includes the most commonly detected bacteria. Most of the bacterial species involved with endodontic infections, including abscesses, are susceptible to penicillins,[6,51,55,59] which make them first-line drugs of choice. Because the use of antibiotics is restricted to severe infections or prophylaxis, it seems prudent to use amoxicillin, a semisynthetic penicillin with a broad spectrum of antimicrobial activity and one that is well absorbed in the alimentary canal. In more serious cases, including life-threatening conditions, combining amoxicillin with clavulanic acid or metronidazole may be required to achieve optimum antimicrobial effects as a result of the extended spectrum of action to include penicillin-resistant strains.[59] In patients allergic to penicillins or in cases refractory to amoxicillin therapy, clindamycin is indicated. Clindamycin has strong antimicrobial activity against oral anaerobes.[55,57,59,61]

The risk/benefit ratio should always be evaluated prior to prescribing antibiotics. Appropriately selected patients will benefit from systemically administered antibiotics. A restrictive and conservative use of antibiotics is highly recommended in endodontic practice. Indiscriminate use (including cases of a reversible or irreversible pulpitis) is contrary to sound clinical practice, as it may cause a selective overgrowth of intrinsically resistant bacteria, predisposing patients to secondary and super-infections, rendering drugs ineffective against potentially fatal medical infectious diseases.

ANALGESICS

As a more thorough description of pain medications can be found in Chapter 4, the following information is merely a summary of pain control using analgesics. Because pulpal and periapical pain involves inflammatory processes, the first choice of analgesics is nonsteroidal anti-inflammatory drugs (NSAIDs).[63] However, no pain medication can replace the efficacy of thoroughly cleaning the root canal system to rid the tooth of the source of infection.[35]

Aspirin has been used as an analgesic for more than 100 years. In some cases, it may be more effective than 60 mg of codeine[18]; its analgesic and antipyretic effects are equal to those of acetaminophen, and its anti-inflammatory effect is more potent.[21] However, aspirin's side effects include gastric distress, nausea, and gastrointestinal ulceration. In addition, its analgesic effect is inferior to that of ibuprofen, 400 mg. When NSAIDs and aspirin are contraindicated, such as in patients for whom gastrointestinal problems are a concern, acetaminophen is the preferred nonprescription analgesic. A recommended maximum daily dose of 4 g of acetaminophen is currently in force, and a further reduction of this dosage has been proposed to reduce the chance of acetaminophen-related liver toxicity.[62,93]

For moderate to severe pain relief, ibuprofen, an NSAID, has been found to be superior to aspirin (650 mg) and acetaminophen (600 mg) with or without codeine (60 mg). Also, ibuprofen has fewer side effects than the combinations with opioid.[18,52] The maximal dose of 3.2 g in a 24-hour period should not be exceeded. Patients who take daily doses of aspirin for its cardioprotective benefit can take occasional doses of ibuprofen; however, it would be prudent to advise such patients to avoid regular doses of ibruprofen.[1] These patients would gain more relief by taking a selective cyclooxygenase (COX)-2 inhibitor, such as diclofenac or celecoxib.

Because of their anti-inflammatory effect, NSAIDs can suppress swelling to a certain degree after surgical procedures. The good analgesic effect combined with the additional anti-inflammatory benefit make NSAIDs, especially ibuprofen, the drug of choice for acute dental pain in the absence of any contraindication to their use. Ibuprofen has been used for more than 30 years and has been thoroughly evaluated.[21] If the NSAID alone does not have a satisfactory effect in controlling pain, then the addition of an opioid may provide additional analgesia. However, in addition to other possible side effects, opioids may cause nausea, constipation, lethargy, dizziness, and disorientation.

LABORATORY DIAGNOSTIC ADJUNCTS

Chapter 14 discusses culturing techniques and indications. Because the results of culturing for anaerobic bacteria usually require at least 1 to 2 weeks, it is not considered routine in the management of an acute endodontic emergency. Thus, in an endodontic emergency, antibiotic treatment, *when indicated* (see Chapter 18), should begin immediately, because oral infections can progress rapidly.

FLARE-UPS

An endodontic flare-up is defined as an acute exacerbation of a periradicular pathosis after the initiation or continuation of nonsurgical root canal treatment.[3] The incidence may be from 2% to 20% of cases.[50,70,76,109] A meta-analysis of the literature, using strict criteria, showed the flare-up frequency to be about 8.4%.[105] Endodontic flare-ups are more prevalent among females under the age of 20 years and may occur more in maxillary lateral incisors; in mandibular first molars, when there are large periapical lesions; and in the retreatment of previous root canals.[102] The presence of pretreatment pain may also be a predictor of potential posttreatment flare-ups.[50,102,109] Fortunately, there is no decrease in the endodontic success for cases that had a treatment flare-up.[54]

Endodontic flare-ups may occur for a variety of reasons, including preparation beyond the apical terminus, overinstrumentation, pushing dentinal and pulpal debris into the periapical area,[36] incomplete removal of pulp tissue, overextension of root canal filling material, chemical irritants (such as irrigants, intracanal medicaments, and sealers), hyperocclusion, root fractures, and microbiologic factors.[94] Although many of these cases can be pharmacologically managed (see Chapter 18), recalcitrant cases may require periapical surgery, reentry into the tooth, the establishment of drainage either through the tooth or via trephination, or, at a minimum, adjustment of the occlusion.[19,89,94] The prophylactic use of antibiotics to decrease the incidence of flare-ups has been met with some controversy. Whereas earlier investigators[71] found that antibiotic administration before treatment of necrotic teeth decreased the incidence of flare-ups, more recent studies found antibiotic use either less effective than analgesics or to have no effect in reducing interappointment emergencies or posttreatment symptoms.[86,110,101]

CRACKED AND FRACTURED TEETH

Described in detail in Chapter 1 and Chapter 21, cracks and incomplete fractures can be difficult to locate and diagnose,

but their detection can be an important component in the management of an acute dental emergency. In the early stages, cracks are small and difficult to discern. Removal of filling materials, applications of dye solutions, selective loading of cusps, transillumination, and magnification are helpful in their detection. As the crack or fracture becomes more extensive, it can become easier to visualize. Because cracks are difficult to find and their symptoms can be so variable, the name *cracked tooth syndrome* has been suggested,[14] even though it is not truly a syndrome. Cracks in vital teeth often exhibit a sudden and sharp pain, especially during mastication. Cracks in nonvital or obturated teeth tend to have more of a "dull ache" but can still be sensitive to mastication.

The determination of the presence of a crack or fracture is paramount because the prognosis for the tooth may be directly dependent on the extent of the crack or fracture. Management of cracks in vital teeth may be as simple as a bonded restoration or a full coverage crown. However, even the best efforts to manage a crack may be unsuccessful, often requiring endodontic treatment or extraction. Fractures in nonvital or obturated teeth may be more challenging. In addition, it must be determined whether the crack or fracture was the cause of pulpal necrosis and whether there has been extensive periodontal breakdown. If so, the prognosis for the tooth is generally poor; thus extraction is recommended.

SUMMARY

The management of endodontic emergencies is an important part of a dental practice. It can often be a disruptive part of the day for the clinician and staff, but it is an invaluable solution for the distressed patient. Methodical diagnosis and prognostic assessment are imperative, with the patient being informed of the various treatment alternatives.

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CHAPTER 19

Managing Iatrogenic Endodontic Events

YOSHITSUGU TERAUCHI

CHAPTER OUTLINE

Sodium Hypochlorite (NaOCl)
Instrument Separation
Causes of Instrument Separation
Management of Separated Instruments
Conditions for Separated Instrument Removal Attempts
Separated Instrument Removal Technique
Prognosis
Ledge Formation
Causes of Ledge Formation
Management of Ledge Formation
Bypassing a Ledge
Potential Complications of Removal or Bypassing a Ledge
Prevention of Ledge Formation
Prognosis
Perforations (Nonsurgical)
Radicular Extrusion of Root Canal Filling Materials
Causes of Extrusion of Obturation Materials Beyond the Radicular Foramen
Management of Obturation Materials Extruded Beyond the Radicular Foramen
Management of Tissue Damage Caused by Extrusion of Root Canal Filling Materials
Sinus Perforation
Inferior Alveolar Nerve (IAN) Injury
Nonsurgical Causes of IAN Injury
Surgical Causes of IAN Injury
Prognosis
Prevention of Complications Due to Nerve Injury
Cervicofacial Subcutaneous Emphysema

An iatrogenic event is defined as a procedure "induced inadvertently by a physician or surgeon" (Merriam-Webster dictionary). This is true for dentistry as well, with cases of endodontic malpractice being among the most prevalent in dentistry.[57,111,252] These occurrences are not limited to the United States, with other countries reporting similar percentages of endodontic malpractice claims.[225]

Although iatrogenic accidents cannot be prevented with absolute certainty, their occurrences can be greatly reduced with the aid of the "Three Ts": training, technique, and technology. Incumbent with the first (training) is expertise with the other two factors, especially regarding technology and, more specifically, the dental operating microscope. It has proved itself indispensable in locating canals,[155,157,250,277] removing separated instruments,[279] repairing perforations,[234] and enhancing the delivery of quality care.[150] The superior visualization permits more accurate clinical evaluations, allowing for a precise assessment of the challenge and the appropriate treatment plan for its resolution. This instrument is indispensable for all of the treatment scenarios that follow, which are therefore assumed to be performed with the aid of magnification.

SODIUM HYPOCHLORITE (NaOCl)

Often happening without warning, the progression of events following sodium hypochlorite extrusion into periradicular tissues is both rapid and startling to witness. In addition to the physical outcomes, the experience is likely to leave the patient wary of both the procedure and practitioner.

Although the properties and methods of delivering sodium hypochlorite are covered in Chapter 6, the gravity of the consequences of its misuse merits a brief highlight. Sodium hypochlorite is an effective irrigating solution in concentrations ranging from 0.5% to 6%[127,317] and is able to disrupt the biofilm,[45] dissolve necrotic tissues,[184] and remove organic components of the smear layer.[17] However, it is extremely cytotoxic, irrespective of the tissue it contacts,[202] with concentrations as low as 0.01% lethal to fibroblasts in vitro.[116] NaOCl injection into vital tissue initiates hemolysis and ulceration, damages endothelial and fibroblast cells, and inhibits neutrophil migration.[89,91] A study investigating the physical effects of NaOCl on bone ex vivo found degradation of the organic matrix collagen and no sign of living cellular contents (Fig. 19-1).[140]

The majority of reported NaOCl complications appear to be due to an inaccurate working length, iatrogenic widening of the radicular foramen, lateral perforation of the root, or wedging of the irrigation needle.[147,271] The pattern of facial distention is comparable to that encountered with a cervicofacial emphysema (discussed later), dissecting fascial spaces in a similar fashion, with the concomitant tissue destruction magnifsying the effect and impeding recovery. This swelling is further exacerbated by the presence of effervescing agents such

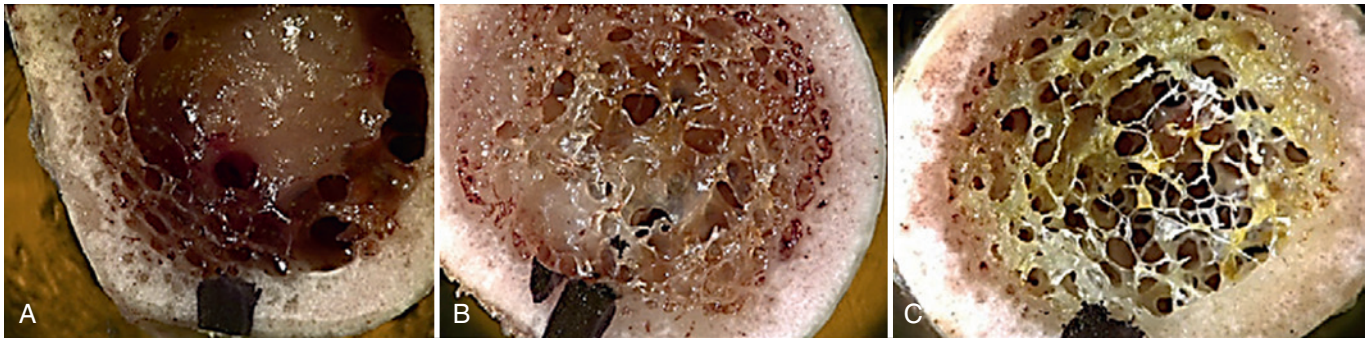

FIG. 19-1 Femur shaft cross sections (**A**) untreated bone section; (**B**) saline-treated bone; (**C**) NaOCl-treated bone section. Grossly, NaOCl caused significant changes in cancellous structure, leaving large structural craters of apparent demineralization. (From Kerbl FM, DeVilliers P, Litaker M, Eleazer PD: Physical effects of sodium hypochlorite on bone: an ex vivo study, *J Endod* 38:357, 2012.)

FIG. 19-2 **A,** Clinical presentation 3 days following the extrusion of NaOCl during treatment of the maxillary right canine. The patient had difficulty opening her right eye, and the swelling had extended to the submandibular/sublingual regions on the affected side; she also experienced limited mouth opening. There was altered sensation infraorbitally and in the region of the upper right lip, but no reported paresthesia of the alveolar or facial nerves. **B,** One month post extrusion, the ecchymosis had resolved, with a return of sensation and full function. Therapy was completed without incident. (From de Sermeño RF, da Silva LA, Herrera H: Tissue damage after sodium hypochlorite extrusion during root canal treatment, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 108:e46, 2009.)

as hydrogen peroxide, potentiating the effect of the hypochlorite and amplifying soft tissue involvement.[20,161] The initial patient reaction to the fluid insult is severe and immediate pain, with marked edema or bruising that may continue to extend over the injured side of the face, cheek, or lips (Fig. 19-2). There may be spontaneous and profuse hemorrhaging from the canal space, and, in the case of maxillary posterior teeth, the patient may report periorbital pain,[21,89] chlorine taste, or irritation of the throat.[63] The amount that has been expressed into the periradicular area, the spatial location of the fluid introduction, and the proximity to sensitive anatomic structures will dictate the severity and duration of the reaction. Paresthesia, either transient[21] or permanent,[223] can occur, especially if interventional treatment is not rendered immediately. In addition to sensory deprivation, motor nerve dysfunction secondary to irreversible chemical damage has been reported (Fig. 19-3).[206] In this case, there was distinct loss of upper lip and cheek function, with problems in the musculature. In mandibular teeth, extension into the submandibular, submental or sublingual regions can compromise the airway, compelling immediate hospitalization and surgical intervention to avert a life-threatening episode.[30]

Upon realizing that an extrusion event has occurred, it is imperative that the clinician cease further treatment and immediately engage in measures targeted at diminishing the effect of the chemical; ignoring the event reduces outcomes.[162,223] Treatment protocols are empirical and are predicated on cause and severity. With the emphasis on pain control, local

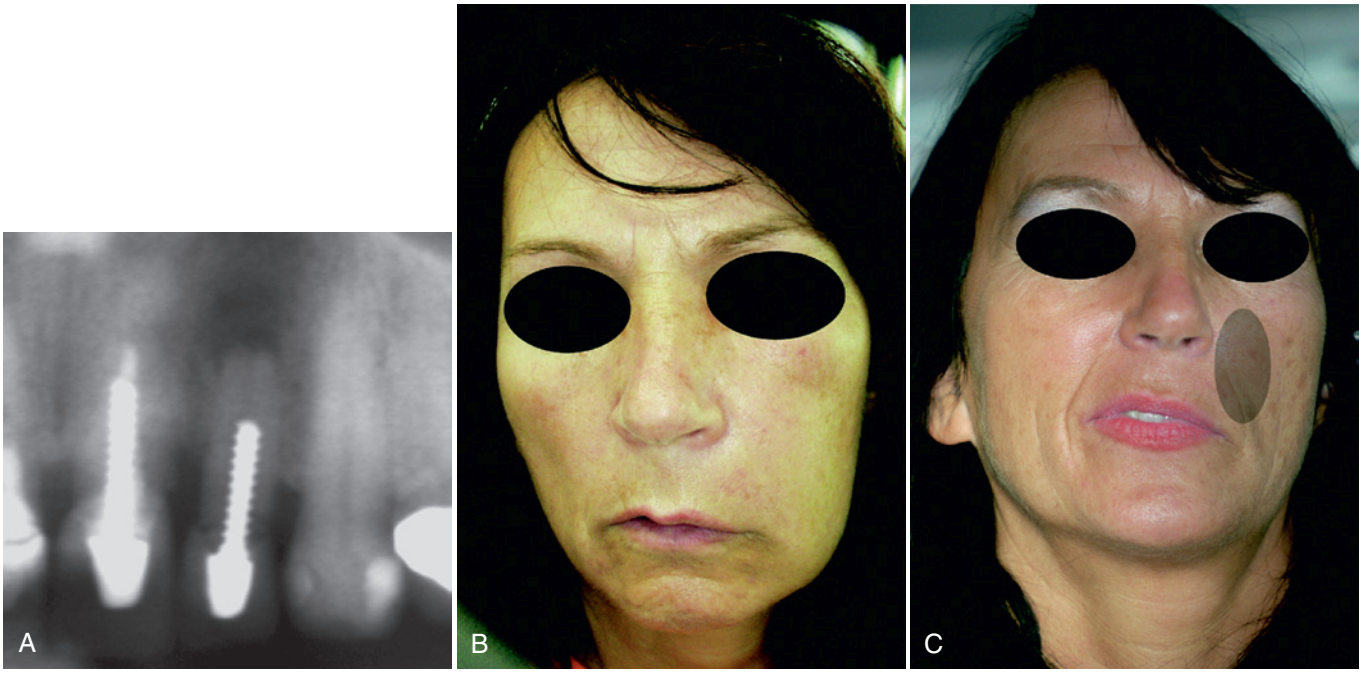

FIG. 19-3 A, Radiograph of the maxillary left lateral incisor (#10, #22) before post removal. Radicular root resection had been completed, and the canal space is empty. Prior to re-cementation, the canal was irrigated with 3% NaOCl, precipitating the extrusion event. B, Twenty-four hours post extrusion, there was altered sensation infraorbitally and from the upper left lip to the left lip corner. In addition, the buccal branch of the facial nerve was affected, as evidenced by a distinct loss of upper lip and cheek function (the corner of the mouth could not be pulled up by the mimic musculature). The mouth opening was limited to 20 mm (C), 3 years post incident. The weakness of the mimic musculature of the left face side is clearly visible. Attempts to laugh resulted in a hanging left lip corner secondary to the weakness of the motor innervation by the facial nerve. In the gray-marked area there is a permanent hypoesthesia. (From Pelka M, Petschelt A: Permanent mimic musculature and nerve damage caused by sodium hypochlorite: a case report, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 106:e80, 2008.)

anesthetics and analgesics can be administered, along with cold compresses, in an effort to moderate swelling and edema. Directed at diluting the effect of the extrusion, it has been suggested that the clinician immediately irrigate the canal with normal saline to encourage bleeding, both diluting the irritant and removing it from the site of the injury.[63,179] In addition, clinicians may immediately irrigate the canal and periradicular areas of the extrusion with saline, using a negative pressure irrigation device with the cannula placed at the radicular foramen. In one report, the subsequent reaction was short lived with this approach.[235]

After one day, warm compresses are substituted for the cold, and warm oral rinses are prescribed to stimulate the local microcirculation. Antibiotics are usually prescribed, and their administration commensurate with the severity of hard and soft tissue destruction and necrosis; milder cases can be treated with oral medications, whereas severe presentations are better controlled and modulated in a hospital setting with intravenous routes of delivery. The use of corticosteroids is equivocal; the control of the spreading edema must be balanced with the increased risk of infection. The patient should be monitored closely; a marked increase in the size or extent of the swelling or signs of impending airway obstruction mandate immediate referral to the hospital or a maxillofacial surgeon for more aggressive treatment and management. Lastly, reassuring patients that recovery, in spite of their current appearance, is usually complete and uneventful, will ease their anxiety. They should be informed as to why it occurred and what to expect during their recovery. Recovery should be monitored and documented daily until the resolution is imminent.

Prevention involves attention to detail and an appreciation for fluid dynamics. In summary, the clinician should do the following:

- Establish an accurate working length and avoid overinstrumentation/enlargement of the radicular foramen.
- If irrigating using positive pressure, employ a small side-vented needle placed no closer than 2 mm from the working length. Express the fluid slowly and observe that it is venting through the access cavity.[29]
- Carefully assess the canal integrity for signs of perforation or other large portals of fluid egress.
- Avoid wedging the needle tip in the canal space or inserting it beyond the working length.
- Confirm the identity of the solution prior to injection or irrigation.[104]

INSTRUMENT SEPARATION

The introduction of NiTi rotary instruments into endodontics revolutionized the way the root canal system is instrumented. However, with the advent of rotary NiTi files, there has also been a perceived increase in the occurrence of separated

instruments,[84,242] with some reports indicating that separation of NiTi rotary instruments occurs more frequently than hand instruments.[294] On radiographic examination, separated instruments can usually be discerned by the distinct radiopacity within the canal. However, if the canal with a separated instrument is subsequently obturated with radiopaque materials, it may not be visualized in the radiograph. Upon removal of filling materials from the canal, a separated instrument may be visualized under the dental operating microscope (DOM) or become apparent on radiographs for the first time (Fig. 19-4).

Causes of Instrument Separation

A common cause for instrument separation is improper use.[84,245] Typical examples of improper use include inadequate access, overuse of the instrument, too much radicular pressure during instrumentation,[247,291] and the continuous use of a large

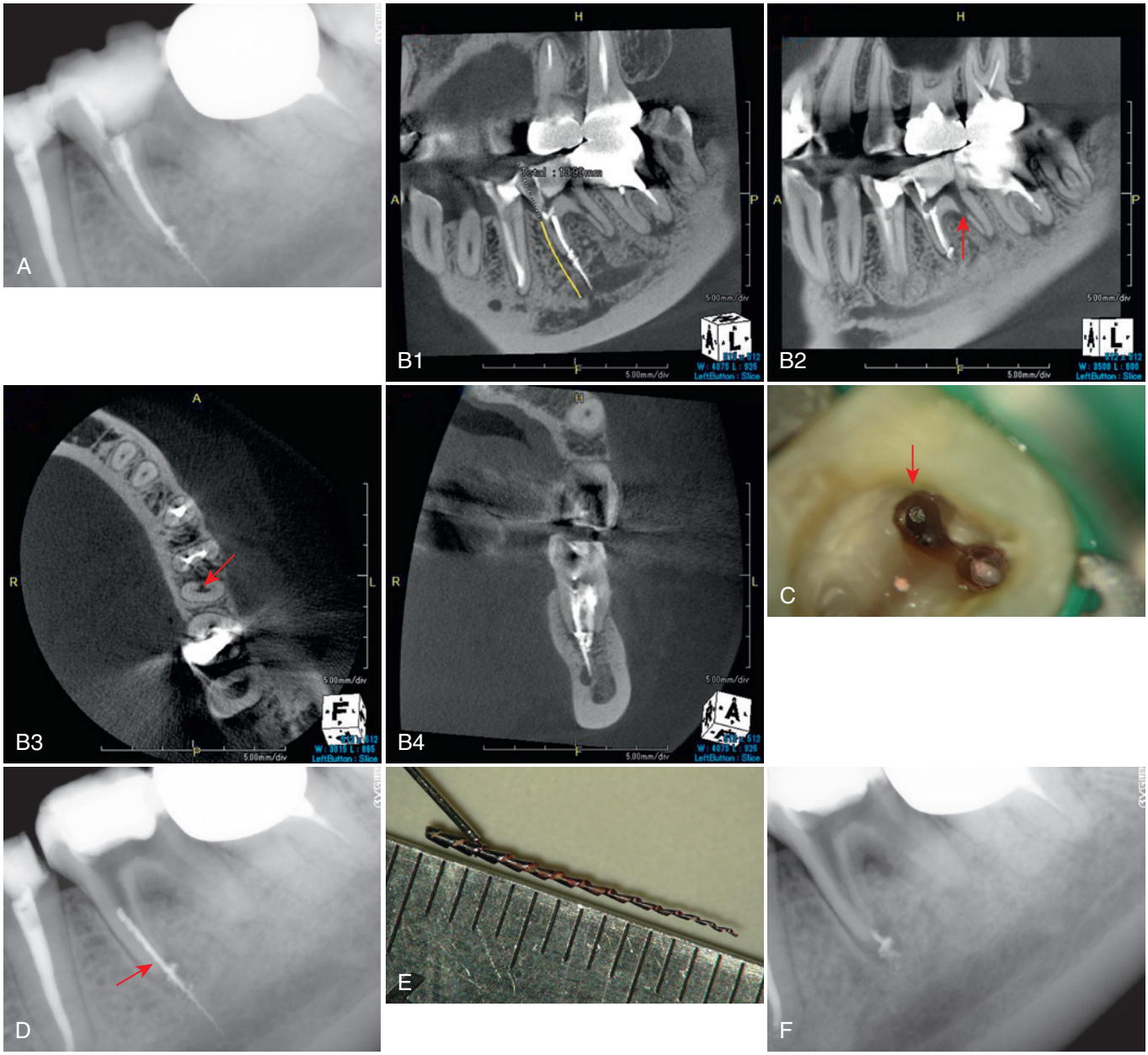

FIG. 19-4 A, Radiograph of the mandibular left first molar with a long indistinct separated instrument obviously longer than 4.5 mm in the mesial root that is extruded beyond the radicular foramen. B1, Sagittal view of CBCT showing the radicular end of the separated instrument is extended into the IAN and it measures 14 mm on it. B2, Sagittal view of CBCT showing root perforation in the distal canal (*yellow arrow*). B3, Axial view of CBCT showing the perforation in the distal canal (*yellow arrow*). B4, Coronal view of CBCT showing the radicular end of the separated instrument is in the IAN. C, Separated instrument in the mesiobuccal canal upon removal of root filling materials (*red arrow*) D, Radiograph showing removal of the root fillings in the mesiobuccal canal revealed the overall length of the separated instrument (*red arrow*). E, Retrieved separated instruments measuring 14 mm on the ruler. F, Radiograph showing completion of separated instrument removal. *Continued*

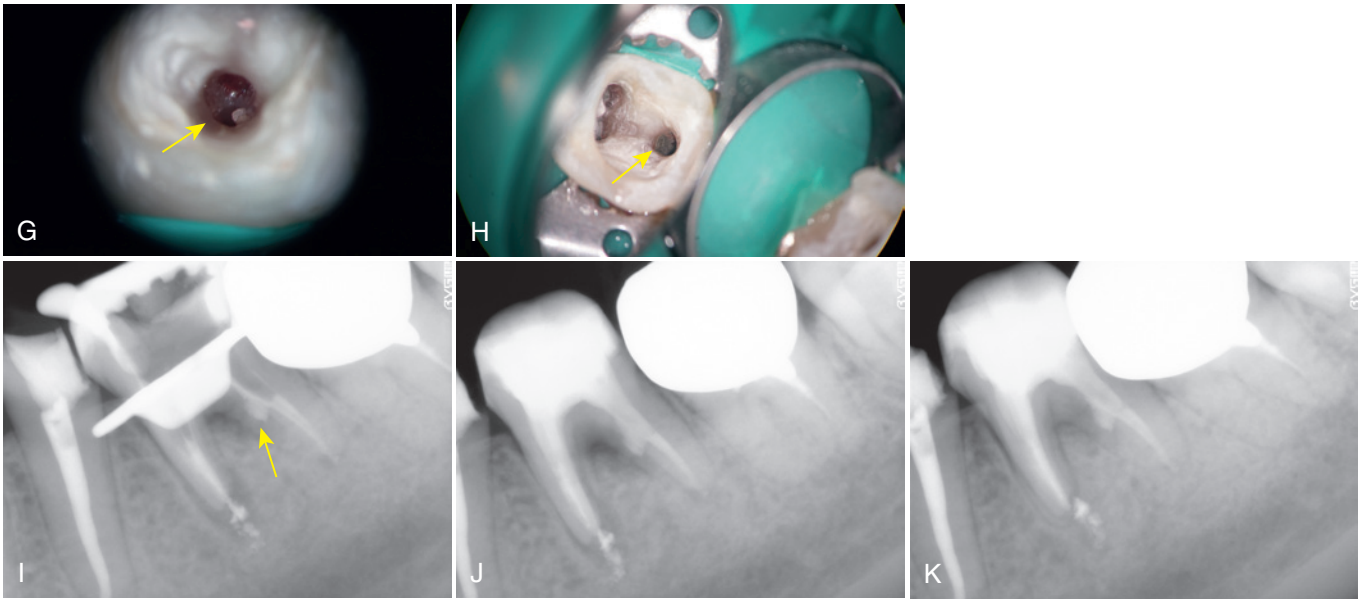

FIG. 19-4, cont'd G, Perforation site in the distal canal (*yellow arrow*). H, Perforation repaired with MTA (*yellow arrow*). I, Postoperative radiograph showing root canal fillings in all the canals and perforation repair (*yellow arrow*). J, One-month postoperative radiograph revealing the periradicular lesions still present. K, Three-month postoperative radiograph showing periradicular healing.

instrument in a curved canal.[107,219] Additional contributing factors are operator experience,[200,285] rotational speed,[53,169] canal curvature (radius),[170,219] instrument design and technique,[33,152] torque setting,[83] manufacturing process,[5] the type of NiTi alloys used,[88] the type of rotational motion (continuous rotations versus reciprocating motions),[205] the type of tooth,[51,191,258] and absence of a glide path.[204] Based on these factors, it is not surprising that the reported incidence of separated NiTi files ranges from 0.4% to 23%.[5,7,210,221,248,272]

Separated instruments seem to occur more frequently in molars than anterior teeth, especially in mandibular molars. This high incidence of instrument fracture in molars may be related to accessibility to the canal, diameter of the root canal, and root canal curvature.[51,191,258] Endodontists have reported the incidence of instrument separation to be about 5%.[5,200] The clinician must inform the patient of the instrument separation and treatment options as well as potential complications of both the separated instrument retention and its attempted removal.

Endodontic instruments fracture as a result of cyclic fatigue or torsional fatigue[47] or a combination of both types of fatigue.[245,257,296] Cyclic fatigue is caused by repetitive compressive and tensile stresses acting on the outer portion of a file rotating in a curved canal, leading to cyclic failure without previous signs of plastic deformation.[200] Torsional failure occurs when the tip of the instrument binds but the shank of the file (driven by the handpiece) continues to rotate.[245] Shear fracture of the material then occurs when the maximum strength of the material is exceeded.[154] Clinically, cyclic fatigue seems to be more prevalent in curved root canals, whereas torsional failure might happen even in a straight canal.[43,316] Cyclic-fatigued instruments are reported to become less resistant to torsional fatigue failure,[145] whereas the use of rotary instruments at a high torque setting is reported to increase the risk of cyclic fatigue failure.[84] Interestingly, the use of reciprocal motions (as opposed to rotational motions) is reported to extend the life span of an instrument[56,315] and increase cyclic fatigue resistance.[205] It is recommended that instruments designed for reciprocating motion be used for shaping a severely curved canal and that the instruments used to prepare such curved root canals be discarded after use to prevent instrument fracture.

Management of Separated Instruments

Management of separated instruments includes nonsurgical orthograde (mechanical or chemical) or surgical approaches. The mechanical removal involves the use of tools dedicated to separated instrument removal such as extractors, wire loops, post removal systems, ultrasonics, and laser irradiation (see also Chapter 8). The chemical removal involves the use of chemical solvents for instrument corrosion and electrochemical process for instrument dissolution. Surgical approaches include radicular surgery, intentional replantation, root amputation, or hemisection.

For a nonsurgical approach in removing a separated file, various methods and devices have been developed (see also Chapter 8). When a separated instrument extends above the root canal orifice, it can often be easily removed with a hemostat, Steiglitz forceps, a modified Castroviejo needle holder,[78] or a Perry plier.[304] A spoon excavator or a Caulfield retriever can also be used to engage and remove the separated instrument with coronally directed pressure. In the event that a hand file can bypass the separated instrument, a "braiding technique" can be used whereby several Hedstrom files are inserted along the bypassed instrument, the files are twisted to grasp the separated instrument, and it is then extracted as one unit. When a fractured instrument is below the canal orifice and cannot be bypassed, one basic method for removing the separated instrument requires the exposure of approximately 2 mm of the coronal portion of the separated file. This allows the use of an extracting device for engaging the file, securing it, and removing it. The Masserann kit (Micro-Mega, Besancon, France) is a

popular system for the removal of separated instruments located within the straight part of the canal. This kit involves the use of trephine burs to expose the coronal portion of the separated instrument and for creating a space for an extractor. An extractor is then used to grasp and remove the separated instrument. The limitation of this system is that it cannot be used for cases involving separated instruments located beyond the midroot or in a curved canal, as this technique involves a considerable amount of dentin sacrifice that can weaken the root structure and increase the risk of perforation.[313] An alternative to the Masserann kit is the Endo Safety System.[306] This system differs from the Masserann kit in that it uses smaller diameter trephine burs and the extractor has its own unique mechanism for grasping instruments. The Endo Extractor (Brasseler Inc., Savannah, GA) can also be used in these types of cases. This system consists of a trephine bur to expose the coronal portion of the separated instrument and a hollow tube extractor that is threaded over the exposed portion of the separated instrument and bonded to it with cyanoacrylate adhesive.[92] The Canal Finder System (FaSociété Endo Technique, Marseilles, France) or the EndoPuls System (EndoTechnic, San Diego, CA) provides a different approach to a separated instrument. The system is composed of a handpiece and files that are designed exclusively for the system.[164] The system produces a vertical movement with a maximum amplitude of 1 to 2 mm, which decreases when the speed increases.[126] This vertical movement of the file will also help bypass an instrument fragment. The flutes of the file can mechanically engage with the separated instrument, and with the vertical vibration, the instrument fragment can be loosened or even retrieved.[125] The mechanical movement of the file can get aggressive, so the clinician must use caution to avoid root perforation, especially in a curved canal. These systems are technique sensitive and therefore results can vary between cases.

Ultrasonic instruments are very effective for the removal of separated instruments.[42,185,241] However, it is important to realize that separated NiTi files tend to fracture repeatedly when ultrasonics are applied to them for broken file removal, whereas stainless steel instruments are more resistant and more easily removed with ultrasonics than NiTi instruments.[241,279,301] Small ultrasonic instruments allow continuous and improved vision of the field of operation. The use of ultrasonics, especially when performed under the microscope, enhances the removal process and can provide relatively safe removal of separated instruments.[191,279,300] The ultrasonic tip is placed on the staging platform between the exposed portion of the separated file and the canal wall, and it is vibrated around it in a counterclockwise direction, applying an unscrewing force to the separated file. This technique will help with removing most rotary instruments that have a clockwise cutting action. If it is known that the separated file has a counterclockwise cutting action, then a clockwise rotation will be needed. The energy applied will aid in loosening the file, and occasionally the file will suddenly exit out of the canal.

However, the use of ultrasonics does have some drawbacks. Upon ultrasonic activation, a separated file can be accidentally extruded into the bone, either radicularly or through a perforation. In addition, the ultrasonic activation of a separated instrument has been reported to separate small fragments from the original fragment,[41] further complicating the management of the case. Smaller fragments of a file are considered more difficult to remove than longer fragments.[170,219,241] These smaller fragments of a separated file can often be pushed back into a more radicular and narrow level of the canal, which would eventually increase the risk of perforation and extra dentin sacrifice upon attempted fragment removal.

The success rate for removing separated files varies from 33% to 95%[7,51,221,240,301] with the time required for using ultrasonic techniques varying between 3 to over 60 minutes.[9,185,285] The variations in success rates are due to the location of fractured instruments, the diameter of the root canal,[258] the degree of canal curvature,[9,220,268] the radius of curvature,[9,219] operator experience,[9,285] operator fatigue, and the length of the separated instrument.[284] Visualization and accessibility to the separated instrument play a major role in file retrieval.[51] In general, the success rate for removal is high for separated files located before the canal curvature, moderate for those located at the curvature, and low for those located beyond the curvature.[170,305] The success rate is also more favorable when the canal curvature is less and the radius of curvature is longer.[9] The combination of ultrasonic techniques and microscopes typically improves success rates in file removal.[51,82,191,301]

It has been recommended that removal attempts of separated instruments from root canals should not exceed 45 to 60 minutes because the success rates may drop with increased treatment time.[279] The reduced success rate could be related to operator fatigue or overenlargement of the canal, which compromises the integrity of the tooth and increases the risk of perforation. Any case where the separated instrument can be visualized under the DOM may be considered less challenging to manage. If the separated instrument is considered irretrievable after a sufficient attempt, the case should be completed by having the separated file incorporated as part of the obturation and kept under observation.[74,79,221,269]

Conditions for Separated Instrument Removal Attempts

File removal can be attempted in either a dry or a wet environment. Dry conditions provide better visibility when using the dental operating microscope, preventing fewer additional procedural accidents.[242,268,279,300] However, heat generation by ultrasonics is inevitable,[96,113,156,173,258,268,279,300] and a temperature rise above 10° C on the external root surface can damage the periodontal tissues.[66,280] In addition, the separated instrument will be susceptible to secondary heat when ultrasonic tips are in contact with the file.[286,300] Therefore, it is recommended that water or some other irrigant be used while the ultrasonic tip is activated at the lowest possible power setting.[37,70,175,300] In addition, the irrigating solution used in the canal should be EDTA solution; the use of an ultrasonic tip as an adjunct with EDTA solution enhances the canal wall cleanliness.[256]

Separated Instrument Removal Technique

The majority of separated instruments are NiTi,[272] with the mean length of separated instruments being 2.5 to 3.5 mm.[135] Many NiTi rotary instruments used have a .04 to .08 taper range with a tip size of #20 to #30, resulting in a coronal diameter of about 0.30 mm to 0.58 mm. If the coronal diameter of the separated instrument is smaller than 0.45 mm, then a #3 modified Gates Glidden bur (#3 MGG bur from the FRK, DentalCadre, Seattle, WA) should be used to enlarge the canal to the separated instrument, followed by the micro-trephine bur (DentalCadre) for exposing the coronal portion of the

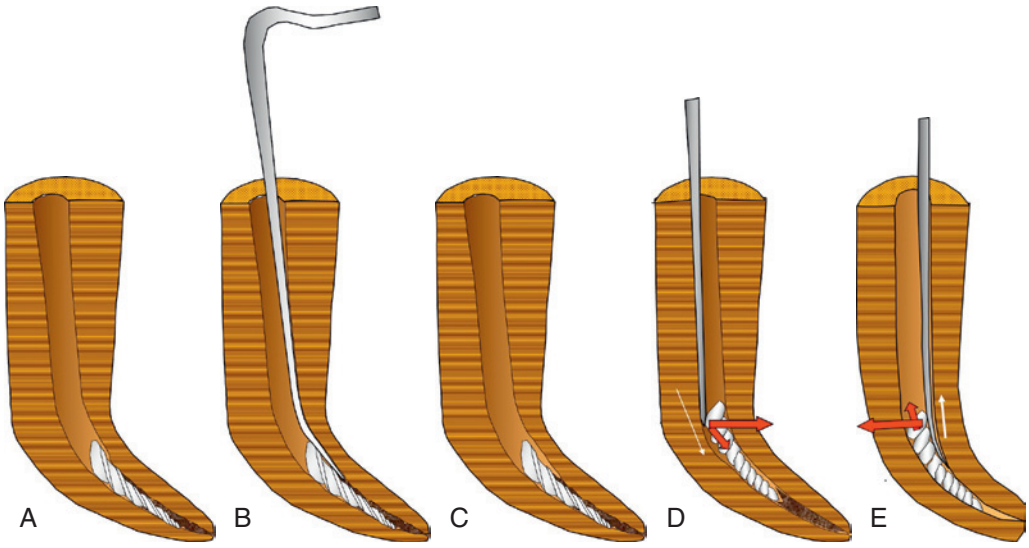

FIG. 19-5 A, Separated file around the curve after the canal enlargement with a NiTi rotary file. B, Space needs to be created on the inside curve with the spoon ultrasonic tip, followed by the straight tip. C, Thin space created on the inner curve. D, Ultrasonic forces applied to the separated instrument from the outside curvature will reorient it in a radicular direction. E, Ultrasonic forces applied to the separated instrument from the inside curvature will reorient it in a coronal direction rather than a radicular direction.

separated instrument, with less risk than ultrasonics for secondary fracture of the separated instrument.[285,286] The inner diameter of the micro-trephine bur is 0.45 mm and it is then used, in a counter clockwise rotation, with the intent of unscrewing the separated instrument. This bur should be used at 600 rpm in wet conditions; if the rotational speed exceeds 600 rpm, unintentional ledge formation may occur, especially in a curved canal.

If the coronal diameter of the separated instrument is larger than 0.45 mm or the canal curvature is greater than 15 degrees, a larger NiTi rotary instrument, with tip size 0.1 mm (two sizes) larger than the coronal diameter of the separated instrument, should be used to enlarge the canal coronal to the separated instrument, followed by ultrasonic instruments to expose the coronal portion.

Firstly, the spoon tip with the concave portion facing the separated instrument (DentalCadre) will be introduced into the thin gap between the separated instrument and the inside curve of the canal to create one quarter space of the circle around the separated instrument. Secondly, the straight tip (DentalCadre) is introduced to expand the space to complete the semicircular space on the inside curve. The ultrasonic tip should always be applied to the area on the inner canal curve being extended in the radicular direction (Fig. 19-5). If the ultrasonic tip is placed in the space outside the curve and ultrasonic energy is given to the separated instrument from the outside of the curve, the force applied will redirect the separated instrument in an apical direction, eventually pushing it more apically and making the instrument retrieval more difficult, whereas the force applied from the inside of the curve to the separated instrument will consequently reorient the separated instrument in a coronal direction. In most cases, the coronal one third of the separated file is the main portion engaged in the canal wall. This portion is typically resistant to mechanical force for disengagement due to the highly stressed concentration on the coronal one third of the separated file. Therefore, the dentin wall contacting the coronal third of the separated file has to be eliminated to free it and facilitate its removal (Fig. 19-6). It is recommended that the ultrasonic tip be used in a counter clockwise direction to trough around the coronal aspect of a separated instrument.[240] However, a fatigued separated instrument is already extremely susceptible to secondary fracture by ultrasonic activation.[286,300]

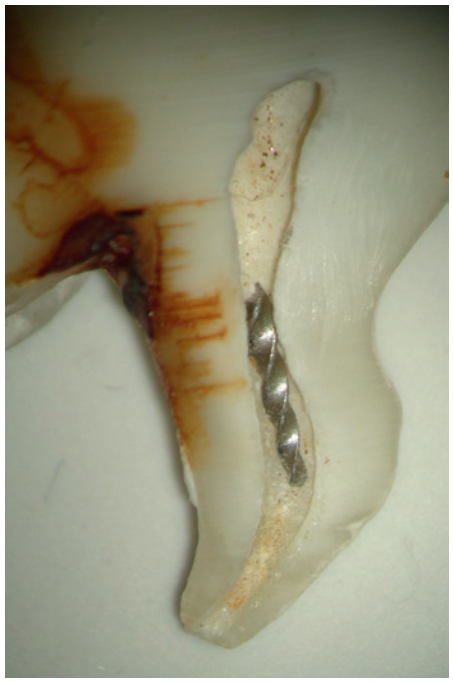

FIG. 19-6 The coronal one third of the file length is typically the part to be stuck in the canal wall, whereas the radicular portion of the file is typically not in contact with the canal wall. Stress is concentrated mostly on the coronal one third of the file length.

For this reason it is safer to cut only a semicircular trough on one side of the separated instrument behind which there is dentin wall support,[286] and a pulsing motion should be applied to it while ultrasonics is activated to prevent instrument fracture and temperature rise, especially in dry conditions.[66,280,286,300] Just a thin space created on one side of the coronal third of the separated instrument is enough to free the separated instrument from being engaged in the canal wall. Especially in a curved canal, ultrasonic activation should always apply to the thin space created between the separated fragment and the inside curve, and the dentin wall supporting the separated file fragment must always be present behind the ultrasonic activation site to prevent secondary fracture. The radicular extent to be created along the separated instrument should be approximately as long as one third of the fragment length. For example, if the separated file fragment is 3 mm long, the semicircular space needs to be extended radicularly about 1 mm on the inside curve to help the ultrasonic tip loosen it. This process of extending the space in an apical direction must continue until it is confirmed that the separated file is seen dancing with ultrasonics under the DOM. The root canal preparation for instrument retrieval is done when the movement of the separated instrument is confirmed. The ultrasonic tip used for this purpose should be as thin as possible, allowing the clinician to both visualize the operative field and prevent overenlargement of the canal wall. During this ultrasonic preparation phase, the separated file may come out by ultrasonic activation at any moment (Fig. 19-7).

When the separated instrument is not visible upon the straight line access to it, instrument retrieval is considered challenging and it requires expertise and a little bit of guesswork to remove it. First, the microexplorer (DentalCadre) is precurved to meet the separated instrument, and then radiographs are taken with the microexplorer in the canal to confirm that it is in the space between the separated instrument and the inside curve. Next, the straight tip is precurved the same way as the microexplorer was precurved to get into the inner thin space, and the ultrasonic tip is activated to extend the space apically until the separated instrument gets loosened (Fig. 19-8).

Once the root canal preparation for instrument retrieval is completed, it is important to confirm that the canal wall is smooth from the separated file to the coronal extent, with no protrusions on the outside canal wall. Following the ultrasonic loosening of the coronal portion of the separated instrument, the canal is filled with EDTA solution to enhance the ultrasonic effect (see Fig. 19-7, *B*). A retrospective study and the author's empirical findings show that when the separated instrument is shorter than 4.5 mm, the clinician will most likely be able to retrieve it with ultrasonics alone (Fig. 19-9).[284] Ultrasonic activation should be applied in push-pull motions in the space created between the separated file and the inside curvature of the canal until the separated file is removed (see Fig. 19-7, *C* and *D*).

If the separated instrument is longer than 4.5 mm, or if it is seen moving with ultrasonic activation, but the instrument retrieval with ultrasonics was not successful, then the loop device (DentalCadre, Seattle, WA) should be applied to it to capture the coronal portion of the separated file and pull the file fragment out of the canal.[284] The probability is that an instrument fragment longer than 4.5 mm is touching all over the canal walls, which creates more friction as it is reoriented in a coronal direction and makes it more resistant to instrument retrieval with ultrasonics alone, requiring more mechanical force to pull it out of the canal. The coronal portion of the separated instrument needs to be peripherally exposed by at least 0.7 mm (see Fig. 19-10, *A*), and a semicircular space needs to be created and extended apically on the inside curvature of the canal the same way as in the root canal preparation for ultrasonic instrument retrieval until the separated instrument is seen dancing with ultrasonics. The loop size must be adjusted to fit the coronal diameter of the separated file using

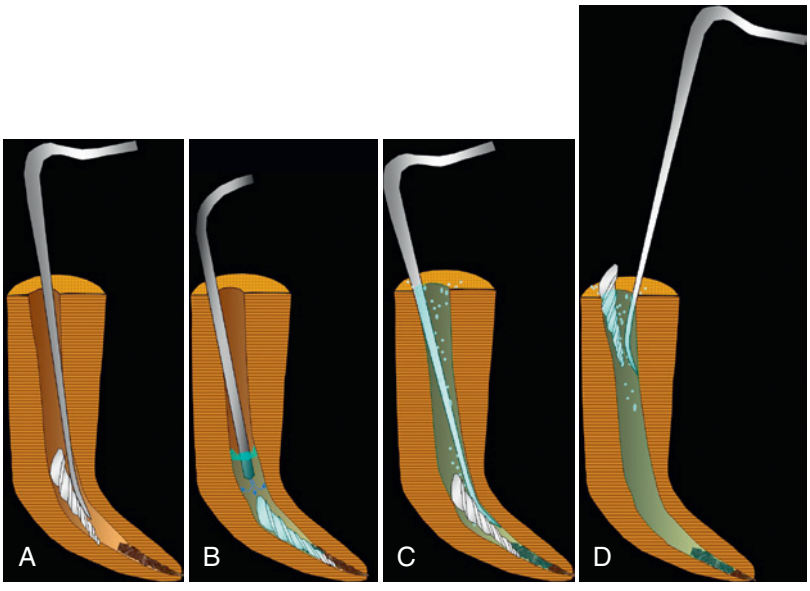

FIG. 19-7 **A**, Separated file may accidentally come out with ultrasonic activation during a preparation phase. **B**, EDTA solution is put into the canal to take advantage of cavitation and acoustic streaming effects for instrument retrieval. **C**, Ultrasonics must be activated within the space created. **D**, Separated file is removed from the canal with ultrasonics in the presence of EDTA solution.

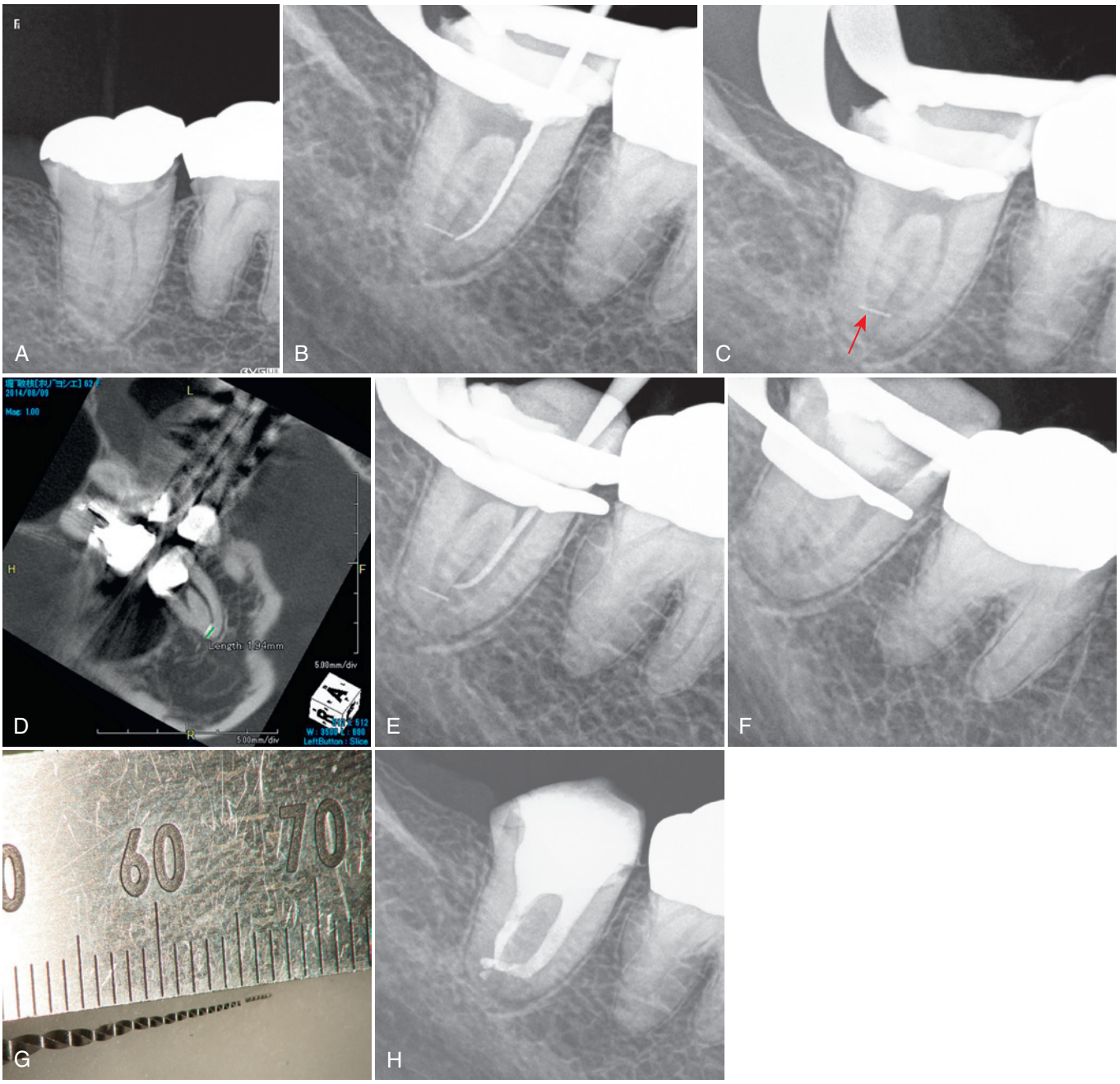

FIG. 19-8 A, Preoperative radiograph showing deep caries lesions underneath the crown with meager looking pulp-capping materials on the pulp chamber. The tooth was diagnosed with irreversible pulpitis. B, NiTi rotary instrument (ProTaper NEXT X1) was separated in the mesial canal during root canal instrumentation. C, Interoperative radiograph showing an instrument fractured beyond the severe curve (*red arrow*). D, Separated instrument measured 1.94 mm on the axial view of CBCT. E, Interoperative radiograph showing the location of the curved microexplorer in relation to the separated instrument in the mesial canal. F, Interoperative radiograph showing the separated instrument was removed with the curved straight ultrasonic tip activated inside the curve. G, Retrieved separated instrument measured actually 2 mm on a ruler in contrast with the coronal portion of the fractured instrument. H, Postoperative radiograph showing the final root fillings, which reveals dentin sacrifice for removal of the separated instrument was minimum.

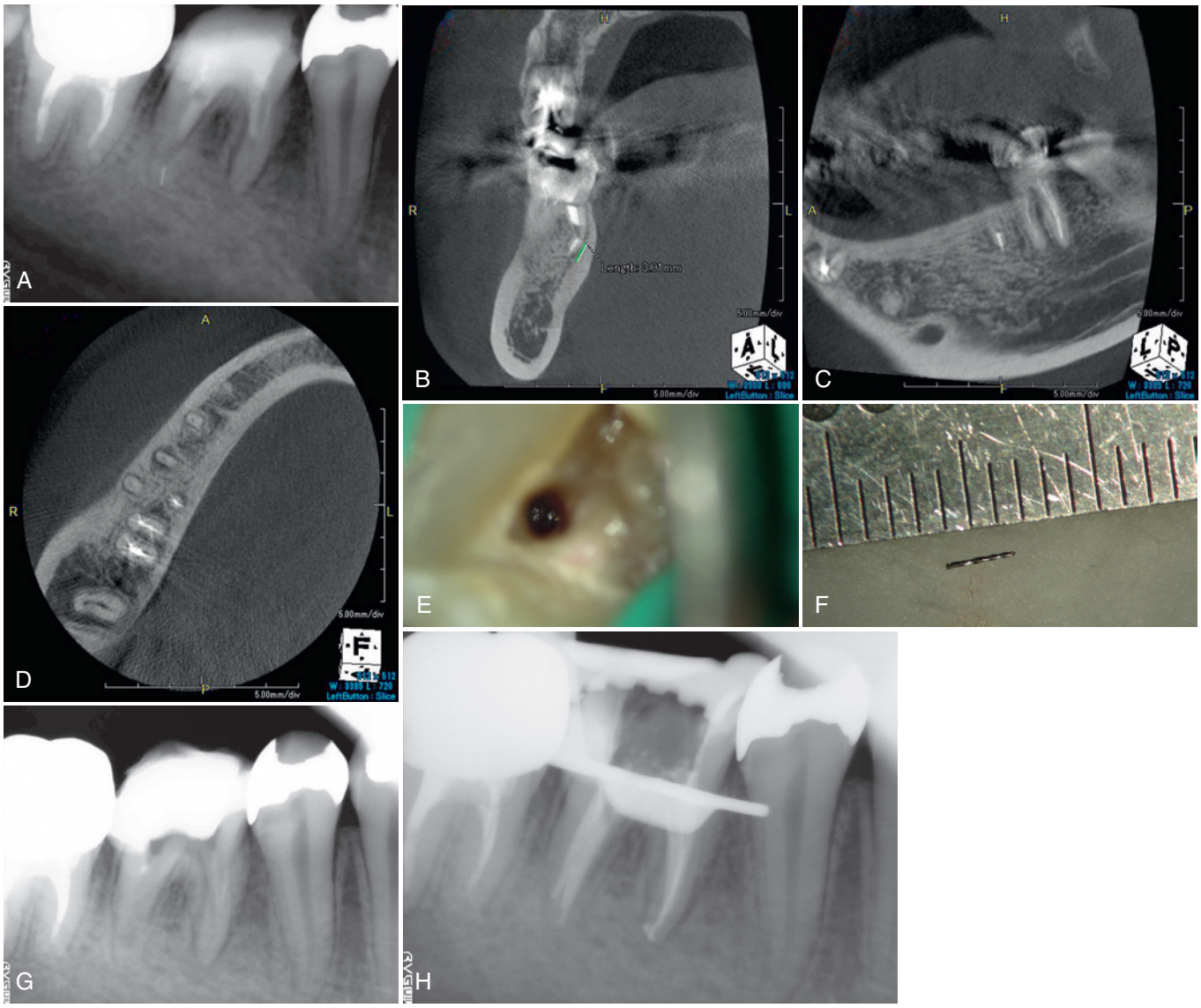

FIG. 19-9 **A**, Preoperative radiograph showing a separated instrument in the canal of the radix entomolaris of the mandibular right first molar. **B**, Coronal view of CBCT showing the separated instrument measuring 3.01 mm beyond the curve of the radix entomolaris, which suggests the separated instrument can be removed with ultrasonics alone. **C**, Sagittal view of CBCT showing the radicular end of the separated instrument is beyond the radicular foramen (*white arrow*). **D**, Axial view of CBCT showing the separated instrument in the canal of the radix entomolaris (*white arrow*). **E**, Magnification of the coronal portion of the separated instrument in the canal (*white arrow*). Removal of this instrument was considered easy as it was visible under the DOM. A small space was created on the inside curve of the canal with the FRK-12 ultrasonic tip. **F**, Retrieved instrument measuring 3 mm on a ruler. **G**, Interoperative radiograph taken after the separated instrument was removed (*white arrow*). **H**, Postoperative radiograph showing root fillings in all the canals revealing the dentin sacrifice for removal of the separated instrument from the radix entomolaris was not significant, compared to the root canal space on the preoperative radiograph.

an endodontic explorer such as a DG16 Endo Explorer (Hu-Friedy, Chicago, IL) (Fig. 19-11). The tip of a DG16 is placed in the loop and the loop is contracted around it. The apical portion of the DG16 is used to make the loop size smaller and the coronal portion is used to make it larger when the loop is tightened around it. The loop is then pre-bent at 45 degrees (Fig. 19-10, *B* and *C*) to facilitate the placement of the loop over the separated instrument. The loop is then brought into the canal and placed over the exposed portion of the separated file as the loop is pushed back at 90 degrees upon it. Subsequently, the loop is tightened over the separated instrument by sliding down the handle (Fig. 19-10, *C*). The separated instrument will be retrieved by pulling the loop out of the canal in various directions in swaying motions to dislodge it from the canal walls (Fig. 19-10, *D*). A couple of pull motions in several directions will usually dislodge the file fragment and complete the case for separated file removal.

Surgical approaches for the management of separated instruments represent a second category of procedures for dealing with this iatrogenic event (see also Chapter 9).

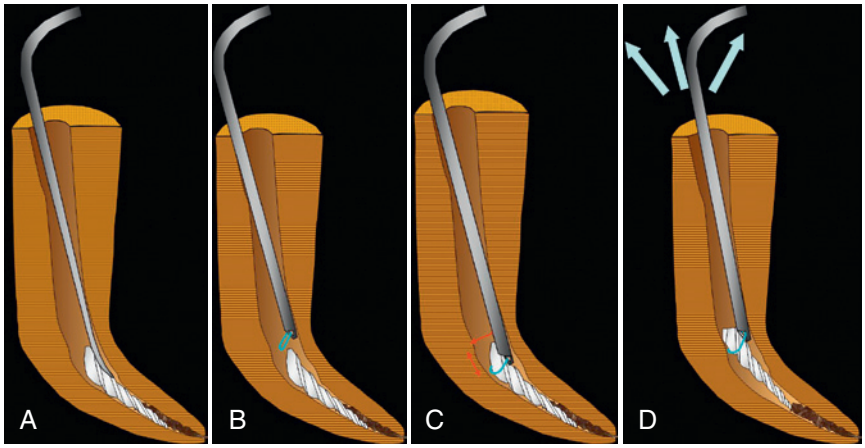

FIG. 19-10 A, Coronal portion of the separated file should be exposed at least 0.7 mm above the bottom of the space created around it. B, Approaching the separated file with the loop bent at 45 degrees against it. C, Loop is pushed back at 90 degrees as it is placed over the separated file. Removal with the loop requires the diameters of the loop device (0.16 mm ~ 0.45 mm) and the separated instrument and a depth of at least 0.7 mm. D, Separated file is pulled out with the loop device in various directions.

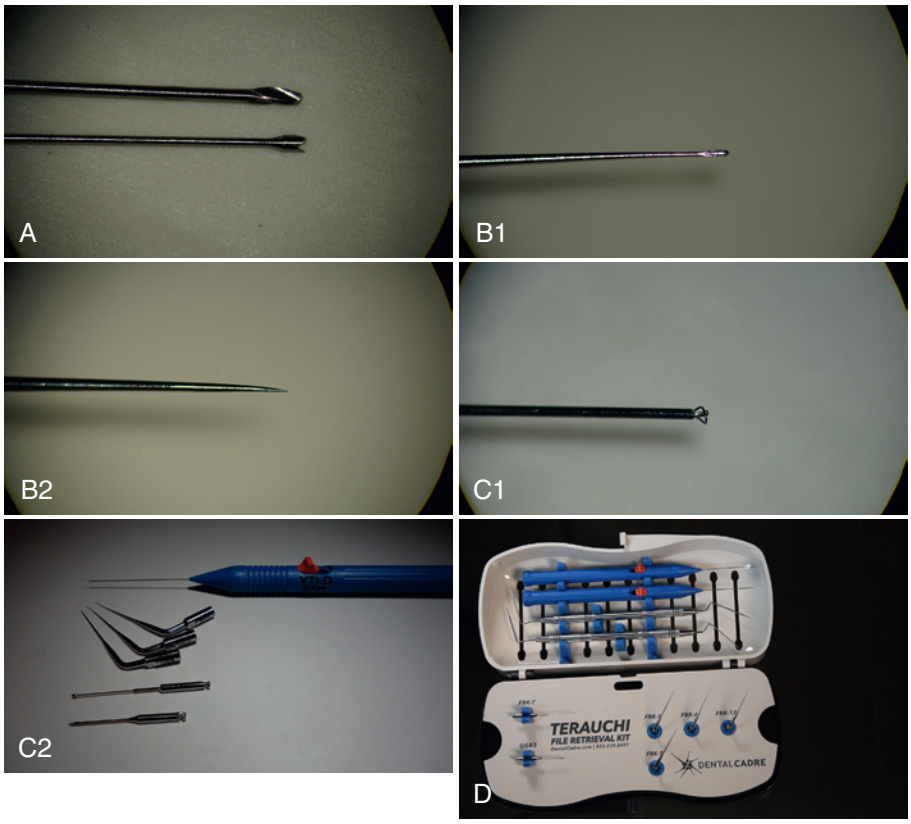

FIG. 19-11 File retrieval kit (FRK). A, Modified #3 Gates Glidden bur that is also called GG-3M (upper) and microtrephine bur that is also called FRK-T (lower). B1, FRK-6 ultrasonic tip (the tip portion looks like a spoon and is also called a spoon tip. It has a micro-concave on the spoon, which turns toward the handpiece at the 6 o'clock position. B2, FRK-S ultrasonic tip (the tip portion looks like a sharp spear and is also called a straight tip). C1, Yoshi loop, which captures a separated instrument. This micro-lasso is comprised of a tiny wire loop at the end of a stainless steel cannula, with a sliding handle that tightens the loop when pulled. C2, Yoshi loop holding a separated file from an actual case. Note that the loop holding the files requires 0.7 mm in length and width. D, File retrieval kit (FRK) in an autoclavable cassette case. The kit includes a GG-3M, FRK-T, FRK-6 ultrasonic tip, FRK-12 ultrasonic tip, FRK-S ultrasonic tip, Yoshi loop, gutta-percha removal (GPR) instrument, and a microexplorer instrument.

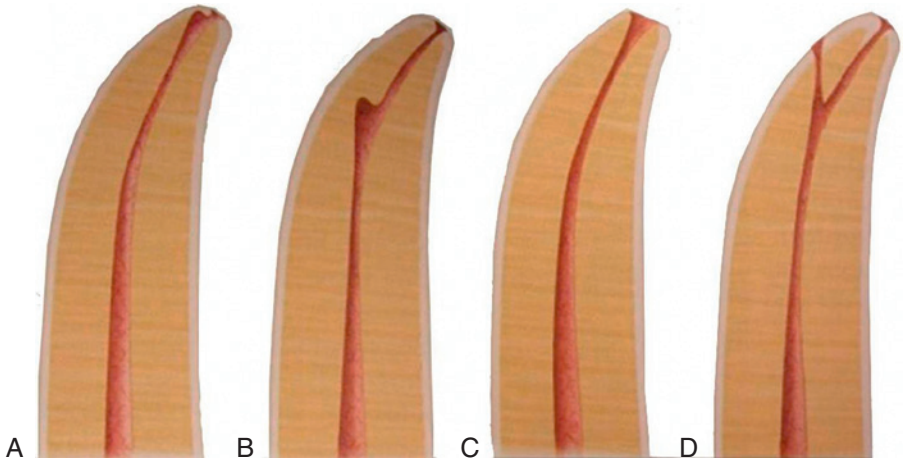

FIG. 19-12 Schematic diagrams showing the most common preparation errors. **A**, Radicular zip. **B**, Ledge. **C**, Radicular zip with perforation. **D**, Ledge with perforation.

Surgical approaches should be considered in the following situations[174]:

- As a last resort if other nonsurgical approaches fail, post-treatment disease develops, and the tooth is strategically important to retain
- As a first approach when periradicular pathosis is present at the time of instrument separation, especially if the separated instrument is found in the apical third of the canal coexistent presumably with persistent extraradicular infection
- As a first approach when the radicular portion of the separated instrument is already outside the root, when an orthograde approach is unsuccessful, or when a significant amount of dentin must be sacrificed for instrument removal

Prognosis

When an endodontic instrument separates in a canal during cleaning and shaping procedures and if it hinders adequate cleaning of the canal, then the prognosis may be affected. Moreover, attempts to remove a separated instrument from a curved canal may cause additional complications such as excessive removal of dentin structure, secondary instrument fracture, root perforation, or even root fracture.[113,246,268,272] However, there are cases in which the prognosis is not compromised, depending on what stage of instrumentation the file fracture occurred, the preoperative diagnosis of the pulp and periradicular tissues, and whether or not the separated file can be removed or bypassed.[272] The presence of a separated instrument per se in a canal or outside the root does not necessarily condemn the case to postoperative disease. Rather, it is the residual presence of any infected tissue that determines the prognosis. The closer the root canal preparation is to the terminus at the time of instrument separation, the better the outcome will be.[291] It has been suggested that instrument separation occurring in a later stage of canal instrumentation, especially if it is at the apex, has the best prognosis because the canal is probably well debrided and may be relatively free from microorganisms.[290] If the preoperative canal is not infected, and no radicular periodontitis is associated with the root, then the presence of the separated instrument should not affect the prognosis[50] and the separated instrument can be incorporated into root canal filling materials. If the separated file can be removed predictably without sacrificing a significant amount of dentin, the prognosis will not be reduced. In other words, the treatment prognosis depends more on the removal of microbial infection than on instrument removal.

LEDGE FORMATION

Ledges may be created as a result of instrumentation causing radicular transportation, which also causes zipping of the canal and possible canal perforation (Fig. 19-12). The Glossary of Endodontic Terms of the American Association of Endodontists[10] defines a ledge as "an artificial irregularity created on the surface of the root canal wall that impedes the placement of an instrument to the apex of an otherwise patent canal." Canal transportation[10] is defined as "the removal of canal wall structure on the outside curve in the radicular half of the canal due to the tendency of files to restore themselves to their original linear shape during canal preparation." The presence of a ledge hinders adequate shaping and cleaning of the canal in the areas radicular to the ledge. Consequently, there is a possible causal relationship between ledge formation and an unfavorable endodontic treatment outcome.[48,49,99,105,112,137,188,319]

Causes of Ledge Formation

Ledge formation can occur during biomechanical preparation of the canal system, especially when the canals are more curved. There are a number of factors associated with ledge formation such as the instrumentation technique, instrument type, root canal curvature, tooth type, working length, master radicular file size, clinician's level of expertise, and canal location. Failure to precurve the instruments, the inability to achieve a proper glide path to the apex, and forcing large files into curved canals are perhaps the most common reasons for ledge formation.[131] The incidence of ledge formation was reported to increase significantly when the curvature of the canal was greater than 20 degrees; more than 50% of the canals in the study were ledged when the canal curvature was greater than 30 degrees.[99] Jafarzadeh[134] described 14 possible causes of ledge formation (Box 19-1).

Management of Ledge Formation

Once a canal is ledged, the endodontic treatment becomes difficult and the prognosis may be compromised. When a ledge is formed, early recognition of its location, using radiographs

BOX 19-1

14 Possible Causes of Ledge Formation

1. Not extending the access cavity sufficiently to allow adequate access to the radicular part of the root canal[48,105,131,137,159,299]
2. Loss of instrument control if endodontic treatment is attempted via a proximal surface cavity or through a proximal restoration[131]
3. Incorrect assessment of the root canal direction[48,137,159]
4. Incorrect root canal length determination[48,137,159]
5. Forcing the instrument into the canal wall[303]
6. Using a noncurved stainless steel instrument that is too large for a curved canal[48,131,137,218,299]
7. Failing to use the instruments in sequential order[48,105,137,159]
8. Rotating the file excessively at the working length[105,303]
9. Inadequate irrigation or lubrication during instrumentation[299]
10. Over-relying on chelating agents[303]
11. Attempting to retrieve separated instruments[48,137,159]
12. Removing root filling materials during endodontic retreatment[48,137,159]
13. Attempting to negotiate calcified root canals[48,137,159]
14. Inadvertently packing debris in the radicular portion of the canal during instrumentation[48,299]

including CBCT and magnification, will facilitate its management.[105] The canal is usually "straightened" at the point where the file stops negotiating the curve, and there is suddenly a looser feeling of the file within the canal, with no more tactile sensation of the tip of the instrument binding in the canal. If the radiograph reveals that the tip of the instrument deviates away from the canal curvature, then it is highly likely that a ledge formation has occurred on the canal wall.

When a ledge formation is identified, the shortest file possible that can reach the working length should be selected to bypass the ledge. Shorter instruments have more stiffness and allow the clinician's fingers to be placed closer to the tip of the instrument, which consequently provides greater tactile sensation and more control over the instrument.[134] It usually requires determination, perseverance, and patience to bypass a ledge once it is formed.[48] The successful management of a ledge depends on whether both the ledge and the orifice of the original pathway can be visualized, reached, or felt with instruments.[166] Any case where the ledge can be visualized under the DOM may be considered less complex to treat. Cases whereby the ledge is not directly visible are more difficult to treat, as tactile sensation and negotiation require patience and experience.[48] However, 3D radiographic images obtained from CBCT reveal the locations and the directions of both the ledge and the original pathway to the canal terminus, which provides additional information for diagnosis and therefore helps the clinician manage the ledge formation with a more predictable outcome and negotiate the original canal.[203]

Bypassing a Ledge

Using Hand Instruments

Precurving of a #8 or #10 hand file is a critical step in bypassing a ledge. The small file should have a distinct curve at the tip (that is, in the radicular 2 to 3 mm) (Fig. 19-13). This file should be used initially to negotiate past the ledge and explore the original pathway of the canal to the radicular foramen.[48,131,299] A rubber stopper with a directional indicator is valuable in this situation because the indicator can be pointed in the same direction as the bend placed in the instrument.[131] A slight rotation of the file combined with a "pecking" motion can often help advance the instrument to the full working length of the canal.[131,299] Whenever resistance to negotiation is met, the file should be retracted slightly, rotated, and then advanced again with the precurved tip facing in a different direction. This action should be repeated until the file consistently bypasses the ledge.[131] If this technique is unsuccessful, the clinician should flare the canal coronal to the ledge in an anticurvature direction to obtain wider straight-line access to the original pathway of the canal beyond the ledge, followed by a #10 K-file with the tip precurved to bypass the ledge (see Fig. 19-13). Small hand files dedicated to the microscopic use (such as the micro-opener with #10/.06 taper) may help flare the orifice of the original pathway beyond the ledge. The use of such small micro–hand files under the DOM with magnification and illumination helps the clinician locate the original pathway and facilitate bypassing the ledge. The micro-opener is available only in 16 mm and it is short enough to provide tactile sensation and better control. However, in some cases a longer instrument is required to reach the ledge present in the radicular third of a long canal. In these instances, a hand instrument holder such as an EndoHandle (Venta, Logan, UT) may help the clinician easily negotiate the canal radicular to the ledge. Any hand files can be attached to the handle for microscopic use. Other hand instruments are available specifically for this purpose (microexplorer instrument, DentalCadre and ELES instrument, DenMat Lompoc, CA) that are long enough to reach the ledge formed in the radicular third. Both of the instruments are made of stainless steel. The microexplorer is used to explore the original canal and penetrate a narrow canal beyond the ledge, while the ELES is used to reduce the ledge as the tip portion is coated with fine diamond particles. They can be used in sequence to reduce or remove the ledge. The microexplorer instrument can be precurved as it has a smooth surfaced tip to get into the original pathway beyond the ledge (Fig. 19-14), whereas the ELES instrument has diamond coating on the tip to enlarge the original pathway (Fig. 19-15). The microexplorer instrument has a 0.1 mm biconical tip with a .08 taper for the first 1-mm portion from the radicular end and a .06 taper for the next 10-mm portion, which gives the instrument more stability in push-pull motions than a .02 tapered K-file, while the tip diameter of the ELES instrument is 0.2 mm with a .06 taper. The maximum diameter of the microexplorer instruments is 0.78 mm, which provides stability and maintains a high visibility under the DOM when it is precurved. On the other hand, the maximum diameter of the ELES instrument is 0.68 mm, which provides flexibility and visibility even around a curve. Once the first microexplorer instrument has successfully bypassed the ledge, subsequently the ELES instrument can be used to enlarge and reestablish the original pathway (Fig. 19-16), followed by hand instruments (such as ProTaper S1 and S2 hand instruments, DENTSPLY Tulsa Dental Specialties, Tulsa, OK) to both reduce the ledge and complete the shape. #15/.04 and #15/.06 Vortex Blue rotary files (DENTSPLY Tulsa Dental Specialties) can also be precurved with ease and used as hand files in short pumping motions to reduce the ledge after the preliminary enlargement of the original pathway with the micro-hand instruments.

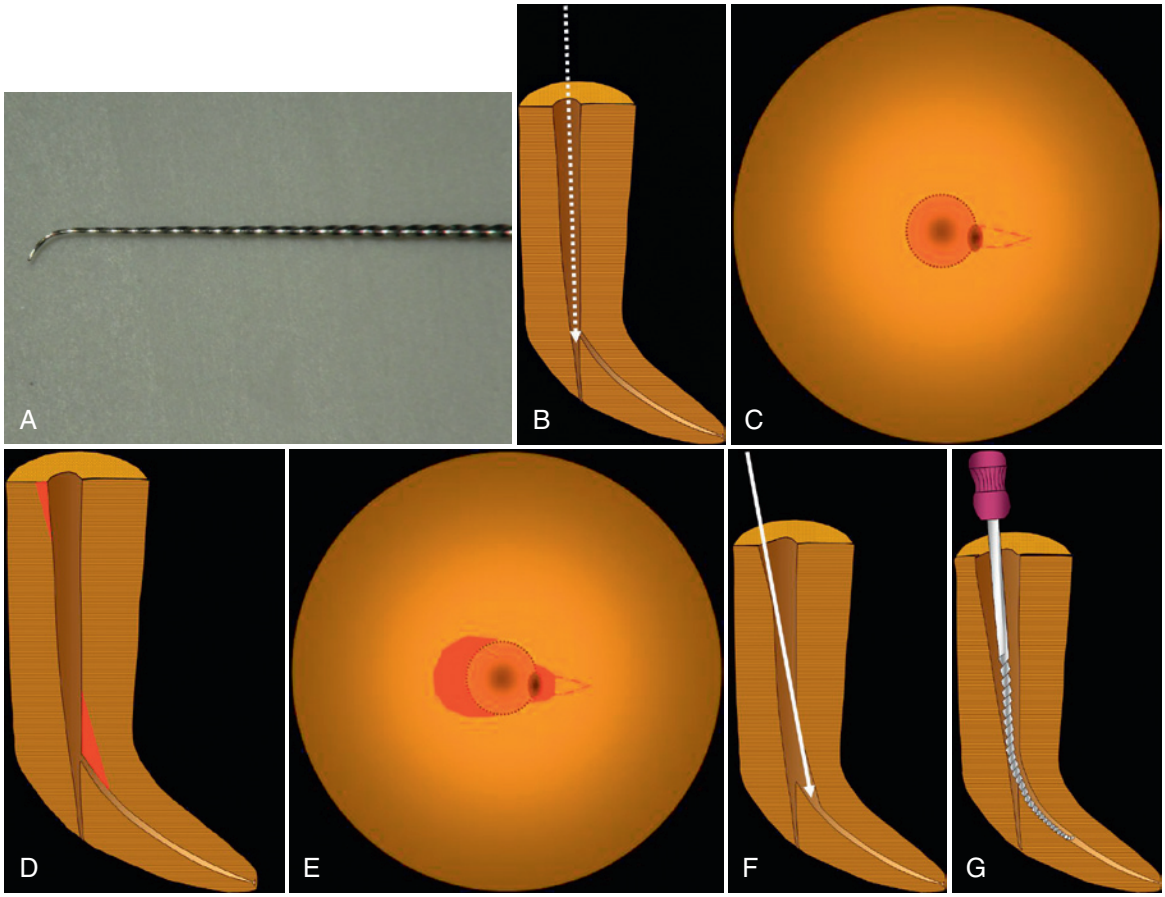

FIG. 19-13 A, Ledge removal instrument (microexplorer) to explore a canal for impediments and to penetrate a narrow canal beyond a ledge. B, Ledge is formed with perforation in a radicular straight direction. C, Microscopic view showing the ledged canal in the center and a narrow access to the original pathway on the right side. D, The canal orifice as well as the access to the original pathway needs to be flared in an anticurvature direction *(red portions)*. E, Microscopic view showing red portions to be removed for easy access to the original pathway. F, Access to the original pathway was flared to facilitate file insertion. G, Bypassing file used in short push-pull strokes to create a distinct pathway to the original canal as the final glide path.

FIG. 19-14 Ledge removal instrument (Microexplorer) to explore and penetrate a canal.

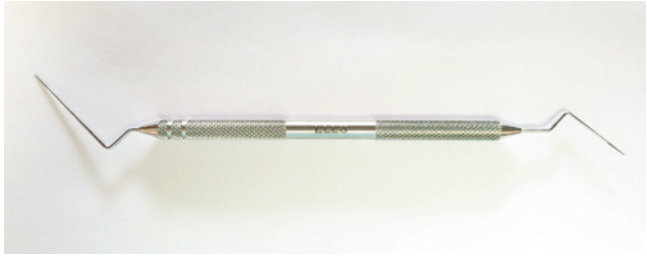

FIG. 19-15 Ledge removal instrument with diamond coating (ELES) to bypass/eliminate/reduce a ledge and enlarge the orifice of the original pathway.

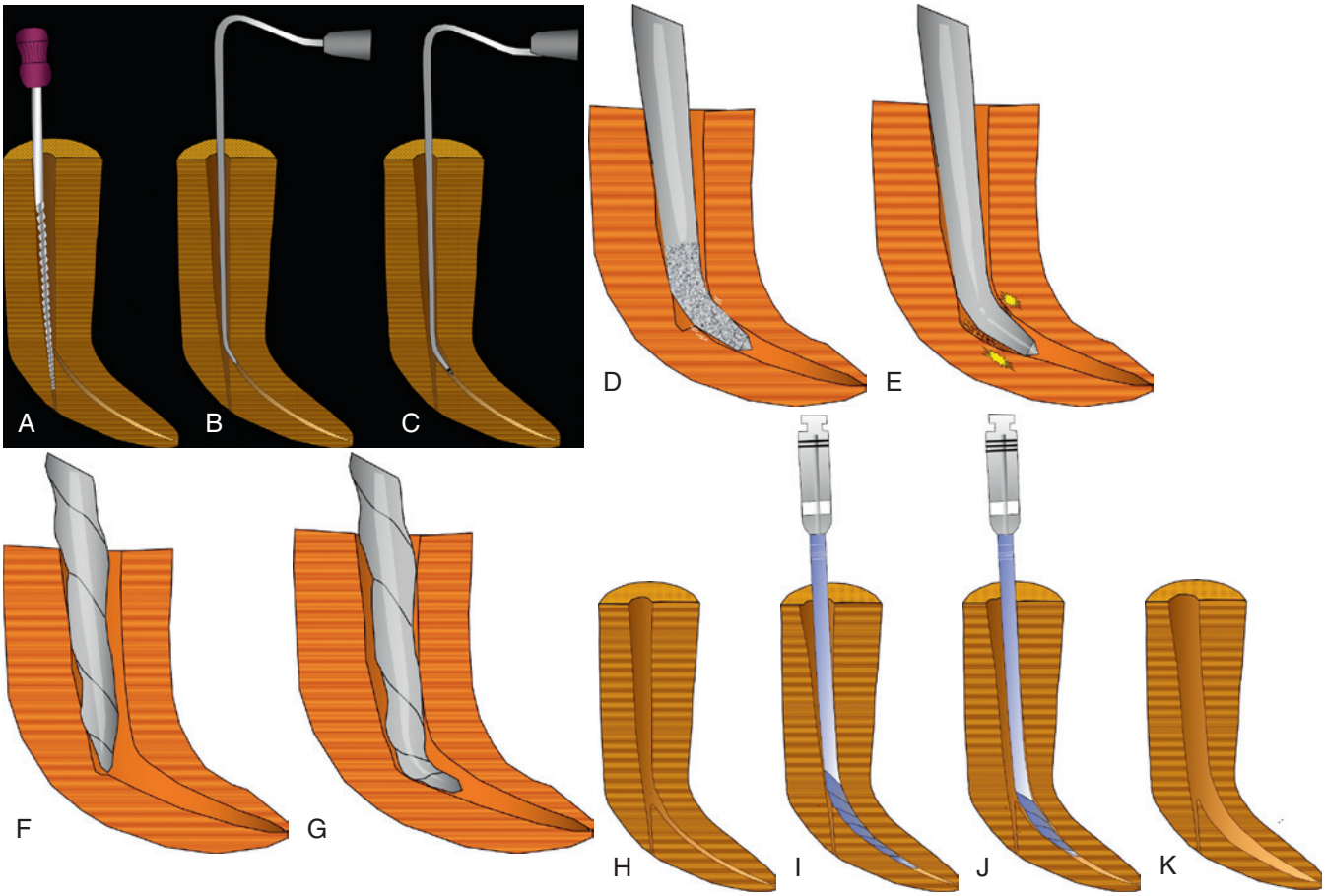

FIG. 19-16 Schematic diagrams showing bypassing the ledge with ledge removal hand instruments. **A,** Ledge is created in a radicular straight direction around a curve. **B,** Original pathway is explored and opened up with the microexplorer instrument in short push-pull strokes. **C,** Original pathway is widened with the ELES instrument. **D,** ELES instrument used in push-pull motions to reduce the ledge. **E,** Precurved straight ultrasonic tip can also be used here to reduce the ledge instead of the ELES instrument. **F,** File with a biconical tip is brought into the canal to eliminate/reduce the ledge and negotiate the original canal. The primary cone of the biconical tip has no cutting tip. As it hits the curve or irregularities on the canal wall, it will slip alongside the curve and scoot over the irregularities. **G,** Secondary cone of the biconical tip removes the points at the transition angles and provides a smooth surface that guides the tip portion into the original pathway. **H,** Orifice of the original canal beyond the ledge needs to be enlarged for easy file insertion after the ledge has been bypassed successfully. **I,** Orifice of the original canal can be efficiently widened with a precurved instrument such as a ProTaper S1 hand file and a #15/.04 Vortex Blue rotary file used as a hand file, and the apical patency established. **J,** Original canal can be sequentially prepared with more tapered NiTi files. **K,** Postoperative canal showing the ledge is significantly eliminated.

Using Ultrasonic Tips

Small diameter ultrasonic tips improve visibility with the added benefit of reducing the need for sacrificing sound dentin in bypassing or removing the ledge.[167] The only drawback to the thinner tips could be that they are more prone to fracture during ultrasonic activation.[298] Therefore, thinner ultrasonic tips should be activated in a pulsing fashion at the lowest possible power setting to prevent fracture and heat generation.

The use of longer ultrasonic tips may also facilitate bypassing or reducing the ledge in the radicular third as long as it is performed under the DOM. The orifice of the original canal just coronal to the ledge should be enlarged. If the ledged portion of the canal can be visualized under the DOM, then ultrasonic instruments should be used over hand instruments (Fig. 19-17). First, an appropriate size of a small diameter ultrasonic tip (such as the straight ultrasonic tip, DentalCadre, Seattle, WA) is selected and precurved in the direction of the original canal. Then it is simply placed in the orifice of the original pathway to the radicular foramen and activated, which will automatically enlarge the opening of the true canal in a few seconds. Ultrasonic tips can cut dentin faster than hand instruments and widen the orifice of the original pathway more easily so that files can consecutively be placed into the original canal for root canal preparation. Ultrasonics should be used on the lowest power setting to avoid overextending the ledge, as ultrasonics may get very aggressive in cutting.[243] Once the orifice of the original pathway gets wide enough for file insertion, negotiation files need to be placed into the canal to establish patency and to smooth out the irregularities on the canal wall that may have been created with ultrasonics,

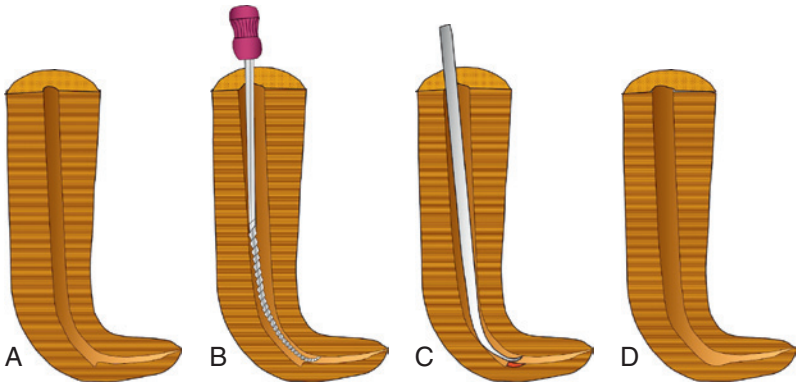

FIG. 19-17 Non-visible ledge removal with ultrasonics. **A,** Ledge formed beyond the curve. CBCT is taken to locate both the ledge and the original pathway. **B,** Canal coronal to the ledge is flared in an anticurvature direction with NiTi rotary files in brushing motions. Original pathway to the canal terminus is explored with a #10 precurved K-file or the precurved microexplorer instrument and radiographs are taken with it inserted in the canal to see if it is in the original canal beyond the ledge. **C,** Straight ultrasonic tip is precurved the same way as the hand instrument used and placed in the canal to eliminate/reduce the ledge. The red portion needs to be smoothed out to facilitate the insertion of an instrument into the original pathway. **D,** Postoperative canal showing the ledge is significantly reduced.

followed by root canal preparation with NiTi rotary or hand files (Figs. 19-18, 19-19).

Using Rotary Instruments

Pre-curved rotary PathFiles are also beneficial for negotiating past a ledge. First the radicular 2 to 3 mm of the #13 PathFile is precurved at 30 to 45 degrees and is rotated at 100 rpm or slower as it is brought slowly into the canal and moved radicularly. If a ledge or sharp radicular curvature is encountered, the instrument is withdrawn about 1 mm and immediately reinserted. This allows the precurved tip to move to a different orientation within the canal as it is reinserted. This may require one or two withdrawals and re-insertions to negotiating the original canal. This procedure is followed in sequence by the #16 and #19 PathFiles to preliminarily enlarge the original pathway and reduce the ledge prior to the conventional root canal preparation with NiTi rotary instruments.

Potential Complications of Removal or Bypassing a Ledge

During ledge removal or bypassing a ledge, complications can occur, including root fracture, root perforation, worsening the ledge, and instrument separation. Since a ledge is likely to form around a curve, attempts to bypass or eliminate it may often involve dentin sacrifice, which may eventually compromise the integrity of the tooth and increase the risk of perforation or root fracture. Care should be taken not to remove excessive root structure or overstress the instruments during the ledge removal process. If a ledge cannot be bypassed with any of the techniques described earlier, and if signs or symptoms of the periradicular lesion persist, then the treatment options are limited to surgical intervention such as periradicular surgery or intentional replantation.[131]

Prevention of Ledge Formation

Passive step-back and balanced force techniques are two beneficial methods of canal preparation that reduce the chances of ledge formation.[131,299] Moreover, it has been proposed that an advantage of the step-back technique is that this method tends to minimize procedural errors such as transportation and ledge formation.[298] Each file is used in sequence with circumferential filing to remove any irregularities before a larger diameter file is placed. The effective use of circumferential filing, especially with Hedström files, will ensure smoothness of the canal walls and flaring toward the coronal end of the canal, which will help to prevent the formation of ledges.[105,299] Precurving the instruments and not forcing them into the canal is one of the most important considerations in the prevention of ledge formation.[131]

The use of NiTi files and instruments with noncutting tips helps to maintain original root canal curvatures.[131] The concept of use of these instruments is that the rounded tip does not cut into the wall but will slip alongside it.[130,131] In addition to the tip design, flexibility of an instrument also plays an important role for keeping the instrument centered in the canal, with NiTi instruments favored over stainless steel instruments.[121,128,196] Compared to stainless steel instruments, NiTi instruments offered less canal transportation, more dentin conservation, and reduced risk of zipping or stripping curved canals.[69,95,201]

Prognosis

A ledge in a canal is analogous to any intracanal obstruction that hinders the cleaning and shaping of the root canal to the radicular terminus. The presence of a non-negotiated ledge may have a similar prognosis as a canal with a retained separated instrument. If the ledge can be bypassed or eliminated, and if the canal beyond the ledge can be cleaned without excessive enlargement or perforation, then the prognosis should not be that greatly reduced.[290,291] The amount of debris and bacteria left in the canal apical to the ledge greatly influences the prognosis.

PERFORATIONS (NONSURGICAL)

The topic of nonsurgical perforations, whether by caries, access bur, or post drill, and whether occurring in the furcation or

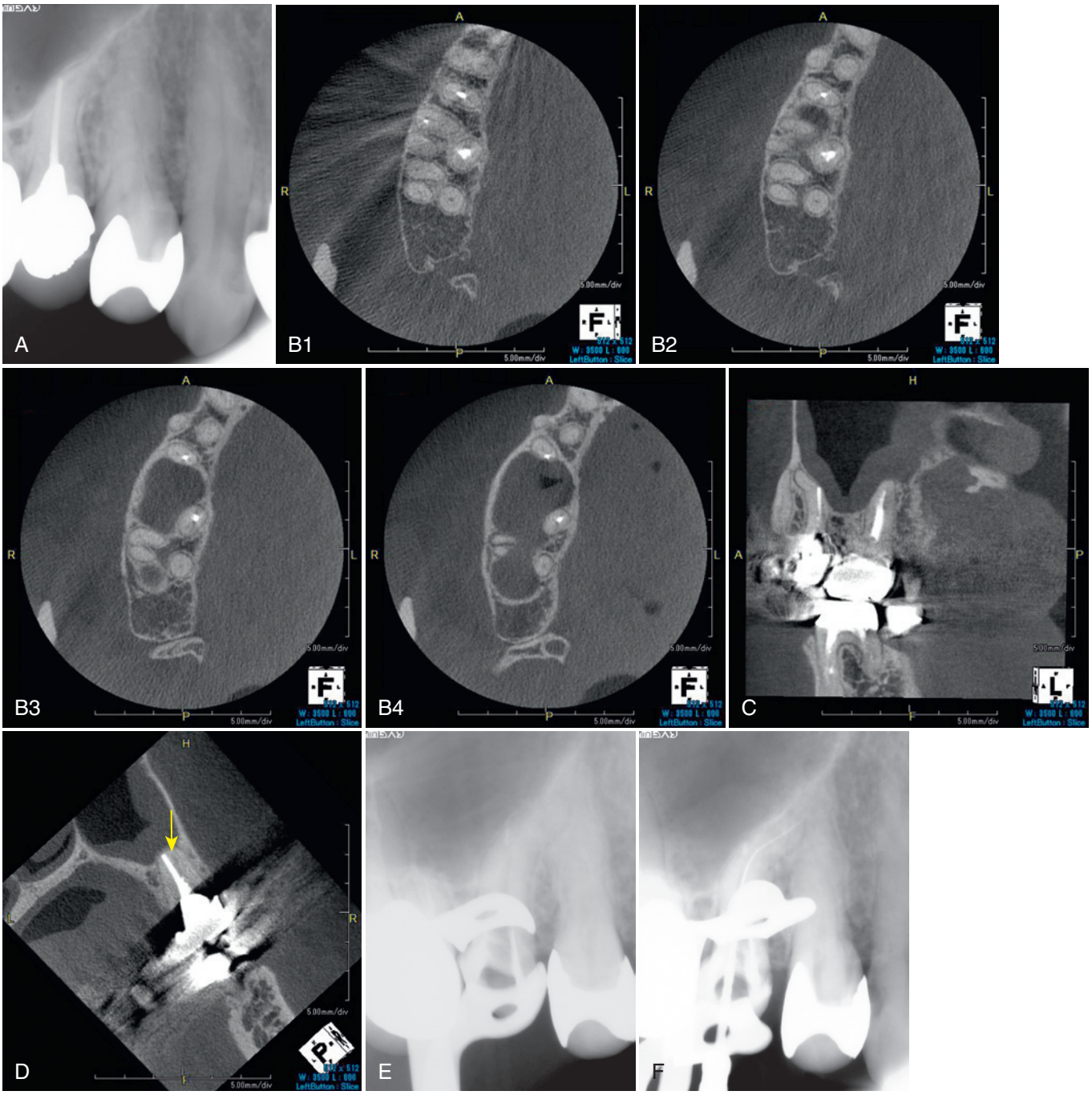

FIG. 19-18 **A**, Preoperative radiograph of a maxillary right second premolar showing a periradicular lesion. Note the root filling in a radicular straight direction deviates away from the radicular foramen. **B**, A series of CBCT images in axial view from the coronal third to the middle third (**B1** to **B4**) reveals the original pathway (*red arrows*) curves in a mesiobuccal direction with the root filling turning away from the original canal. **C**, CBCT image in sagittal view clearly shows the original pathway in relation to the root filling. **D**, CBCT image in coronal view also shows the root filling alongside the original pathway (*red arrow*). **E**, Radiograph showing the root filling coronal to the ledge was removed. Original pathway to the radicular foramen was enlarged with an ultrasonic tip. **F**, Radiograph showing the original canal beyond the ledge was renegotiated with a #10 K-file.

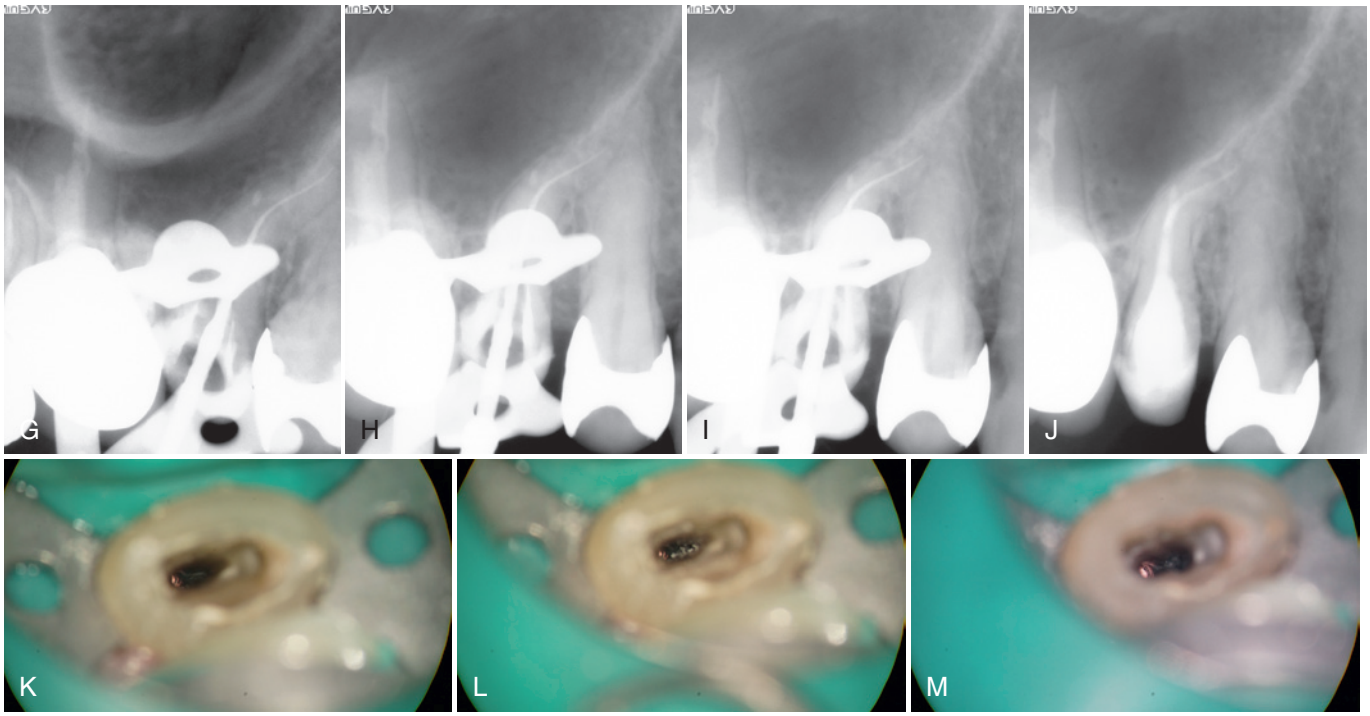

FIG. 19-18, cont'd **G,** Radiograph showing the original canal was flared with a TF adaptive SM1 rotary file used as a hand file in push-pull motions. **H,** Radiograph showing the original canal was sequentially more tapered with a TF Adaptive SM2 rotary file used as a hand file. **I,** Radiograph showing the final root canal preparation of the original canal with a #20/.06 Vortex Blue rotary file used as a hand file. **J,** Postoperative radiograph showing the obturation of the original canal. **K,** Magnification of the original canal orifice side by side with the root filling in the ledged canal. Note the locations of the original canal and the ledge are exactly the same as shown on the axial view of CBCT **B3. L,** Magnification of the original canal showing it was flared with the precurved straight ultrasonic tip. **M,** Magnification of the original canal showing the original canal was completely shaped to the canal terminus with NiTi instruments.

canal space, has been extensively discussed in other chapters of this text. For ease of reference, the various locations and topics are as follows:

- Chapter 8: Nonsurgical Retreatment. Clinical examples of nonsurgical repair of furcation and midlevel perforations, with descriptions of the latest techniques and materials, illustrated with clinical photographs and radiographs. Listed also are the appropriate literature references, indications and contraindications for the repairs presented, and protocols for treatment management.
- Chapter 5: Tooth Morphology and Access Cavity Preparation. Diagrams of perforations and repairs.
- Chapter 29: Endodontic Records and Legal Responsibilities. Legal implications and clinician responsibilities.
- Chapter 26: Effects of Age and Systemic Health on Endodontics. Radiographs of perforations secondary to endodontic attempts in calcified canals.

RADICULAR EXTRUSION OF ROOT CANAL FILLING MATERIALS

The extrusion of root filling materials beyond the radicular foramen may occur either when the canal is obturated or during the removal of obturation materials. Radicularly extruded materials such as obturating materials, necrotic pulp tissue, bacteria, medicaments, and irrigants have been associated with posttreatment inflammation and flare-up or the lack of radicular healing.[186,254,261,289] In vivo histologic studies show the most favorable histologic outcome when the root filling material remains within the radicular constriction. When gutta-percha is extruded into the periradicular tissue, there can be a severe inflammatory reaction.[232] However, other materials such as ProRoot MTA (DENTSPLY Tulsa Dental Specialties) are more biocompatible[1,98,144] and do not seem to produce much periradicular inflammation if extruded beyond the root of the tooth.[75,267]

Studies show that almost all instrumentation techniques promote the radicular extrusion of debris including root filling materials.[8,122,283] Furthermore, gutta-percha and Resilon filling materials obtained from teeth associated with refractory radicular periodontitis have indicated the presence of bacterial biofilm and *E. faecalis*.[102,187,196,255] Another study stressed the possibility of gutta-percha–centered *E. faecalis* biofilm as well as the role of tissue fluids such as saliva and serum in the biofilm formation.[90] *E. faecalis* adheres well to gutta-percha with and without canal sealers, with the biofilm formation more effective when it is in contact with serum in periradicular tissues. Because bacterial biofilm is considered the primary cause of chronic and recurrent endodontic infections,[292] it is crucial to keep gutta-percha filling materials within the canal system as much as possible to prevent potential periradicular periodontitis.

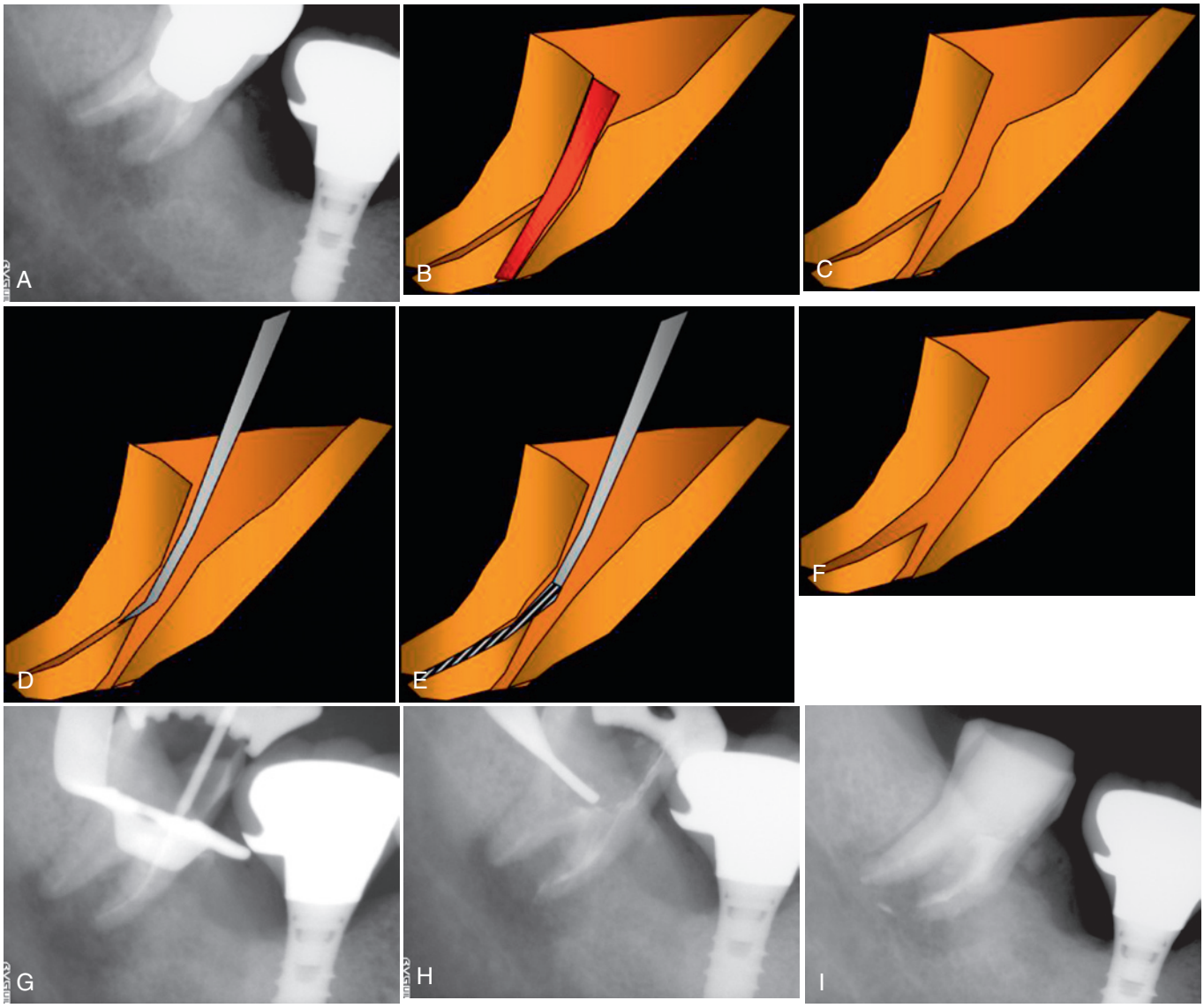

FIG. 19-19 **A,** Preoperative radiograph of a mandibular right second molar showing periradicular lesions associated with the mesial and distal roots. Note the root filling in the mesial root deviates away from the center of the root. **B,** Schematic diagram showing gutta-percha root filling materials in the ledged canal of the mesial root. **C,** Root filling materials were removed chemomechanically from the mesial root. **D,** Original pathway to the radicular foramen was explored and enlarged with the microexplorer and a pre-curved FRK-S ultrasonic tip. **E,** Original canal beyond the ledge was renegotiated with a #10 K-file, followed by a #15 K file. **F,** Schematic diagram showing the original canal shaped to the canal terminus. **G,** Interoperative radiograph showing root canal preparation of the original canal with a ProTaper S1 file. **H,** Postoperative radiograph showing root canals filled with MTA both in mesial and distal roots. **I,** Three-month postoperative radiograph showing periradicular healing.

Uncontrolled obturation can cause the extrusion of root canal filling materials into the maxillary sinus[67] or into the territory of the inferior alveolar nerve (IAN).[214] Serious complications such as maxillary sinusitis, aspergillosis infection, paresthesia, dysesthesia, and similar neural complications may occur after extruding the root filling materials into such sensitive areas.[176]

Endodontic-related paresthesia can be caused mechanically or chemically via the passage of endodontic materials into the vicinity of the major nerves such as the IAN or its branches.[3] These materials include root canal irrigants, sealers, and paraformaldehyde-containing pastes.[297] Because of the potential for chemical degeneration of the nerve axons, the use of paraformaldehyde-containing pastes is not recommended for endodontic obturation.[183,217] The optimum pH of an endodontic medicament should be as close as possible to that of body fluids, which is around 7.35. Significantly higher or lower pHs are likely to cause cellular necrosis of tissues when they are in direct contact with these materials. The clinician must also consider the pH of some of the routinely used endodontic and related dental materials. It has been reported that an alkaline pH of root canal sealers could neutralize the lactic acid from osteoclasts and prevent dissolution of mineralized components of teeth; therefore, root canal sealers, especially calcium silicate–based

cements or sealers, can contribute to hard tissue formation by activating alkaline phosphatase.[276] Commonly used endodontic medicaments are as follows[39,87,226,260]:

- Formocresol: pH 12.45 +/– 0.02
- Sodium hypochlorite: pH 11 to 12
- Calcium hydroxide (Calyxl): pH 10 to 14
- ProRoot MTA: pH 11.7 to 7.12 at 3 hours to 28 days
- Endosequence BC Sealer: pH 10.31 to 11.16 at 3 hours to 240 hours
- MTA Fillapex: pH 9.68 to 7.76 at 3 hours to 168 hours
- AH plus: pH 7.81 to 7.17 at 3 hours to 240 hours
- Antibiotic-corticosteroid paste (Ledermix): pH 8.13 +/– 0.01
- Eugenol: pH 4.34 +/–0.05
- Iodoform paste: pH 2.90 +/–0.02

Neural damage from these materials may be permanent and can cause neuropathic pain (i.e., dysesthesia) or anesthesia.

Orbital pain and headache may occur secondary to the local compression of overextended material into the maxillary sinus.[311] Sinus infection caused by zinc-oxide-eugenol–based filling materials extruded into the maxillary sinus is often associated with aspergillus growth[18,19,93,151,236] (Fig. 19-20). Although aspergillosis of the paranasal sinuses is a relatively rare finding in non-immunocompromised patients, it is known as an opportunistic infection.[143] Many studies have demonstrated that maxillary sinus aspergillosis is generally caused by extruded root canal filling material containing zinc oxide and formaldehyde.[18,19,151,163,274] Besides maxillary sinus infections, the overextension of root filling materials such as gutta-percha into the maxillary sinus may also cause mechanical irritation.[149] When paresthesia is involved, it is thought to occur more from mechanical or thermal insult to the affected neurons.[217] In addition, although such substances are not considered toxic,[270] they may cause an additional foreign body reaction. Inflammation caused by mechanical irritation is likely to persist until the extruded materials are removed, and it may become permanent secondary to the mechanical, thermal, or chemical insult.[114] The incidence of IAN damage has been reported to be up to 1% of cases when performing a root canal filling of a lower premolar.[148] This overextension may cause an unnecessary mechanical or chemical irritation, which hinders repair of the periradicular tissue and thus diminishes the probability of a successful prognosis. The patient may also experience sharp, localized posttreatment pain, which may or may not resolve. When the inferior alveolar nerve is injured, the pain is not only located in the immediate periradicular area but may radiate and refer to the surrounding or distant structures.[195]

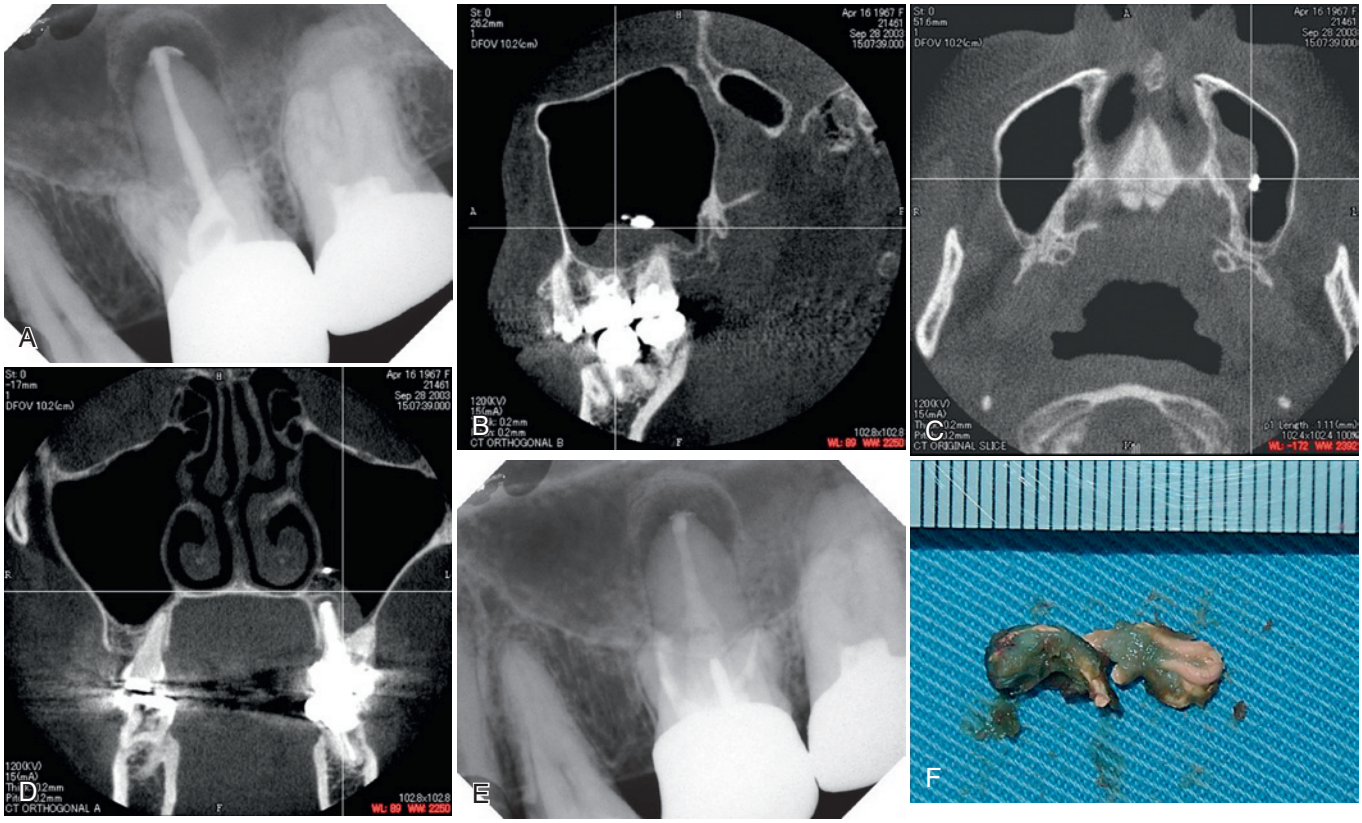

FIG. 19-20 Maxillary sinus aspergillosis. **A,** Preoperative radiograph showing a lesion associated with the palatal root, from which the extrusion of the root filling materials occurred in the maxillary sinus. **B,** CBCT image in sagittal view showing the relation between mucositis on the maxillary sinus floor and the gutta-percha filling material. **C,** CBCT image in axial view showing the relation between mucositis on the mesial wall of the maxillary sinus and the extruded root filling materials. **D,** CBCT image in coronal view showing the relation between the palatal root and the extruded root filling materials. **E,** Postoperative radiograph of orthograde root canal retreatment on the maxillary left first molar showing the mesiobuccal, distobuccal, and palatal root canals were adequately filled without overextension of the filling materials. **F,** Aspergillus was found on the filling materials retrieved from the maxillary sinus.

One study with a 10-year follow-up reported that the overall success rate of endodontic treatment was 91%, with the best prognosis achieved when the obturation was under 0 to 2 mm from the apex.[262] Consequently, radicular extrusion of root canal filling materials must be avoided as much as possible to obtain optimal results.

Causes of Extrusion of Obturation Materials Beyond the Radicular Foramen

The extrusion of obturation material that was thought to be the cause of endodontic failure is reported to be as low as 3%,[266] with the majority of causes thought to be more from coronal leakage. However, several researchers have seen a higher frequency of failure and unhealed periradicular lesions in cases where the root filling material had extruded beyond the radicular foramen.[23,34,209] Overextension of the root canal filling materials occurs mainly for lack of material control.[36,106] The degree of extrusion of obturation materials into periradicular tissues depends on the techniques used for root canal preparation and obturation,[233] which include accidental overinstrumentation and incorrect canal working length measurement. Caution should also be taken when obturating a canal with open apices and larger minor foramen diameters, which are often found in cases with radicular inflammatory resorption. Warm vertical compaction techniques produce a homogeneous obturation that adapts well to the canal walls[249,309] but may result in the extrusion of gutta-percha filling into the periradicular tissues.[36] Carrier-based obturation was also found to have more radicular extrusion of gutta-percha when compared with warm vertical compaction.[158] The type of canal shaping may also be a contributing factor. The canal shape created with the Profile GT system is more prone to extrusion of gutta-percha when obturated with the carrier-based technique than the canal shape created using Profile 0.06 instruments. The Profile GT system created a more parallel preparation coronally and a tapered preparation radicularly, whereas the Profile 0.06 system created a preparation with uniform taper from the apex to its most coronal extent.[233] Therefore, possibly the more continuously tapered the canal shape is, the less obturation material is extruded beyond the radicular foramen.

When endodontic retreatment is performed, irritants in the form of root filling materials can be extruded into the periradicular tissues.[8,122,283] Techniques involving push-pull filing strokes usually create a greater mass of debris than those involving some sort of rotational motions.[6,32,76,142,178,222]

Management of Obturation Materials Extruded Beyond the Radicular Foramen

Nonsurgical Management of Extrusion of Obturation Materials

Methods to remove the gutta-percha filling material from the root canal include the use of heat-carrying instruments, ultrasonics, solvents, and rotary or hand files. Mechanized methods to remove filling materials are faster but possibly not more effective than manual methods.[141,282] Heat-carrying instrument may damage the periradicular tissues if the removal attempts extend beyond the radicular foramen. The complete removal of gutta-percha from the root canal system has proven difficult.[108,282] Poorly compacted gutta-percha obturation can easily be removed with Hedstrom files and solvents such as chloroform,[46,132] although the extruded portion of root canal fillings will often remain in the periradicular tissue[46] (Fig. 19-21). The relative ease of gutta-percha removal suggests a failure of previous endodontic treatment, possibly related to poor obturation compaction or canal shaping, which resulted in both radicular extrusion and failure of the radicular seal. Comparatively, well-filled canals with radicular extrusion do not appear to have a significant failure rate.[13,132] On the other hand, the extrusion of the MTA root filling material into the periradicular tissues does not appear to negatively affect the periradicular healing due to the high biocompatibility and antibacterial properties that the calcium silicate–based cement possesses,[171,281] compared to gutta-percha filling materials (Fig. 19-22).

When gutta-percha is overextended into the periradicular space, removal can be attempted by inserting a new Hedstrom file, usually a #15 or #20, into the radicular obturation, using a gentle clockwise rotation to a depth of 0.5 to 1 mm beyond the radicular constriction. The file is then slowly and firmly withdrawn with no rotation, thereby engaging and removing the overextended material.[180] This technique works frequently, but care must be taken not to force the instrument, or further extrusion of the gutta-percha or separation of the file may result. The overextended radicular portion of the gutta-percha should not be softened with heat or solvent, because this application can decrease the likelihood of the Hedstrom file engaging the obturation, hindering its retrieval.[273] In addition, care should be taken not to overextend the file into the area of the inferior alveolar nerve canal, which could cause irreversible damage to the associated nerves.

The use of rotary NiTi files that are specifically designed for gutta-percha removal appears to be effective[124,265] include ProTaper Universal Retreatment files (DENTSPLY Tulsa Dental Specialties), Mtwo R rotary files (VDW, Munich, Germany), R-Endo retreatment files (Micromega, Besancon, France), and hand instruments such Micro-debriders (DENTSPLY Maillefer, Ballaigues, Switzerland), EGPR-L/R/U/D (G. Hartzell & Son, Concord, CA), and the gutta-percha removal instrument (DentalCadre). The self-adjusting file (SAF) (ReDent-Nova, Ra'anana, Israel) is also effective in removing obturation material, especially the remaining gutta-percha after conventional retreatment. Its variable shape and scraping motion allow it to touch a higher percentage of root canal walls than rotary instruments, facilitating effective cleaning.[181] Literature has shown that significantly less debris was extruded with the rotary retreatment files during endodontic retreatment compared with other traditional techniques using Hedström files with chloroform,[124] although all techniques resulted in some radicular extrusion.[124,265] ProTaper Universal Retreatment files have been reported to remove gutta-percha from the canals in large pieces around the spirals of instruments, whereas Hedström files generally remove gutta-percha in small increments.[124] Furthermore, a combination of both rotary instruments for initial quick removal of gutta-percha and hand instruments to refine and complete the cleaning of the canal, especially in the radicular third of the root, shows the better protocol for obtaining clean canal walls during endodontic retreatment.[24,129]

In difficult retreatment cases where root canal fillings persistently remain in the periradicular tissues, the use of a solvent, such as chloroform, may help to dissolve the remaining gutta-percha. In addition, chloroform has been reported to be capable of reducing the intracanal levels of *Enterococcus*

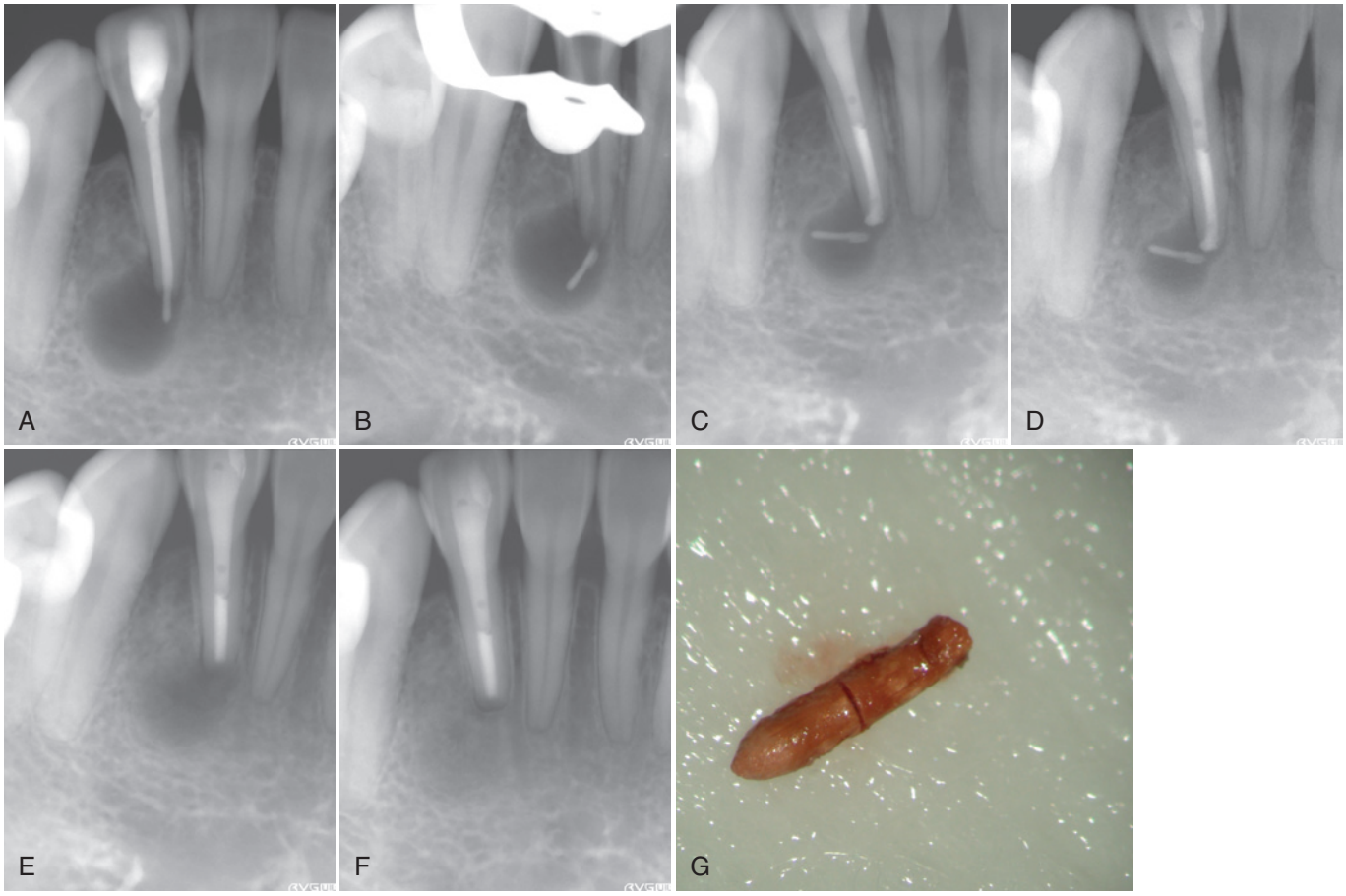

FIG. 19-21 Root filling materials extruded beyond the radicular foramen. **A,** Preoperative radiograph showing overextension of the root filling material with a large lesion associated with the root. **B,** Root filling in the canal was mechanically removed, but the extruded portion of it remained in the periradicular tissues. **C,** MTA was filled in the canal with the extruded portion remaining in the lesion. **D,** Six-month postoperative radiograph showing the reduced size of the lesion, but the lesion surrounding the extruded portion stayed the same in size for the next 6 months, indicating infection of the extruded gutta-percha point. The extruded portion was surgically removed from the lesion at this point. **E,** 3-month follow-up radiograph after the surgery showing the healing lesion. **F,** 12-month follow-up radiograph after the surgery showing a more periradicular healing. **G,** Gutta-percha point retrieved from the lesion.

faecalis.[62,182] When chloroform is used during the early stages of gutta-percha removal, more filling material will remain in the canal and some of it may be extruded beyond the radicular foramen.[124,172] Although the carcinogenicity of chloroform in humans is suspect, chloroform is regarded as a safe and effective endodontic solvent, when used carefully.[44,177]

Unlike rotary instruments, the EGPR-L/R/U/D hand instruments and the gutta-percha removal instrument are 30 mm in length, allowing them to remove obturation material not only from the radicular third of the canal but also from the periradicular tissues through the radicular foramen. The microdebriders are designed to be used in conjunction with a dental operating microscope. They have a Hedström cutting configuration with a standard .02 taper in sizes 20 and 30 with 16 mm of cutting flutes, which may not be long enough to reach the gutta-percha filling material beyond the radicular foramen. The EGPR instrument has a tiny projection on one side of the tip for scratching and engaging the gutta-percha filling left on the canal wall. The overall diameter of the tip is 0.3 mm and the length of the projection is 0.1 mm. This hand instrument works like a scraper on the canal wall. First, a small hole is created with a small diameter ultrasonic tip between the canal wall and the root filling material, and the instrument is brought into the canal with the projection facing away from the gutta-percha filling. When the radicular portion comes down to the bottom of the hole created, the projection is turned back on the gutta-percha filling so that it can be hooked on the projection pressed against it. Then the gutta-percha filling should be scraped and come apart in large pieces from the canal wall (Fig. 19-23, *A* to *C*).

The gutta-percha removal instrument has a tiny conical harpoon-shaped tip that is sharp and small enough to penetrate into a mass of gutta-percha filling and hook it on the rim on the return stroke. The tip portion of this instrument has a .40 taper with the minimum tip diameter of 0.1 mm and the maximum diameter of 0.3 mm with a 0.2-mm diameter shank supporting the tip portion. The 0.1-mm gap between the 0.3-mm and the 0.2-mm diameters works practically as a tiny circular projection over the shank portion that can hook a mass of gutta-percha fillings. Gutta-percha fillings can be separated

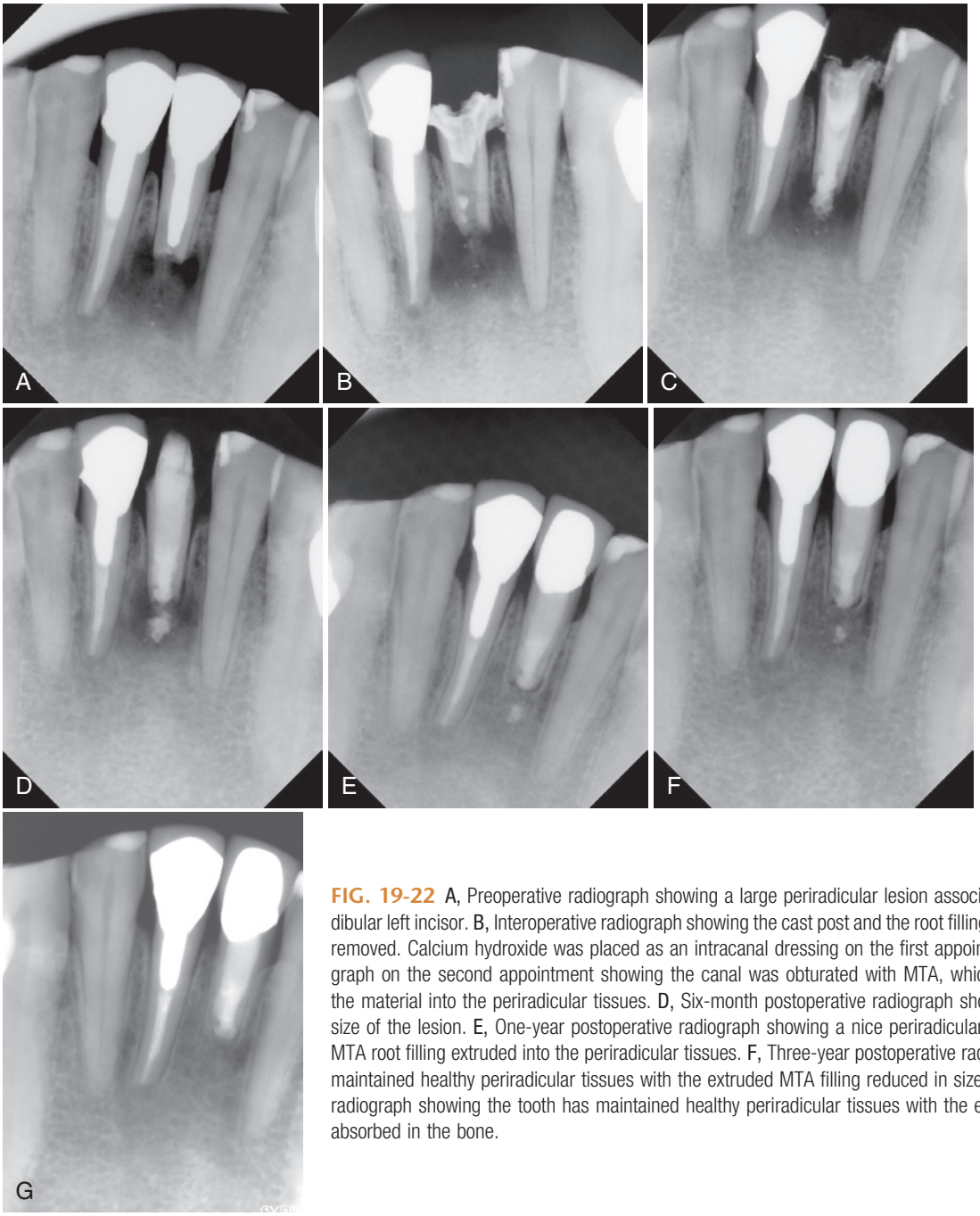

FIG. 19-22 A, Preoperative radiograph showing a large periradicular lesion associated with the root of the mandibular left incisor. B, Interoperative radiograph showing the cast post and the root filling in the canal was mechanically removed. Calcium hydroxide was placed as an intracanal dressing on the first appointment. C, Postoperative radiograph on the second appointment showing the canal was obturated with MTA, which resulted in overextension of the material into the periradicular tissues. D, Six-month postoperative radiograph showing the significantly reduced size of the lesion. E, One-year postoperative radiograph showing a nice periradicular healing with a residual of the MTA root filling extruded into the periradicular tissues. F, Three-year postoperative radiograph showing the tooth has maintained healthy periradicular tissues with the extruded MTA filling reduced in size. G, Twelve-year postoperative radiograph showing the tooth has maintained healthy periradicular tissues with the extruded MTA filling completely absorbed in the bone.

from the canal wall by wedging the tip between the gutta-percha and the canal wall. The gutta-percha filling can also be hooked by pressing the projecting rim against it, and then the gutta-percha filling should be removed in large pieces from the canal wall.

It is recommended that a small hole be created in the center of the gutta-percha filling with a small diameter ultrasonic tip to facilitate the root filling removal with the hand gutta-percha removal instrument placed into the hole. If the gutta-percha filling is poorly adapted to the canal wall with a large radicular diameter, the insertion space for the hand instrument should be created between the canal wall and the gutta-percha filling to avoid radicular extrusion of the root filling materials with the hand instrument or an ultrasonic tip. When a small amount of gutta-percha filling remains on the inside curve or in the radicular third of the canal after the mechanical removal attempts, chloroform should be filled in the canal and the gutta-percha removal instrument can be used to agitate it in short push-and-pull motions in the canal, or even outside the canal if root filling is extruded beyond the radicular foramen, to actively dissolve the residual root filling.

Management of Tissue Damage Caused by Extrusion of Root Canal Filling Materials

When root canal filling material extends into the periradicular spaces, tissue damage may occur. The majority of these cases are managed by nonsurgical methods such as analgesics, cold

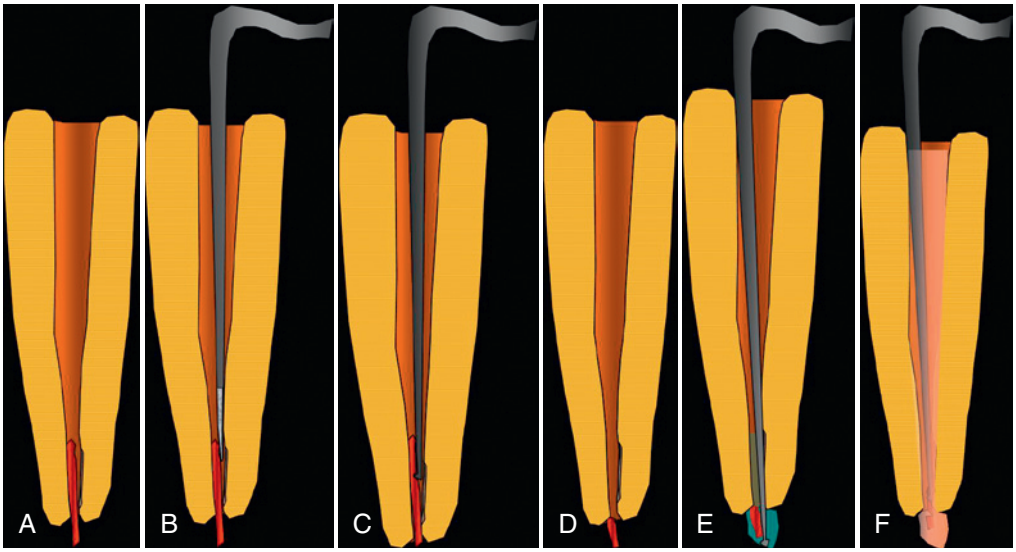

FIG. 19-23 Diagrams showing removal of gutta-percha filling materials extruded beyond the radicular foramen. **A**, Solid gutta-percha filling extruded beyond the radicular foramen. **B**, Small space is created between the canal wall and the gutta-percha filling with a thin ultrasonic tip (such as a FRK-S ultrasonic tip). **C**, Gutta-percha removal hand instrument, such as the GPR instrument and the EGPR instrument, is brought into the canal with the tiny projection pressed against the gutta-percha filling to pull it out. **D**, Extruded gutta-percha root filling portion separated from the coronal portion within the canal. **E**, Chloroform is filled in the canal using a gutta-percha removal hand instrument and agitating it to dissolve the extruded portion. **F**, Gutta-percha filling material is diluted and dissolved with the hand instrument in push-and-pull motions.

packs, corticosteroids, and antibiotics.[72] Dexamethasone is a corticosteroid that has been widely used in dentistry, and it seems to decrease periradicular inflammation caused by a foreign body[183] with minimal side effects.[52] If the tissue damage is extended to the IAN or maxillary sinus, a different approach may be necessary.

SINUS PERFORATION

There is a close proximity of maxillary premolars and molars to the sinus, and the apices of these teeth can be less than 2 mm from the floor of the sinus.[61] Pathologic destruction of the sinus floor may predispose a maxillary sinus communication,[253] producing an oroantral communication,[80,165,239,302] also known as an OAC. In general, exposure of the sinus lumen is well tolerated,[133,208] with no overt repercussions on healing. One investigator[302] found no significant difference in the healing of the mucosa in patients with and without intraoperative perforations and regeneration of the membrane, complete with cilia, in approximately 5 months following complete removal.[22] However, introduction of foreign materials during the access or apical resection/manipulation can initiate thickening of the sinus mucosa with symptoms of maxillary sinusitis.[65]

Conventional radiographs appear to be of limited value in predicting the potential for OAC, with the position of the root tip, periapical lesion, or outline of the lesion not statistically significant in that respect.[197] In contrast, a CBCT survey of the intended surgical field is much more informative, providing accurate measurements of the osseous thickness, distance to the sinus floor, and presence of pathosis.[28]

Maxillary sinus complications secondary to the extrusion of root canal filling material need to be managed differently. Approximately 10% of all patients with chronic sinusitis are found to have an aspergilloma.[101,168] It has been suggested that root canal treated teeth with overextension of the root canal sealer into the maxillary sinus might be the main cause for aspergillosis in healthy patients.[18,19] Surgical intervention is usually the most predictable method for removing extruded root canal filling material and allowing for adequate healing secondary to IAN or sinus complications.[287]

BOX 19-2

Common Causes of Damage to the IAN

1. Local anesthetic injections
2. Third molar surgery
3. Implants
4. Endodontics (extrusion of the materials used in endodontic treatment, overinstrumentation, elevated temperatures)
5. Ablative surgery
6. Trauma
7. Orthognathic surgery

INFERIOR ALVEOLAR NERVE (IAN) INJURY

Prior to considering any treatment to resolve IAN damage, the etiology must be first determined. Common causes of injury to the IAN are listed in Box 19-2.[226] The IAN may be damaged when an increase in temperature proximal to the IAN is greater than 10° C.[68] IAN damage has been suggested to occur in up to 1% of mandibular premolars that receive root canal treatment.[68] The type of sealer cement used may influence the symptoms of IAN damage.[189] If the patient exhibits neuropathic signs after the local anesthesia has worn off, posttreatment radiographs should be exposed to observe the possibility of any obturation material extending into the IAN canal.

Management should be performed by immediate removal of endodontic materials within 24 to 48 hours via periapical surgery, tooth extraction, or surgical debridement, whichever will be the most effective option with the least potential damage to the IAN.[59,153,214,226]

Nonsurgical Causes of IAN Injury

Trigeminal nerve injury is the most challenging consequence of contemporary dental surgical procedures with significant medical-legal repercussions (see also Chapter 29).[38] An approximate estimation places the incidence if IAN injury at 5%, with approximately one third of these patients afflicted with neuropathies, including neuropathic pain.[228] Neuropathic pain is characterized by symptoms that differ significantly from other chronic pain states, including paresthesia, either as a tingling or formication (ants crawling on the skin), burning or shooting pain, electric shock and evoked pain in the form of hyperalgesia, and mechanical allodynia. There is almost always an area of abnormal sensation, and the patient's maximum pain is often coexistent with the site of sensory deficit.[227] This is an important diagnostic feature, as the sensory deficit is usually to noxious and thermal stimuli, indicating damage to small diameter afferent fibers of the spinothalamic tract.[224]

The highest incidence of reported damage to the inferior alveolar and lingual nerves is associated with third molar extractions.[15,118,119] Local anesthesia was the second most common cause of nerve injury, and its exact mechanism can be confusing. It may be physical (needle damage, epineural/perineural hemorrhage) or chemical (local anesthetic contents). Reports have implicated certain local anesthetics of high concentrations (prilocaine 4% and articaine 4%) with IAN neuropathies (see also Chapter 4).[14,110,120,216]

A relatively small percentage of IAN injury cases (8%) are associated with an endodontic procedure.[229] Mandibular second molars are most commonly associated with this population, but cases involving treatment of mandibular first molars and premolars have also been reported.[148] The three primary factors responsible for instigating IAN damage during endodontic treatment are as follows:

1. Chemical factors: cytotoxic materials employed either in the preparation (e.g., irrigation liquids, intracanal medicaments)[2,91] or the obturation of the canal space[238]
2. Mechanical factors: repeated insertions of endodontic instruments through the apex into the IAN and surrounding tissues[57]
3. Thermal factors: inappropriate/prolonged heat application[71]

The presence of bone between the apices and the IAN canal does not necessarily protect against injury to the nerve. Research has provided an enlightening explanation to this puzzle (Figs. 19-24, 19-25, and 19-26).[288] Using fresh cadaver specimens (mandibles), researchers examined the true anatomic relationship among the root apices, inferior alveolar nerve, and the artery. They drew the following conclusions:

- There was never any compact layer of cortical bone surrounding the mandibular nerve sheath. In some cases a slightly denser cancellous bone was found, but it was replete with multiple perforations that would not afford significant resistance to penetration.[115]
- The proximity of the IAN canal to the apices of the second molars was < 1 mm, and it was more variable (1 to 4 mm) with first molars. This could explain the higher percentages of paresthesias reported with these teeth.

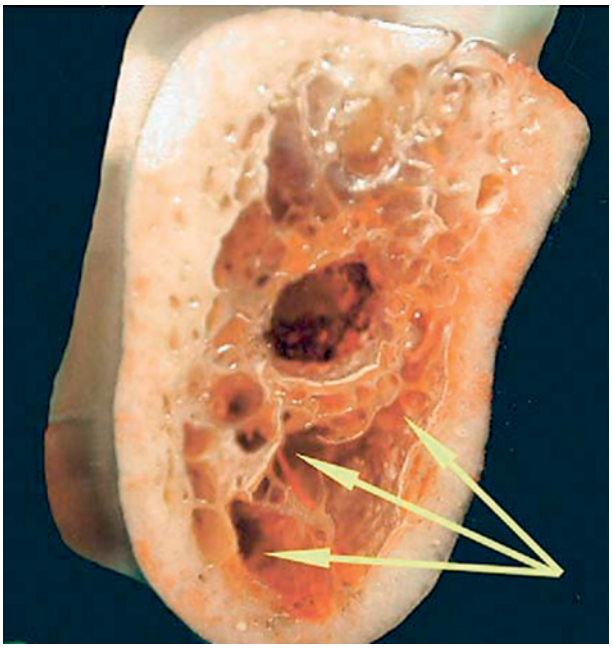

FIG. 19-24 Cross section of mandible in the molar region showing the presence of bone vacuoles *(yellow arrows)*. (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

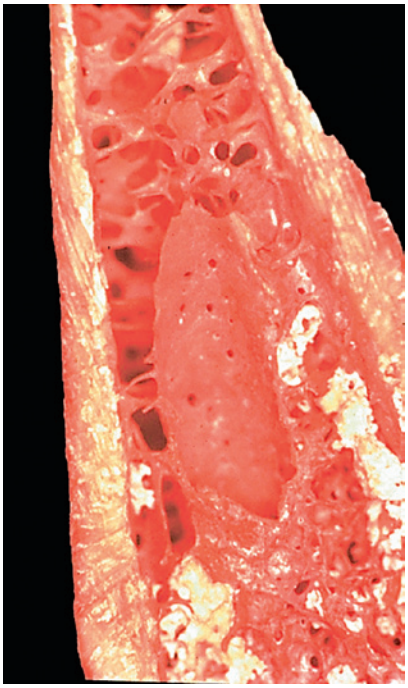

FIG. 19-25 Longitudinal mandible cross section in the molar region showing the spongy bone cover of the neurovascular bundle. Note the multiple fenestrations and honeycombed trabeculations. (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

- The mandibular artery ran along the superomedial side of the IAN up to the first molar, and then crossed on the upper side until the mental foramen. Perforation of this vessel, with the concomitant hemorrhage exerting pressure on the pedicle, could feasibly produce a paresthetic event via the resulting ischemia.[58]
- Only with root apices in direct contact with the IAN canal were extrusions able to penetrate the nerve. If cancellous bone was interspersed between the apices and pedicle, the spread of the extrusion through the trabeculations remained at the periphery of the sheath.
- The low density of trabeculations and presence of numerous vacuoles provided an avenue for the diffusion of extruded irrigation fluids and obturation materials toward the IAN bundle. In a tooth with apical resorption, the foramen is frequently enlarged[310]; these two factors in concert could explain the occurrence of sensory symptoms in the absence of clinical or radiographic signs of overexpression or close proximity to the mandibular nerve.

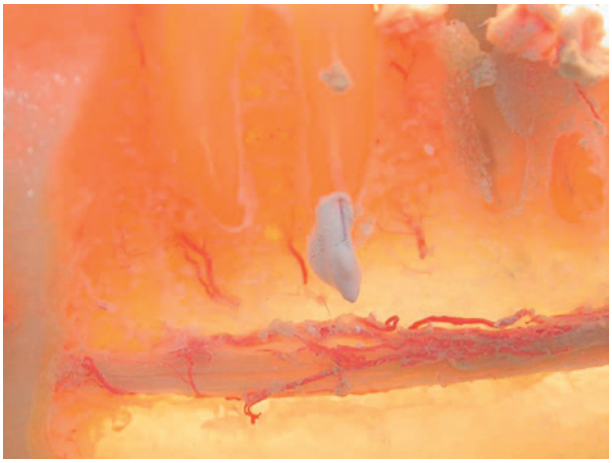

FIG. 19-26 Overfill of the distal root under direct observation. Note the proximity of the extruded material to the artery and nerve bundle (pedicle). (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

Prevention is the key to successful treatment. Damage, either directly to the IAN or to the vessel(s) that nourish it, will result in sensory events that may be transient or permanent. Pretreatment radiographic assessment using periapical images is mandatory; in critical cases, CBCT may be more appropriate and precise in establishing proximity (Fig. 19-27). Every attempt should be undertaken to confine the instrumentation and irrigation processes to within the canal space; damage from extrusions can be chemical or mechanical in origin,[251] and toxic products can easily diffuse through the trabecular spaces and vacuoles surrounding the nerve bundle. This latency in diffusion, and the potential onset of paresthetic symptoms, dictates close monitoring for 72 hours postoperatively in cases where extrusion is apparent.[214] Imprudent heat application during thermoplastic compaction of gutta-percha may also lead to IAN injury.[26]

There is a strong correlation between duration, origin, significance of the injury, and the prognosis for resolution of the paresthesia. The longer the irritation persists, either chemical of mechanical, the more fibers are involved in a degenerative process and the more likely the paresthesia will become permanent.[244] If injury to the nerve is demonstrated by postoperative neuropathy and radiographs confirm the presence of a significant extrusion into the IAN canal, management entails the surgical removal of the offending material.[214,226,228] However, in spite of the best efforts to eliminate the causative agent, the symptoms may persist because of the associated chemical damage to the nerve. If sensory improvement has not been evident after 2 months, with or without repair during interventional surgery, then the prognosis for recovery is poor, and steps should be taken to alleviate the patient's concerns through counseling and systemic medications for chronic pain.[229]

Surgical Causes of IAN Injury

Surgical endodontic procedures need to be designed to minimize trauma to nerves including the IAN, mental nerve, and mental foramen. Although the mental foramen is most often

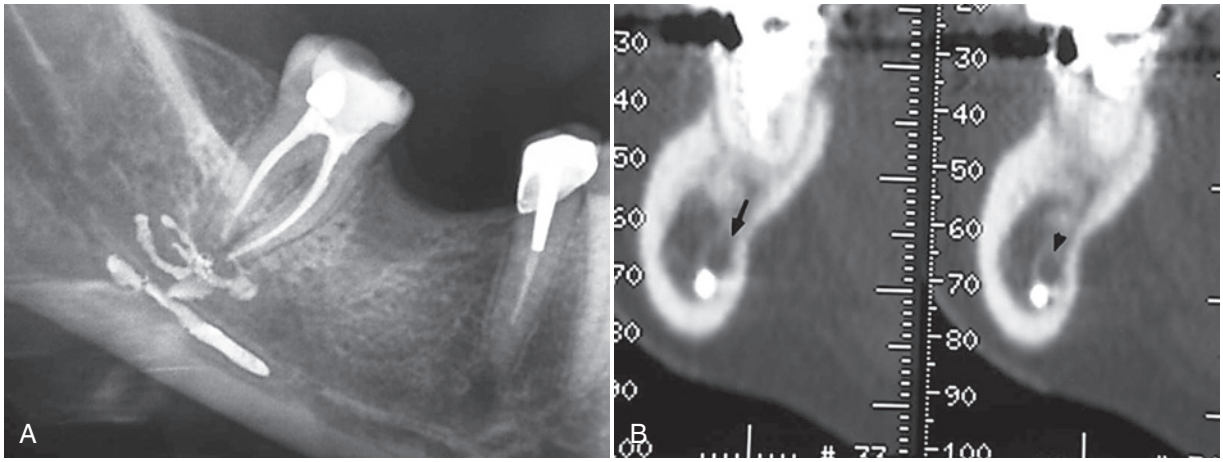

FIG. 19-27 **A**, Panoramic radiograph demonstrating significant overfilling and extension in the radicular vacuoles and overlying the mandibular canal. **B**, A coronal CT section demonstrating that the extruded material is lateral and below the mandibular bundle *(arrows).* The patient's sensory complications were temporary. (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

located apically to the second premolar,[190,211–213] it can vary in anterior-posterior locations,[139,193] coronal-apical locations,[77] and number. Pogrel[215] concluded that the severity of the injury was proportional to the process by which the injury was created, and these processes included scalpel blade wounding/transection, crushing, and stretching damages. These would be analogous to the dissection and reflection of the flap, improper retraction location/application, and postoperative edema.

If the transected ends are closely approximated following the injury, either naturally or surgically, proliferating axons will attempt to bridge the gap and, if they find a perineural tube to populate, gradually restore function. However, in cases where the gap is beyond a critical length, a neuroma may develop at the proximal end. Neuromas consist of dense fibrous tissue intermingled with nerve fascicles, with a disproportionate proliferation of unmyelinated fibers. Treatment, if initiated within 3 months of the initial event, often entails resection of the neuroma and repair by tension-free anastomosis or nerve grafting.

Careful presurgical assessment via radiographs and CBCT provides critical information on the size, location, and spatial relation of the planned surgery to nearby trigeminal nerves.[27] Before exiting the mandibular canal, the nerve often loops anteriorly, then superior and posteriorly in the apical portion of the premolar region.[100] If an apical procedure is planned in an area commensurate with this anatomic variant, then CBCT is warranted and strongly advised. Controlled reflection of the tissue overlying the suspected location of the foramen will diminish the incidence of transection. Cautious reflection of the flap will reveal evidence of tissue "funneling" at its base (Fig. 19-28); this is the telltale sign of the exiting nerve bundle.[194] This exiting tissue, upon discovery, can be marked with a dot of gentian violet or methylene blue dye. By highlighting it in this fashion, the surgeon can avoid unintentional impingement with a retractor. The tissues should be minimally distended and the underside of the flap kept well hydrated.

Transient paresthesia may occur in cases where a protracted surgical procedure, inadvertent stretching, or excessive postoperative edema has occurred. Appropriate reevaluation appointments include neurosensory testing and mapping the outline of the sensory deficit. Return of sensation within the first 4 weeks indicates a neuropraxia and implies an excellent prognosis.

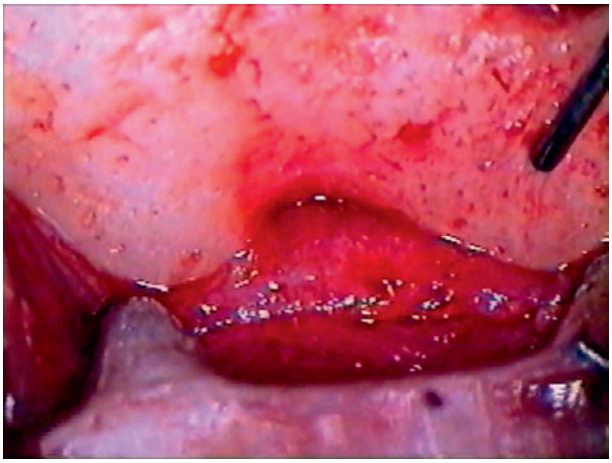

FIG. 19-28 Full-thickness reflection of the flap in the region of the premolar root apices reveals the mental nerve exiting the foramen; note how the tissue "funnels" from the reflected flap to the foramen. (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

Prognosis

Although the prognosis in the case of the IAN damage caused by resorbable or inert material is favorable, as it is dissolved over time, the toxicity of the material, whether chemical or pH, may cause permanent damage if left in place.[195] In addition, pressure from the material on the IAN may be caused by a hematoma or inflammatory, which can also contribute to temporary or permanent IAN damage.[72,195]

The prognosis of the IAN damage depends on the severity of the direct injury to the nerve, toxicity of the irritant, the amount and speed of its resorption, and how rapidly the causative agent is removed.[195,198,214] Because the paresthesia can be of both a mechanical and a chemical nature, removal of the excess material might not be sufficient if the nerve fibers have already undergone degeneration from a chemical irritant. Nevertheless, paresthesia caused by an instant irritation of the nerve, like a minor overinstrumentation or swelling from infection or minor inflammation, usually subsides within days. If no signs of healing are observed within 6 months, the chances of healing are considered much lower, although normal sensory function might still return after this time.[94,288] Counseling and therapeutic management are indicated within 48 hours for injuries caused by endodontic treatment or local anesthetic injections.[81]

Radiographs, especially CBCT, are often useful in revealing the extent of tissue damage in relation to hard tissues (e.g., the size and location of the lesion or the location of extruded materials in relation to the mandibular canal or the maxillary sinus).[26,85,94]

Prevention of Complications Due to Nerve Injury

Prevention of complications during root canal treatment should be the main focus, as once permanent nerve injury has occurred the patient may not regain normal sensation.[307] Careful consideration should also focus on the distance between the root apices and the mandibular canal. For mandibular first molars, this distance varies between 1 and 4 mm, and for mandibular second and third molars, the distance can be less than 1 mm.[288] With mandibular premolars, the proximity of a mental foramen should always be considered.[192] In addition, a study using CBCT has shown that the mesiobuccal root apex of maxillary second molars is frequently in very close proximity with the sinus floor[199] (Fig. 19-29).

CERVICOFACIAL SUBCUTANEOUS EMPHYSEMA

Subcutaneous emphysema is defined as the penetration of air or other gases beneath the skin and submucosa, resulting in soft tissue distention. A specific type, termed *cervicofacial subcutaneous emphysema* (CFSE), is a relatively rare occurrence and may be limited to traumatic, iatrogenic, or spontaneous events.

The first recorded instance was reported by Turnbull in 1900.[293] It details the account of a musician inducing CFSE by

playing his bugle immediately after the extraction of a mandibular tooth; the subsequent swelling resolved a few days after he ceased playing the instrument. Indeed, most early reports of CFSE usually follow tooth extractions and were the result of activities the patient engaged in that raised intraoral pressures.[259] With the introduction of air-driven handpieces, there was an increased risk of CFSE.[11,54,314] Although tooth extraction, especially of mandibular third molars, remains the most commonly reported reason for CFSE,[41,54,123,237,312,314] it can also result from restorative treatment,[40,86,136,275,295] periodontal surgery,[81,264] crown and bridge treatments,[97,318] and root canal therapy.[25,127,138,207,278,295,308]

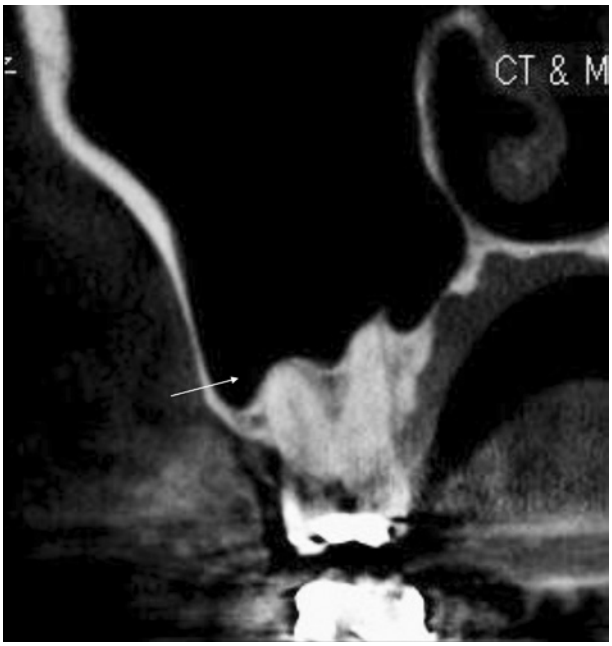

FIG. 19-29 CBCT image in coronal view showing that the mesiobuccal root apex of a maxillary second molar is in close proximity with the sinus floor.

Compressed air, delivered through an air syringe, a high-speed handpiece, or a combination of the two during the operative procedure is associated with 71% of the cases.[117] A more recent study[12] reviewing 47 computed tomography (CT)–documented cases of CFSE found the exclusive cause was the high-speed handpiece in 66% of these patients. Other means of introducing air into the facial spaces include the following:

- Use of the dental laser (Er:YAG) in the gingival pocket: compressed air is used to cool the tip but is of sufficient pressure to dissect the sulcular wound.[4]
- Hydrogen peroxide beyond the canal space: most often reported when the fluid is introduced through a perforation in the canal[25] or over instrumented apex.[138]
- Lack of rubber dam isolation: a compressed air syringe was implemented to increase visibility in these molar retreatments, with air infiltration through the gingival sulcus.[60,146]
- Barotrauma secondary to Boerhaave syndrome: spontaneous rupture of the distal esophagus as a result of intractable vomiting, causing an increase in intraluminal esophageal pressure.[103]

The deposition of air beyond the canal space is a function of both the diameter of the canal at the apex[64] and the position of the syringe tip.[263] An air emphysema has the potential to spread along the facial planes in a similar manner (Fig. 19-30 and Fig. 19-31). Air entering the parapharyngeal and retropharyngeal spaces can lead to soft tissue infections,[73] airway compromise,[230] optic nerve damage,[35] and even death.[55,231] The majority of the cases, however, present as a soft, skin-colored swelling without redness that occurs during or shortly after the dental appointment. The cervicofacial swelling is always associated with crepitus (a crunchy feeling to the tissues upon

FIG. 19-30 A, Facial presentation after the rubber dam was removed. B, Close-up view reveals marked distention of the left suborbital region, with ptosis of the eye and loss of the nasolabial fold. There was marked crepitus upon palpation, but the patient was pain-free. C, She was placed on an antibiotic regime, as per the protocol, 2 days after the incident, the emphysema has resolved and the tissues are normal in color and texture.

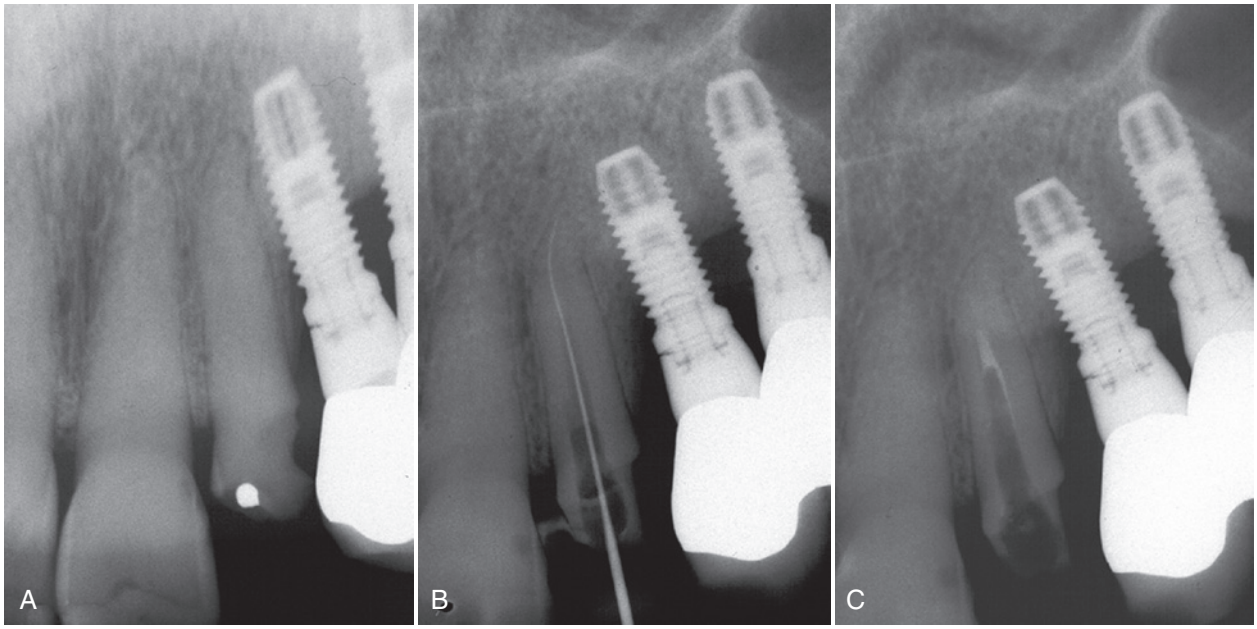

FIG. 19-31 A, Preoperative radiograph tooth #10[136]; dystrophic calcification has obliterated the pulp chamber and the coronal half of the canal space. B, A #10 file inserted to estimated working length; the file is actually through a facial perforation of the root at the radicular extent of the ultrasonic excavation. Repeated drying of the canal space using an air syringe forced air through the perforation site and precipitated the emphysema seen in Fig. 19-26 (*A* and *B*). C, The radicular perforation site was sealed with mineral trioxide aggregate (MTA) before the canal space was obturated. The tooth has remained asymptomatic since the incident.

palpation), which may not be palpable before a latency period of several hours. The more serious presentation of pneumomediastinum is characterized by dyspnea with a brassy voice, chest or back pain, and Hamman sign (a bubbling or crunching sound caused by air or movement accompanied by cardiac pulsation).[109] Secondary complications such as pyogenic mediastinitis or necrotizing fasciitis must be considered, and appropriate means taken to evaluate and manage them (blood cultures, broad-spectrum intravenous antibiotics).[60] Differential diagnosis should include an allergic reaction (more pronounced dermal signs prior to cardiac/respiratory symptoms), hematoma (a rapid accumulation of fluid without initial discoloration and without crepitation), and angioneurotic edema (a defined, circumscribed area of edema, often preceded by a burning sensation in the mucous membranes or skin).[81]

Treatment is largely empirical and dependent on the extent of the CFSE. Antibiotics are usually prescribed due to the nonsterility of the air supply and the possibility of necrotic debris or microorganisms forced into surrounding tissues.[230] Administration of 100% oxygen via a non-rebreather mask is reported to hasten the resolution of the emphysema because the oxygen, which replaces the trapped air, is more readily absorbed at the site.[146] More severe presentations warrant hospitalization with intravenous antibiotics, where the extent and regression of the CFSE can be closely monitored using CT scans and complications can be managed more effectively.[31] Resolution, barring any severe complications, is characteristically rapid and uneventful, with the patients returning to their normal appearance within 4 to 7 days. A reevaluation of the cause of the emphysema should be examined and corrected before treatment resumes, especially in instances of a perforation.

From an endodontic perspective, minimizing compressed air at the site, from either a high-speed handpiece or an air syringe, is key to preventing this consequence. Isolation with a rubber dam, the use of paper points or high-volume aspiration for drying canal spaces, and avoiding the use of hydrogen peroxide during the procedure will also reduce the risk of introducing or entrapping air beneath the overlying tissues. For endodontic microsurgery, the use of a rear-exhausting high-speed handpiece for root resection as well as ultrasonics for root end preparation would decrease the likelihood of precipitating a CFSE during root end procedures.[16]

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PART III

Expanded Clinical Topics

CHAPTER 20
The Role of Endodontics After Dental Traumatic Injuries

CHAPTER 21
Cracks and Fractures

CHAPTER 22
Restoration of the Endodontically Treated Tooth

CHAPTER 23
Vital Pulp Therapy

EXPERT CONSULT CHAPTERS

CHAPTER 24
Pediatric Endodontics: Endodontic Treatment for the Primary and Young Permanent Dentition

CHAPTER 25
Endodontic and Periodontal Interrelationships

CHAPTER 26
Effects of Age and Systemic Health on Endodontics

CHAPTER 27
Bleaching Procedures

CHAPTER 28
Understanding and Managing the Fearful Dental Patient

CHAPTER 29
Endodontic Records and Legal Responsibilities

CHAPTER 30
Key Principles of Endodontic Practice Management

CHAPTER 20

The Role of Endodontics After Dental Traumatic Injuries

MARTIN TROPE | FREDERIC BARNETT | ASGEIR SIGURDSSON | NOAH CHIVIAN

CHAPTER OUTLINE

Unique Aspects of Dental Trauma
Most Common Types of Dental Trauma
Crown Fractures
Crown-Root Fractures
Root Fractures
Luxation Injuries and Avulsion
Follow-Up After Dental Trauma
Radiographic Examinations
Cone Beam Computed Tomography and Dentoalveolar Trauma
Root Resorption
Horizontal (Transverse) Root Fractures
Luxation Injuries
Crown Fractures
Crown Infraction
Uncomplicated Crown Fracture
Treatment
Complicated Crown Fracture
Treatment
Vital Pulp Therapy: Requirements for Success
Treatment Methods
Treatment of the Nonvital Pulp
Crown-Root Fracture
Root Fracture
Diagnosis and Clinical Presentation
Treatment
Healing Patterns
Treatment of Complications
Luxation Injuries
Definitions
Incidence
Treatment
Biologic Consequences
Clinical Management of the Avulsed Tooth
Consequences of Tooth Avulsion
Treatment Objectives
Clinical Management
Diagnosis and Treatment Planning
Preparation of the Root
Extraoral Dry Time Less Than 60 Minutes
Extraoral Dry Time More Than 60 Minutes
Preparation of the Socket
Splinting
Management of the Soft Tissues
Adjunctive Therapy
Second Visit
Endodontic Treatment
Extraoral Time Less Than 60 Minutes
Extraoral Time More Than 60 Minutes
Temporary Restoration
Root Filling Visit
Permanent Restoration
Follow-Up Care

A traumatic injury to the tooth results in damage to many dental and periradicular structures, making the management and consequences of these injuries multifactorial. Knowledge of the interrelating healing patterns of these tissues is essential. This chapter concentrates on the role of the dentinopulpal complex in the pathogenesis of disease subsequent to dentinal trauma and how treatment of this complex can contribute to favorable healing after an injury.

UNIQUE ASPECTS OF DENTAL TRAUMA

Most dental trauma occurs in the 7- to 12-year-old age group and is mainly due to falls and accidents near home or school.[23,143] It occurs primarily in the anterior region of the mouth, affecting the maxillary more than the mandibular jaw.[28] Serious accidents, such as automobile crashes, can affect any tooth and can occur in all age ranges. In many cases, after a

traumatic dental injury, endodontic treatment is provided to caries-free, single-rooted, young permanent teeth. If quick and correct treatment for these teeth is provided after injury, the potential for a successful endodontic outcome is very good.

MOST COMMON TYPES OF DENTAL TRAUMA

Crown Fractures

Most crown fractures occur in young, caries-free anterior teeth.[95,107] This makes maintaining or regaining pulp vitality essential. Luckily, vital pulp therapy supports a good prognosis in these situations if correct treatment and follow-up procedures are carefully followed.

Crown-Root Fractures

Crown-root fractures are first treated periodontally to ensure that there is a sufficient and good margin to allow restoration. If the tooth can be maintained from a periodontal point of view, then the pulp is treated as for a crown fracture.

Root Fractures

A surprisingly large number of pulps in root-fractured teeth will survive this rather dramatic injury. In almost every case, the apical segment stays vital, and in many cases, the coronal segment stays vital or regains vitality subsequent to the injury. If the coronal segment permanently loses vitality, it should be treated as an immature permanent tooth with nonvital pulp. *The apical segment rarely needs treatment.*

Luxation Injuries and Avulsion

Luxation injuries and avulsion often result in pulp necrosis and damage to the cemental protective layer of the root. The potential complication of pulp infection in a root that has lost its cemental protective layer makes these injuries potentially catastrophic. Correct emergency and follow-up evaluation, which may include timely endodontic treatment, is critical.

FOLLOW-UP AFTER DENTAL TRAUMA

The reader is referred to Chapter 1 for specific descriptions of pulp tests, but a few general statements about pulp tests on traumatized teeth may be helpful in trying to interpret the results.

For decades, controversy has surrounded the validity of thermal and electric tests on traumatized teeth. Only generalized impressions may be gained from these tests following a traumatic injury. They are in reality sensitivity tests for nerve function and do not indicate the presence or absence of blood circulation within the pulpal space. It is assumed that subsequent to traumatic injury, the conduction capability of the nerve endings and/or sensory receptors is sufficiently deranged to inhibit the nerve impulse from an electrical or thermal stimulus. This makes the traumatized tooth vulnerable to false-negative readings from such tests.[123]

Teeth that give a response at the initial examination cannot be assumed to be healthy and to continue to give a response over time. Teeth that yield no response cannot be assumed to have necrotic pulps because they may give a response at later follow-up visits. It has been demonstrated that it may take as long as 9 months for normal blood flow to return to the coronal pulp of a traumatized fully formed tooth. As circulation is restored, the responsiveness to pulp tests returns.[73]

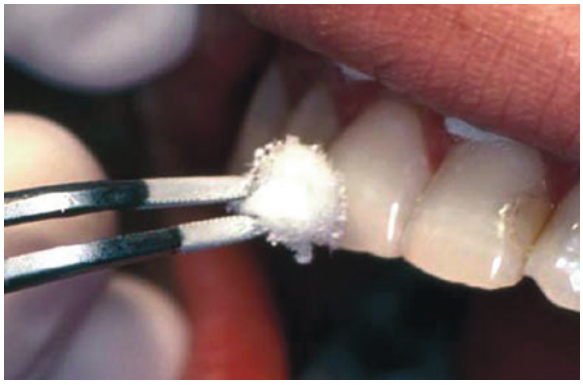

FIG. 20-1 Difluorodichloromethane (–40° F [–40° C]) gas is sprayed on a cotton pellet and then placed on the incisal edge of the maxillary incisor.

The transition from a negative response to a positive response at a subsequent test may be considered a sign of a healing pulp. The repetitious finding of responses may be taken as a sign of a healthy pulp. The transition from a response to no response may be taken as an indication that the pulp is probably undergoing degeneration and therefore an intervention in the form of endodontic treatment might be indicated. The persistence of no response would suggest that the pulp has been irreversibly damaged, but even this is not absolute.[32]

Thermal and electrical pulp tests of all anterior teeth (canine to canine) of the maxillary and mandibular jaws should be performed at the time of the initial examination and carefully recorded to establish a baseline for comparison with subsequent repeated tests in later months. These tests should be repeated at 3 weeks; at 3, 6, and 12 months; and at yearly intervals after the trauma. The purpose of the tests is to establish a trend as to the physiologic status of the pulps of these teeth. Particularly in traumatized teeth, carbon dioxide snow (CO_2, –78° C) or dichlorodifluoromethane (–40° C) placed on the incisal third of the facial surface gives more accurate responses than a water-ice pencil (Fig. 20-1).[71,72] The intense cold seems to penetrate the tooth and covering splints or restorations and reach the deeper areas of the tooth. Neither the dry ice nor the dichlorodifluoromethane spray forms ice water, which could disperse over adjacent teeth or gingiva to give a false-positive response. In trauma evaluation, there is no question that using water-ice pencils should be avoided because of this. Dichlorodifluoromethane spray is a very inexpensive alternative to CO_2 snow; its coldness elicits much more reliable responses than water-ice. The electrical pulp test relies on electrical impulses directly stimulating the nerves of the pulp. These tests have limited value in young teeth but are useful when the dentinal tubules are closed and do not allow dentinal fluid to flow in them. This situation is typical of teeth in elderly patients or in traumatized teeth that are undergoing premature sclerosis. In these situations, the thermal tests that rely on fluid flow in the tubules cannot be used, and the electrical pulp test becomes important.

Laser Doppler flowmetry (LDF) was introduced in the early 1970s for measurement of blood flow in the retina.[88] The technique has also been used to assess blood flow in other tissue systems, such as the skin and renal cortex. It uses a beam of

FIG. 20-2 Laser Doppler machine. (Courtesy Moor Instruments, Devon, United Kingdom.)

infrared (780 to 820 nm) or near-infrared (632.8 nm) light that is directed into the tissue by optical fibers. As light enters the tissue, it is scattered by moving red blood cells and stationary tissue cells. Photons that interact with moving red blood cells are scattered, and the frequency shifts according to the Doppler principle. Photons that interact with stationary tissue cells are scattered but not Doppler shifted. A portion of the light is returned to a photodetector, and a signal is produced (Fig. 20-2).

Attempts have been made to use LDF technology for pulp vitality diagnosis in traumatized teeth because this would provide a more accurate reading of the vitality status of the pulp.[113,175,176] Studies have shown promising results, indicating that laser Doppler can detect blood flow more consistently and earlier than the standard vitality tests would be expected to render a response. In a study on young dogs, laser Doppler was able to correctly detect blood flow as early as 2 to 3 weeks after avulsion of an immature tooth and at the same time indicate no flow in those that would remain necrotic.[176]

Presently the cost of a laser Doppler flowmetry machine limits its use in private dental offices; these are used primarily in hospitals and teaching institutions.

RADIOGRAPHIC EXAMINATIONS

Radiographic imaging is essential for thorough examination, diagnosis, and management of dentoalveolar trauma. Imaging may reveal root fractures, subgingival crown fractures, tooth displacements, bone fractures, root resorptions, and embedded foreign objects. A single radiograph, even a panoramic image, is insufficient to properly diagnose just about any dental trauma case. In its 2012 guidelines for the management of traumatic dental injuries, and in the current recommendations on its interactive website (*www.dentaltraumaguide.org*), the International Association of Dental Traumatology (IADT) has recommended taking at least four different radiographs for almost every injury[65-67]: a direct 90-degree on the axis of the tooth, two with different vertical angulations, and one occlusal film (Fig. 20-3).

Multiple radiographs increase the likelihood of diagnosing root fractures, tooth displacement, and other possible injuries. However, two-dimensional (2D) imaging methods have well-known inherent limitations, and their lack of three-dimensional (3D) information may prevent proper diagnosis and adversely affect long-term treatment outcomes. The interpretation of an

FIG. 20-3 An occlusal film of a luxation injury of central incisors. Both were diagnosed to be laterally luxated with apical translocation. Note that the left central is completely obliterated and has a history of being luxated some years prior.

image can be confounded by a number of factors, including the regional anatomy and superimposition of both the teeth and surrounding dentoalveolar structures. As a result of superimposition, periapical radiographs reveal only limited aspects (i.e., a 2D view) of the true 3D anatomy.[44,119] Additionally, there is often geometric distortion of the anatomic structures imaged with conventional radiographic methods.[3] These problems can be overcome by using cone beam computed tomography (CBCT) imaging techniques, which produce accurate 3D images of the teeth and surrounding dentoalveolar structures and have shown promise in improving the clinician's ability to properly diagnose luxation injuries, alveolar fractures, root fractures, and root resorption (Fig. 20-4).[42,43]

In instances of soft tissue laceration, it is advisable to radiograph the injured area before suturing to be sure that no foreign objects have been embedded. A soft tissue radiograph with a normal-sized film briefly exposed at reduced kilovoltage should reveal the presence of many foreign substances, including tooth fragments (Fig. 20-5).

Cone Beam Computed Tomography and Dentoalveolar Trauma

CBCT is accomplished by using a rotating gantry to which an x-ray source and detector are fixed (see Chapter 2). A divergent pyramidal or cone-shaped source of ionizing radiation is directed through the middle of the area of interest onto an area x-ray detector on the opposite side of the patient. The x-ray source and detector rotate around a fixed fulcrum within the region of interest. During the exposure sequence, hundreds of planar projection images are acquired of the field of view (FOV) in an arc of at least 180°. In this single rotation, CBCT provides precise, essentially immediate, and accurate 3D radiographic images. Because CBCT exposure incorporates the

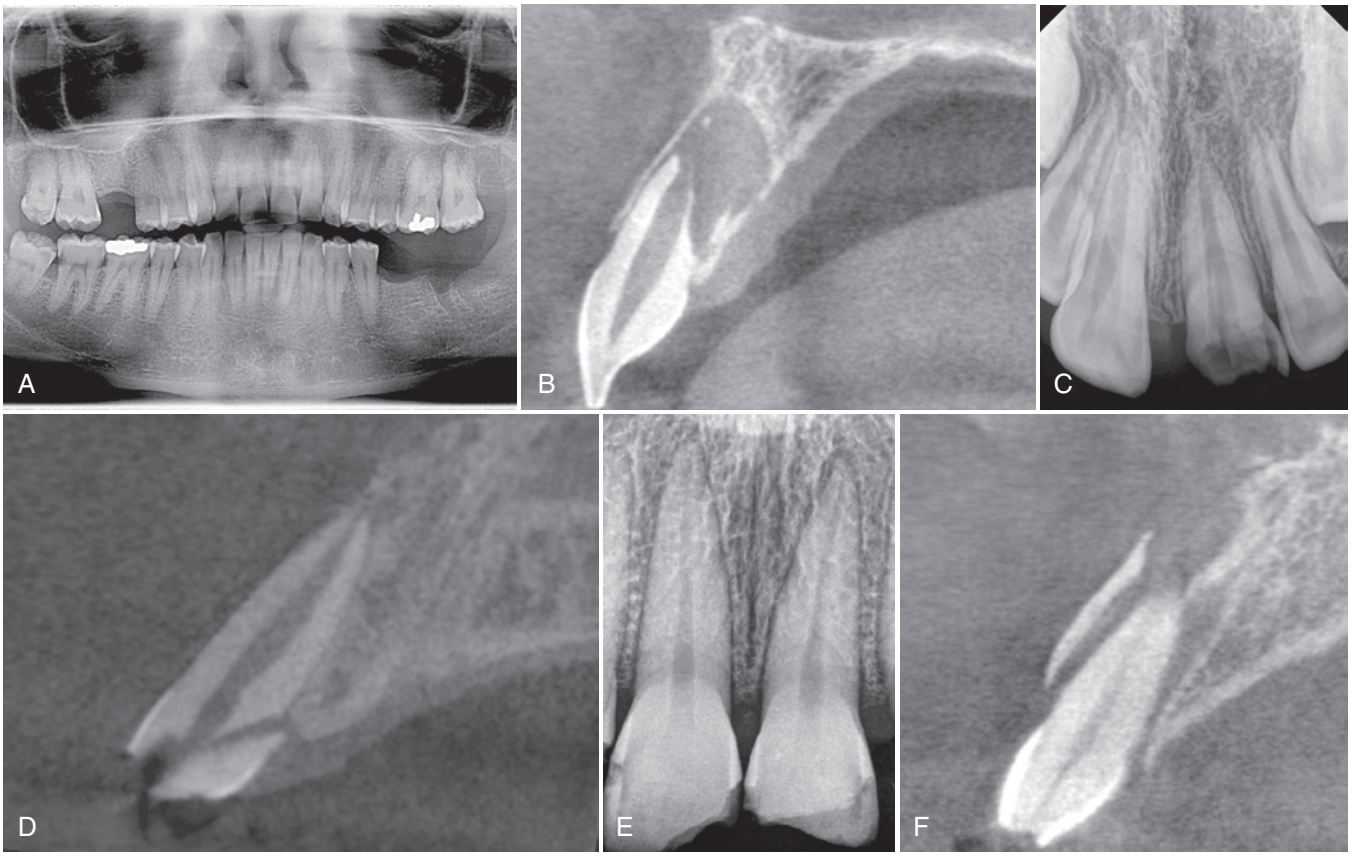

FIG. 20-4 **A,** Panoramic radiograph taken on a patient with past history of dentoalveolar trauma. Tooth #9 appeared to have arrested root development with a periapical radiolucency. **B,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that tooth #9 had arrested root development, a large PA radiolucency, and extensive root resorption along the palatal surface that was *not* evident on the panoramic image. **C,** Periapical radiograph of tooth #9 with extensive complicated crown fracture. The clinical examination revealed the crown fracture extended subgingivally on the palatal aspect. **D,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that the apical extent of the fractured palatal portion extended just apical to the crest of bone. The correct diagnosis of this injury was complicated crown-root fracture. **E,** Periapical radiograph of teeth #8, #9 revealed crown fractures. **F,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that tooth #9 suffered a lateral luxation displacement injury with extensive concomitant alveolar fracture. The necessary treatment of repositioning and splinting was apparent.

Continued

entire FOV, only one rotational sequence of the gantry is necessary to acquire enough data for image reconstruction.

CBCT has been suggested as an adjunct imaging tool when the true nature of the dentoalveolar root fracture and dental injuries cannot be confidently diagnosed from a conventional examination and radiographs.[43,111,120] However, it has been repeatedly demonstrated that the use of CBCT provides improved diagnostic images in cases of dentoalveolar injury.* Perhaps the use of CBCT imaging for dentoalveolar injuries will be considered "best practice" sometime in the near future.

Root Resorption

Because the development and progression of root resorption occur without clinical signs or symptoms, their early detection is challenging. The definitive diagnosis of root resorption, therefore, depends on its radiographic demonstration, which in turn is limited by the diagnostic accuracy of the imaging device used to determine its presence (see Chapter 16).[120] The radiodensity of the root requires that a significant amount of root substance be removed to cause enough contrast on the radiograph to allow it to be detected. Thus, only resorptive defects on the mesial or distal aspects of the root can be predictably detected after some time; the facial and palatal or lingual aspects are much harder to see. To overcome these difficulties, it is essential to take as many different horizontal-angled radiographs as feasible in cases of suspected root resorption. Early detection of small resorption defects has been shown to be poor with conventional dental radiographs, and the extent of the resorptive defect is grossly underestimated compared to CBCT.[31,57,61]

The available literature supports the use of CBCT as a diagnostic tool to assess the true nature of teeth diagnosed with root resorption to improve diagnosis and aid management. This should ultimately improve the prognosis of teeth with

*References 30, 31, 42-44, 57, 61, 103, 111, 119, and 120.

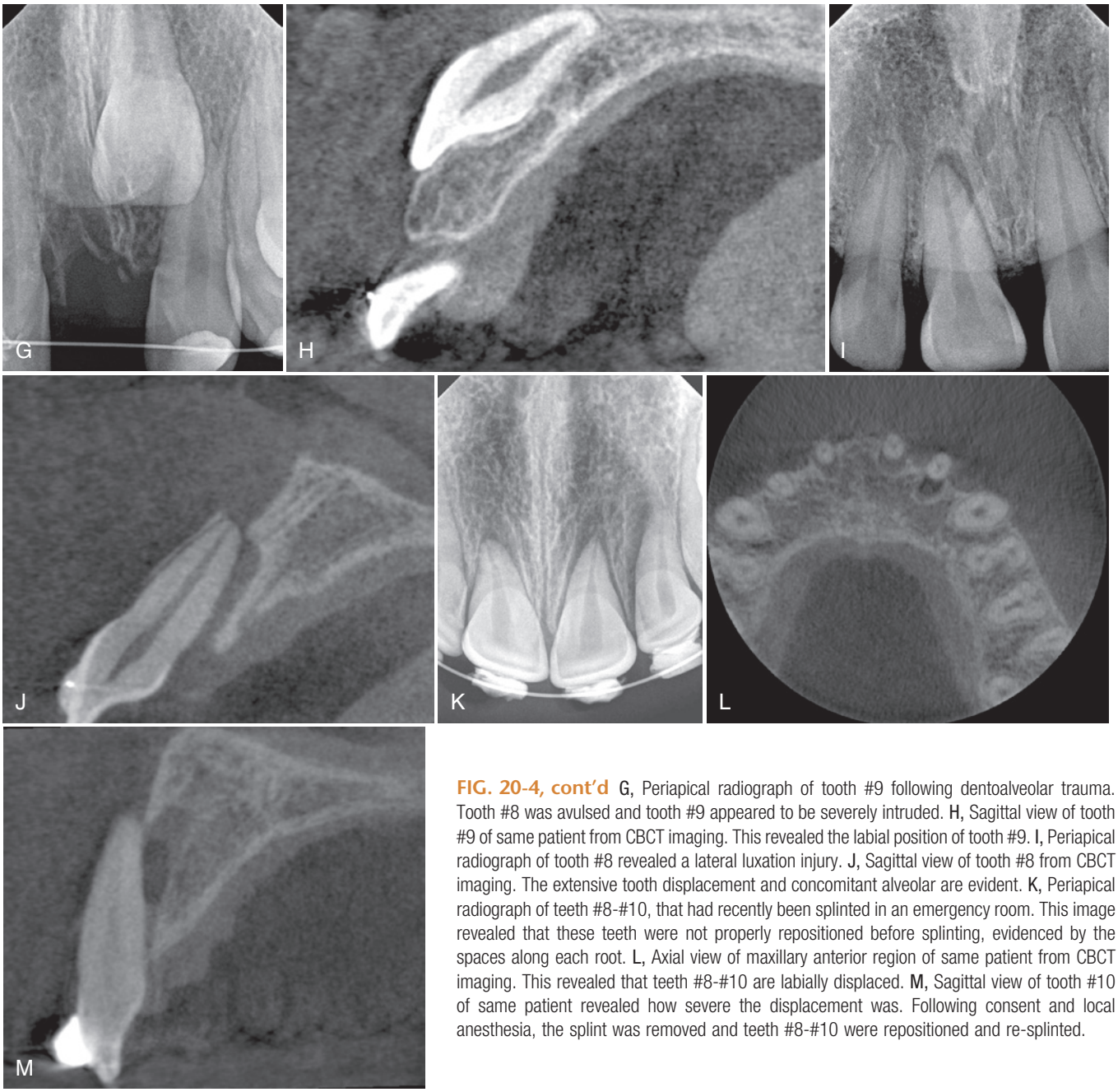

FIG. 20-4, cont'd **G,** Periapical radiograph of tooth #9 following dentoalveolar trauma. Tooth #8 was avulsed and tooth #9 appeared to be severely intruded. **H,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed the labial position of tooth #9. **I,** Periapical radiograph of tooth #8 revealed a lateral luxation injury. **J,** Sagittal view of tooth #8 from CBCT imaging. The extensive tooth displacement and concomitant alveolar are evident. **K,** Periapical radiograph of teeth #8-#10, that had recently been splinted in an emergency room. This image revealed that these teeth were not properly repositioned before splinting, evidenced by the spaces along each root. **L,** Axial view of maxillary anterior region of same patient from CBCT imaging. This revealed that teeth #8-#10 are labially displaced. **M,** Sagittal view of tooth #10 of same patient revealed how severe the displacement was. Following consent and local anesthesia, the splint was removed and teeth #8-#10 were repositioned and re-splinted.

root resorption that require endodontic management (see Fig. 20-4, *A, B*).[120]

Horizontal (Transverse) Root Fractures

There is a significant risk of misdiagnosing the true location of a root fracture in anterior teeth when intraoral radiography is used because of the possibility of the oblique course of the fracture line in the sagittal plane. It has been shown that horizontal root fracture can be detected sooner using CBCT than with periapical views, and the fracture can be assessed in coronal, axial and cross-sectional views (see Fig. 20-4, *C, D*).[98] Compared to conventional radiographs, CBCT increased the accuracy of diagnosing the actual nature of horizontal root fractures.[30,111]

Luxation Injuries

As previously mentioned, conventional intraoral radiography provides poor sensitivity in the detection of minimal tooth displacements and root and alveolar fractures.[103] CBCT has significantly improved the ability to accurately diagnose traumatic injuries and has the potential to overcome most of the technical limitations of the plain-film projection (see Fig. 20-4, *E-M*).*

*References 30, 31, 42, 43, 57, 61, 98, 103, 111, and 120.

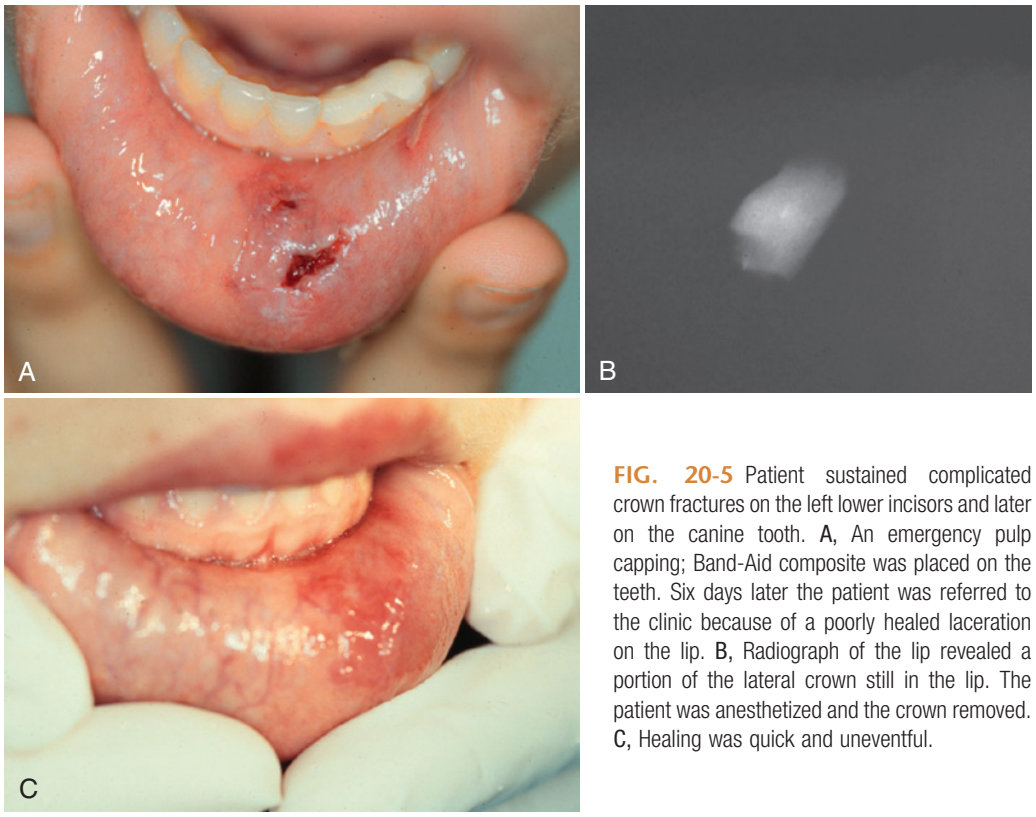

FIG. 20-5 Patient sustained complicated crown fractures on the left lower incisors and later on the canine tooth. **A,** An emergency pulp capping; Band-Aid composite was placed on the teeth. Six days later the patient was referred to the clinic because of a poorly healed laceration on the lip. **B,** Radiograph of the lip revealed a portion of the lateral crown still in the lip. The patient was anesthetized and the crown removed. **C,** Healing was quick and uneventful.

CROWN FRACTURES

As already mentioned, the primary aim from an endodontic point of view is to maintain pulp vitality after crown fractures.

Crown Infraction

Crown infraction can be defined as an incomplete fracture of or a crack in the enamel, without loss of tooth structure.[16]

Biologic Consequences

Crown infractions are injuries that carry little danger of resulting in pulp necrosis. Meticulous follow-up over a 5-year period is the most important endodontic preventive measure in these cases. If, at any follow-up examination, the reaction to sensitivity tests changes, or if, on radiographic assessment, signs of apical or periradicular periodontitis develop or the root appears to have stopped development or is obliterating, endodontic intervention should be considered.

Uncomplicated Crown Fracture

Uncomplicated crown fracture can be defined as fracture of the enamel only or the enamel and dentin without pulp exposure.[16]

Incidence

Uncomplicated crown fracture is likely to be the most commonly reported dental injury. It is estimated to account for at least one third to one half of all reported dental trauma.

Biologic Consequences

Uncomplicated crown fractures are also injuries that have little danger of resulting in pulp necrosis. In fact, the biggest danger to the health of the pulp is through iatrogenic causes during the aesthetic restoration of these teeth.

Treatment

There are two key issues in the treatment of crown fractures. First, all exposed dental tubules need to be closed as soon as reasonably possible. If the broken-off piece is not available or if it is not possible to reattach it and there is no time to do a full composite restoration at the time of the emergency appointment, a composite Band-Aid or temporary coverage should be placed on all exposed dentin. This prevents any ingress of bacteria into the tubules and reduces the patient's discomfort. The second issue is the remaining dentin thickness. Several studies have confirmed that if the remaining dentin is more than 0.5 mm thick, the tooth can be restored with the restoration of choice, including etching and bonding, and no special attention needs to be given to the pulp.[3,46,60,115,151] However, if the remaining dentin is less than this thickness, a protective layer of hard-setting calcium hydroxide in the deepest part of the dentin exposure does reduce, if not completely prevent, reactive inflammation of the underlying pulp, which is a significantly different reaction compared to the negative reaction to composite bonding systems.[3,46,60,115,151]

Complicated Crown Fracture

A *complicated crown fracture* involves enamel, dentin, and pulp (Fig. 20-6).[1]

FIG. 20-6 Complicated crown fracture involving enamel, dentin, and pulp.

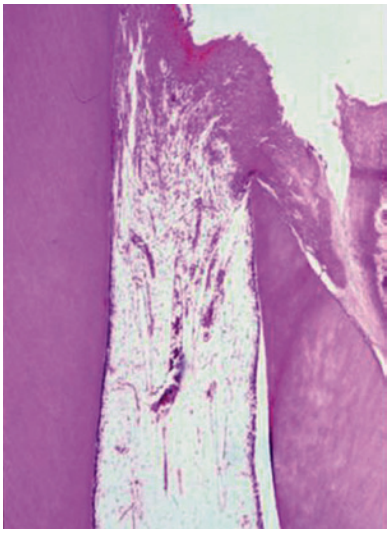

FIG. 20-7 Histologic appearance of the pulp within 24 hours of a traumatic exposure. The pulp proliferated over the exposed dentinal tubules. There is approximately 1.5 mm of inflamed pulp below the surface of the fracture.

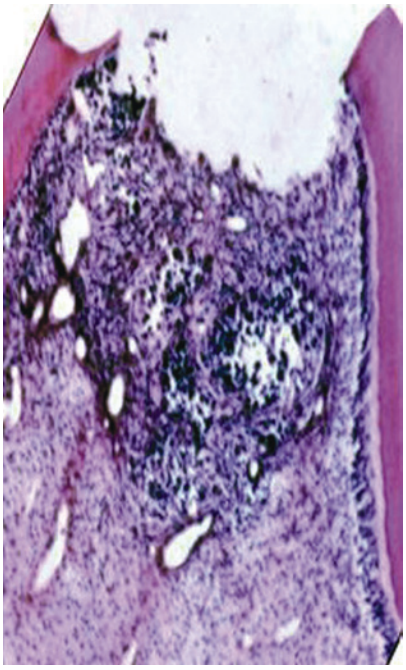

FIG. 20-8 Histologic appearance of the pulp days after a traumatic exposure. Superficial necrosis above a zone of inflamed pulp is seen.

Incidence

Complicated crown fractures occur in 0.9% to 13% of all dental injuries.[40,130,153]

Biologic Consequences

A crown fracture involving the pulp, if left untreated, always results in pulp necrosis.[97] However, the manner and time sequence in which the pulp becomes necrotic allow a great deal of potential for successful intervention to maintain pulp vitality. The first reaction after the injury is hemorrhage and local inflammation (Fig. 20-7).

Subsequent inflammatory changes are usually proliferative but can be destructive over time. A proliferative reaction is favored in traumatic injuries because the fractured surface is usually flat, allowing salivary rinsing with little chance of impaction of contaminated debris. Unless impaction of contaminated debris is obvious, it is expected that in the first 24 hours after the injury, a proliferative response with inflammation extending not more than 2 mm into the pulp will be present (see Fig. 20-7).[49,52,82] In time, the bacterial challenge results in local pulpal necrosis and a slow apical progression of the pulpal inflammation (Fig. 20-8).

Treatment

Treatment options for complicated crown fracture are (1) vital pulp therapy, comprising pulp capping, partial pulpotomy, or full pulpotomy; and (2) pulpectomy. The choice of treatment depends on the stage of development of the tooth, the time between trauma and treatment, concomitant periodontal injury, and the restorative treatment plan.

Stage of Development of the Tooth

Loss of vitality in an immature tooth can have catastrophic consequences. Root canal treatment on a tooth with a blunderbuss canal is time consuming and difficult. It is probably more important that necrosis of an immature tooth leaves it with thin dentinal walls that are susceptible to fracture both during and after the apexification procedure.[99] Every effort must be made to keep the tooth vital, at least until the apex and cervical root have completed their development.

Removal of the pulp in a mature tooth is not as significant as in an immature tooth because a pulpectomy in a mature

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tooth has an extremely high success rate.[142] However, it has been shown that under optimal conditions, vital pulp therapy (rather than removal) can be carried out successfully on a mature tooth.[110,172] Therefore, this form of therapy can be an option under certain circumstances, even though a pulpectomy is the treatment that affords the most predictable success.

In an immature tooth, vital pulp therapy should always be attempted if at all feasible because of the tremendous advantages of maintaining the vital pulp.

Time Between Trauma and Treatment

For 48 hours after a traumatic injury, the initial reaction of the pulp is proliferative, with no more than a 2-mm depth of pulpal inflammation (see Fig. 20-8). After 48 hours, chances of direct bacterial contamination of the pulp increase, with the zone of inflammation progressing apically[52]; as time passes, the likelihood of successfully maintaining a healthy pulp decreases.

Concomitant Attachment Damage

A periodontal injury compromises the nutritional supply of the pulp. This fact is particularly important in mature teeth, in which the chance of pulp survival is not as good as for immature teeth.[13,59]

Restorative Treatment Plan

Unlike in an immature tooth, in which the benefits of maintaining vitality of the pulp are so great, pulpectomy is a viable treatment option in a mature tooth. As pointed out, if performed under optimal conditions, vital pulp therapy after traumatic exposures can be successful. If the restorative treatment plan is simple and a composite resin restoration will suffice as the permanent restoration, this treatment option should be given serious consideration. If a more complex restoration is to be placed (e.g., a crown or bridge abutment), pulpectomy may be the more predictable treatment method.

Vital Pulp Therapy: Requirements for Success

Vital pulp therapy has an extremely high success rate if the following requirements can be met (also see Chapter 23).

- *Treatment of a noninflamed pulp*. Treatment of a healthy pulp has been shown to be an important requirement for successful therapy.[152,162] Vital pulp therapy of the inflamed pulp yields an inferior success rate,[152,162] so the optimal time for treatment is in the first 24 hours when pulp inflammation is superficial. As time between the injury and therapy increases, pulp removal must be extended apically to ensure that noninflamed pulp has been reached.
- *Bacteria-tight seal*. In our opinion, this requirement is the most critical factor for successful treatment.[152] Challenge by bacteria during the healing phase causes failure (see also Chapter 14).[45] If the exposed pulp is effectively sealed from bacterial leakage, successful healing of the pulp with a hard tissue barrier will occur independent of the dressing placed on the pulp and after more extended time periods between accident and treatment.[46,80]
- *Pulp dressing*. Calcium hydroxide has traditionally been used for vital pulp therapy. Its main advantage is that it is antibacterial[39,141] and disinfects the superficial pulp. Pure calcium hydroxide causes necrosis of about 1.5 mm of pulp tissue, which removes superficial layers of inflamed pulp if present (Fig. 20-9).[112] The high pH (12.5) of calcium hydroxide causes a liquefaction necrosis in the most superficial layers.[134] The toxicity of calcium hydroxide appears to be neutralized as the deeper layers of pulp are affected, causing a coagulative necrosis at the junction of the necrotic and vital pulp, resulting in only mild irritation. This mild irritation initiates an inflammatory response, and in the absence of

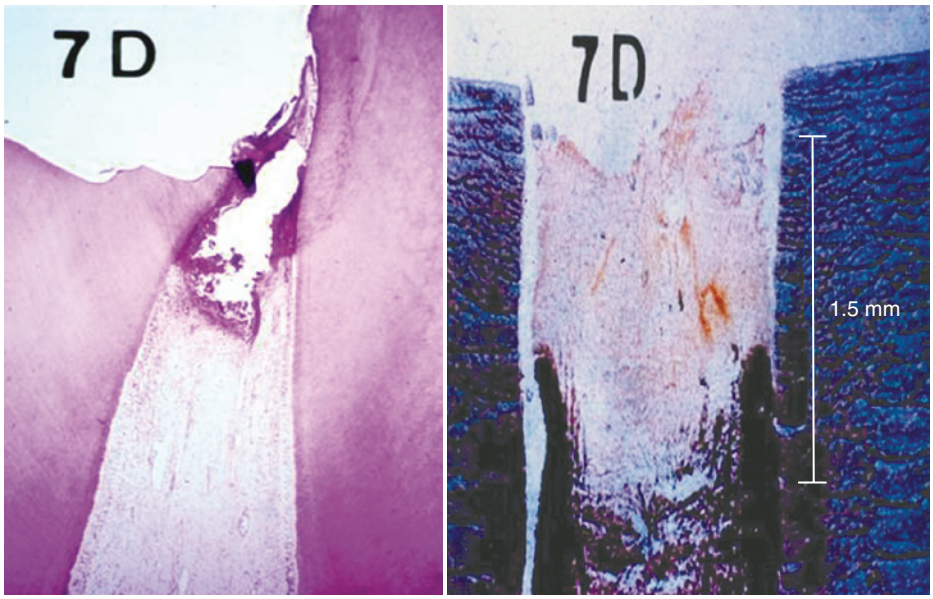

FIG. 20-9 Pulp necrosis of 1.5 mm as a result of the high pH of calcium hydroxide.

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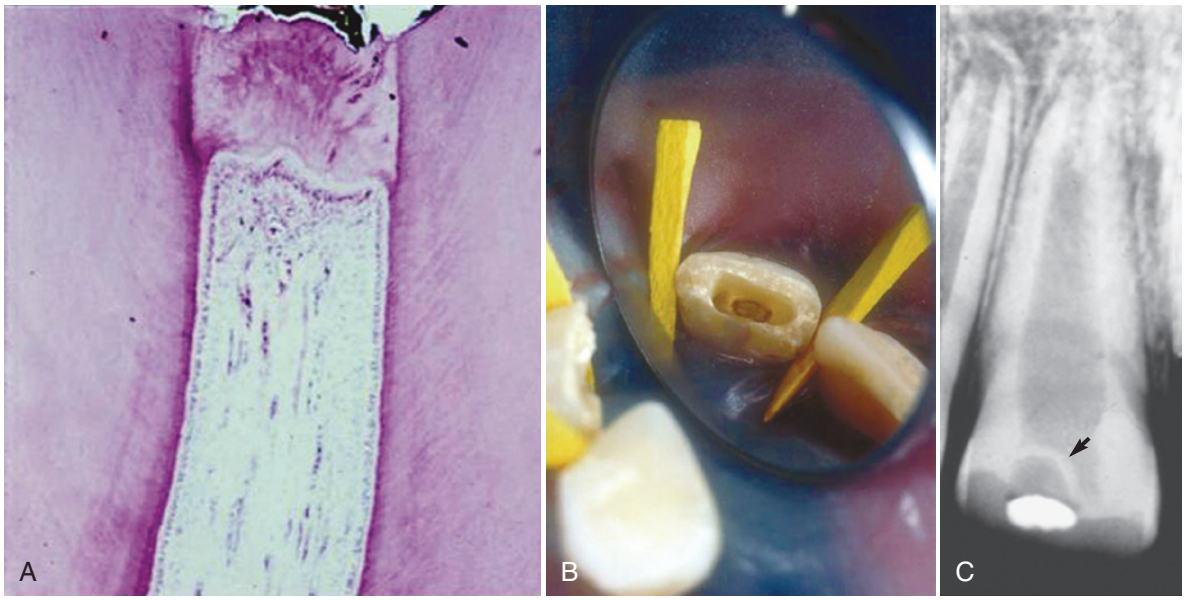

FIG. 20-10 Hard tissue barrier after calcium hydroxide partial pulpotomy. **A,** Histologic appearance of replacement odontoblasts and a hard tissue barrier. **B,** Clinical appearance of the barrier on removal of the coronal restoration 3 months after placement of the calcium hydroxide. **C,** Radiographic appearance of the hard tissue barrier.

bacteria,[134] the pulp will heal with a hard tissue barrier (Fig. 20-10).[133,134] Hard-setting calcium hydroxide does not cause necrosis of the superficial layers of pulp; it has also been shown to initiate healing with a hard tissue barrier.[150,159]

The major disadvantage of calcium hydroxide is that it does not seal the fractured surface. Therefore, an additional material must be used to ensure that bacteria do not challenge the pulp, particularly during the critical healing phase.

Many materials, such as zinc oxide eugenol,[25,162] tricalcium phosphate,[84] and composite resin,[24] have been proposed as medicaments for vital pulp therapy. None to this date have afforded the predictability of calcium hydroxide used in conjunction with a well-sealed coronal restoration.[60,102,139,151] In a dog study, for example, pulps capped directly with various adhesive agents showed moderate to severe inflammatory reactions, with progressive extension of tissue necrosis with time and total absence of continuous hard tissue bridge formation.[102] Application of a calcium hydroxide–based material was characterized by inflammatory cell infiltration, limited tissue necrosis, and partial to complete hard tissue bridging.[102] In a similar study on human teeth in vivo, it was found that Scotchbond Multi-Purpose Plus (3M, St. Paul, MN) caused inflammatory changes when applied directly to exposed pulp tissue; however, direct capping with Dycal followed by sealing with Scotchbond Multi-Purpose Plus showed favorable results in pulp tissue.[151]

Currently, bioceramic materials are considered the pulp capping agents of choice.[4] Mineral trioxide aggregate (MTA), a first-generation bioceramic material, has been shown to be a good pulp capping agent.[62,116,118,174] It has a high pH, similar to that of calcium hydroxide, when unset[156] and after setting creates an excellent bacteria-tight seal.[157] It is also hard enough to act as a base for a final restoration.[156] Yet MTA does not enjoy the same popularity as calcium hydroxide as a pulp capping agent in the treatment of traumatic exposures. There are probably three main reasons. The first is likely to be that because MTA needs a moist environment for at least 6 hours to set properly, the treatment becomes a two-step procedure, compared with a one-step procedure for other medicaments. Using MTA as a pulp capping agent thus necessitates that a wet cotton pellet be placed over it until it is set, and then the permanent restoration can be fabricated at a later time. The second likely reason is that originally, MTA was gray in color and reported to cause discoloration in the tooth crown when used as a capping agent in anterior teeth.[37] To counter this discoloration problem, a new white version of MTA was marketed a few years ago. Initially there were some concerns that the pulp did not respond to the white version as favorably as it had to the gray one.[121] Relatively few studies have been done comparing the white MTA to the gray one, but most have indicated that the pulp seems to react as well to one as the other.[91,116,131] Unfortunately, the white MTA has been found to discolor in a similar fashion to the gray, presumably because of the bismuth oxide filler in the material.

The third likely reason is that recently, newer generation bioceramic materials have come to the market that have the same positive properties as MTA but not the disadvantages described previously. They set quickly enough for a one-visit procedure and do not discolor the tooth. These materials are now considered superior to calcium hydroxide as the capping agent for traumatic pulp exposures.

Treatment Methods

Pulp Capping

Pulp capping implies placing the dressing directly on the pulp exposure without any removal of the soft tissue.[24]

Indications

There are few indications for pulp capping when traumatic pulp exposures are treated. The success rate of this procedure

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(80%),[69,125] compared to partial pulpotomy (95%),[49] indicates that a superficial pulp cap should not be considered after traumatic pulp exposures. The lower success rate is not difficult to understand because superficial inflammation develops soon after the traumatic exposure. If the treatment is at the superficial level, a number of inflamed (rather than healthy) pulps will be treated, lowering the potential for success. In addition, a bacteria-tight coronal seal is much more difficult to attain in superficial pulp capping because there is no depth to the cavity to aid in creating the seal, as there is with a partial pulpotomy.

It must be acknowledged that the studies suggesting that an inflamed pulp is contraindicated for an attempt at pulp capping were performed in the 1970s with amalgam as the standard coronal restoration. If we consider that amalgam leaks and calcium hydroxide washes out in the presence of moisture as the reasons for the poor results, then it may be poor coronal seal, rather than the inflamed pulp, that leads to the failure in these cases. More recent studies suggest that with the bioceramic as the pulp cap, the inflamed pulp is not the impediment as previously thought, but rather that the seal seems to be the major factor for success. Thus this material used as a base in these situations may give us more leeway for capping the inflamed pulp.

However in a situation of a traumatic pulp exposure of a tooth (injuries in which most patients are young [large pulps]), the patient typically presents for treatment within 48 hours because of sensitivity and aesthetic concerns. Therefore, the pulpal inflammation is usually only superficial. In these cases, we still feel it is prudent to remove the superficial layer of inflammation and place a bioceramic agent for pulp capping.

Partial Pulpotomy

Partial pulpotomy implies the removal of coronal pulp tissue to the level of healthy pulp. Knowledge of the reaction of the pulp after a traumatic injury makes it possible to accurately determine this level. This procedure is commonly called the *Cvek pulpotomy.*

Indications

As for pulp capping.

Technique

Administration of an anesthetic (possibly without a vasoconstrictor), rubber dam placement, and superficial disinfection are performed. A 1- to 2-mm deep cavity is prepared into the pulp, using a high-speed handpiece with a sterile diamond bur of appropriate size and copious water coolant (Fig. 20-11).[77] A slow-speed bur or spoon excavator should be avoided. If bleeding is excessive, the pulp is amputated deeper until only moderate hemorrhage is seen. Excess blood is carefully removed by rinsing with sterile saline, and the area is dried with a sterile cotton pellet. Use of 5% sodium hypochlorite (NaOCl; bleach) has been recommended to rinse the pulpal wound.[46] The bleach causes chemical amputation of the blood coagulum; removes damaged pulp cells, dentin chips, and other debris; and provides hemorrhage control with minimal damage to the "normal" pulp tissue underneath.

Care must be taken not to allow a blood clot to develop because this would compromise the prognosis.[49,133] A thin layer of pure calcium hydroxide is mixed with sterile saline or anesthetic solution to a thick mix and carefully placed on the pulp stump. If the pulp size does not permit additional loss of pulp tissue, a commercial hard-setting calcium hydroxide can be used.[150] The prepared cavity is filled with a material with the best chance of a bacteria-tight seal (zinc oxide eugenol or glass ionomer cement) to a level flush with the fractured surface. The material in the pulpal cavity and all exposed dentinal tubules are etched and restored with bonded composite resin. Alternatively, after hemostasis has been obtained, the pulp can be capped with MTA, a wet cotton pellet can be placed on top of it, and the access can be temporized for the appropriate time. The cotton pellet needs to be removed as early as possible and the tooth then restored with composite restoration.

Follow-Up

The follow-up is the same as for pulp capping. Emphasis is placed on evidence of maintenance of responses to sensitivity tests and radiographic evidence of continued root development (Fig. 20-12).

Prognosis

This method affords many advantages over pulp capping. Superficial inflamed pulp is removed during preparation of the pulpal cavity. Calcium hydroxide disinfects dentin and pulp and removes additional pulpal inflammation. Most important, space is provided for a material that can achieve a bacteria-tight seal to allow pulpal healing with hard tissue under optimal conditions. Additionally, coronal pulp remains, which allows sensitivity testing to be carried out at the follow-up visits. The prognosis is extremely good (94% to 96%).[49,70]

Full Pulpotomy

Full pulpotomy involves removal of the entire coronal pulp to a level of the root orifices. This level of pulp amputation is chosen arbitrarily because of its anatomic convenience. Because inflamed pulp sometimes extends past the canal orifices into the root pulp, many mistakes are made that result in treatment of an inflamed rather than a noninflamed pulp.

Indications

Full pulpotomy is indicated when it is anticipated that the pulp is inflamed to the deeper levels of the coronal pulp. Traumatic exposures after more than 72 hours and carious exposure of a young tooth with a partially developed apex are two examples of cases in which this treatment may be indicated. Because of the likelihood that the dressing will be placed on an inflamed pulp, full pulpotomy is contraindicated in mature teeth. In the immature tooth, benefits outweigh risks for this treatment form only with incompletely formed apices and thin dentinal walls.

Technique

The procedure begins with administration of an anesthetic, rubber dam placement, and superficial disinfection, as for pulp capping and partial pulpotomy. The coronal pulp is removed as in partial pulpotomy, but to the level of the root orifices. A calcium hydroxide dressing, a bacteria-tight seal, and a coronal restoration are carried out as for partial pulpotomy.

Follow-Up

Follow-up is the same as for pulp capping and partial pulpotomy. A major disadvantage of this treatment method is that

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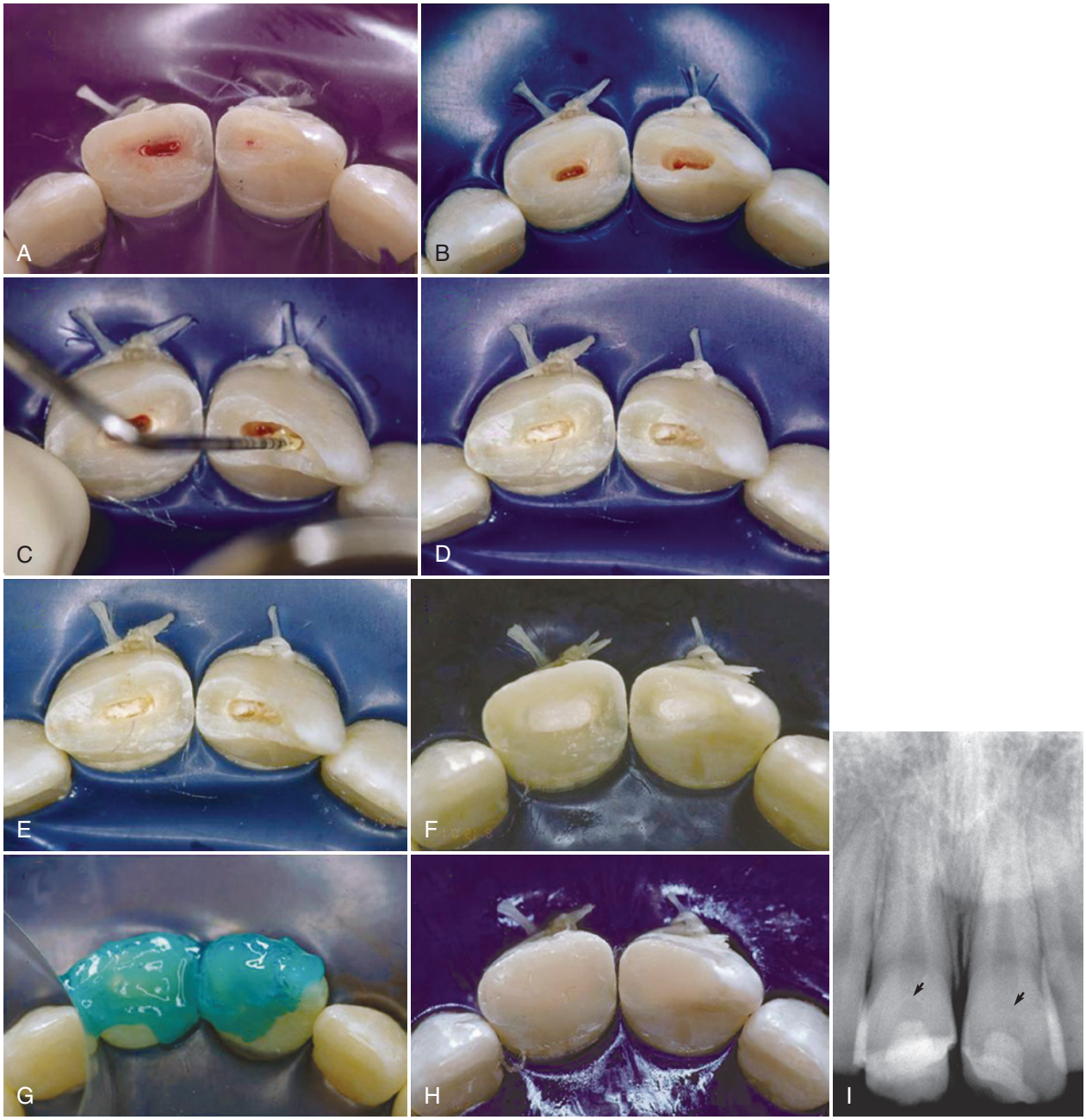

FIG. 20-11 Cvek partial pulpotomy. **A,** The fractured teeth are cleaned and disinfected; a rubber dam is placed. **B,** Cavities are prepared at high speed with a round diamond bur 1 to 2 mm into the pulpal tissue. **C,** Calcium hydroxide on a plugger **(D)** is placed on the soft tissue of the pulp. **E,** Care is taken to avoid smearing the walls of the preparation with the calcium hydroxide. **F,** Cavity preparations are filled with glass ionomer cement. The exposed dentin is etched **(G)** and then covered with composite resin **(H). I,** Radiograph 6 months later shows formation of hard tissue barriers in both teeth. (Courtesy Dr. Alessandra Ritter, Chapel Hill, NC.)

sensitivity testing is not possible, owing to the loss of coronal pulp, so radiographic follow-up is extremely important to assess for signs of apical periodontitis and to ensure the continuation of root formation.

Prognosis

Because cervical pulpotomy is performed on pulps that are expected to have deep inflammation and the site of pulp amputation is arbitrary, many more mistakes are made that lead to treatment of the inflamed pulp. Consequently the prognosis, which is in the range of 75%, is poorer than for partial pulpotomy.[74] Because of the inability to evaluate pulp status after full pulpotomy, some authors have recommended pulpectomy routinely after the roots have fully formed (Fig. 20-13). This philosophy is based on the pulpectomy procedure having a success rate in the range of 95%, whereas if apical periodontitis develops, the prognosis of root canal treatment drops significantly to about 80%.[136,142]

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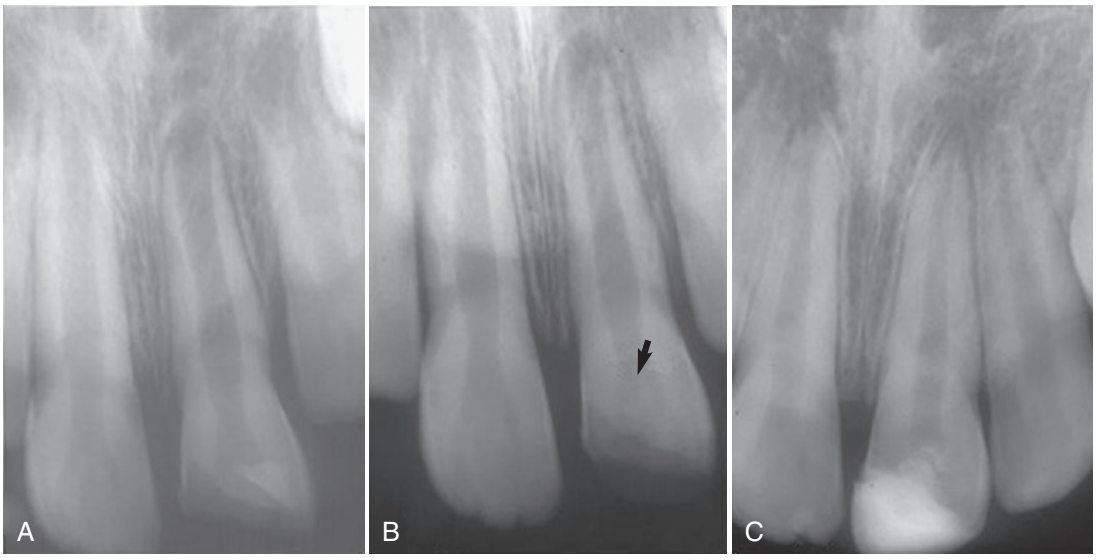

FIG. 20-12 Continued root development after partial pulpotomy. **A,** Radiograph of immature tooth with a complicated crown fracture. **B,** At the time of placement of calcium hydroxide after partial pulpotomy. **C,** Follow-up radiograph confirming that the pulp maintained vitality and the root continued to develop.

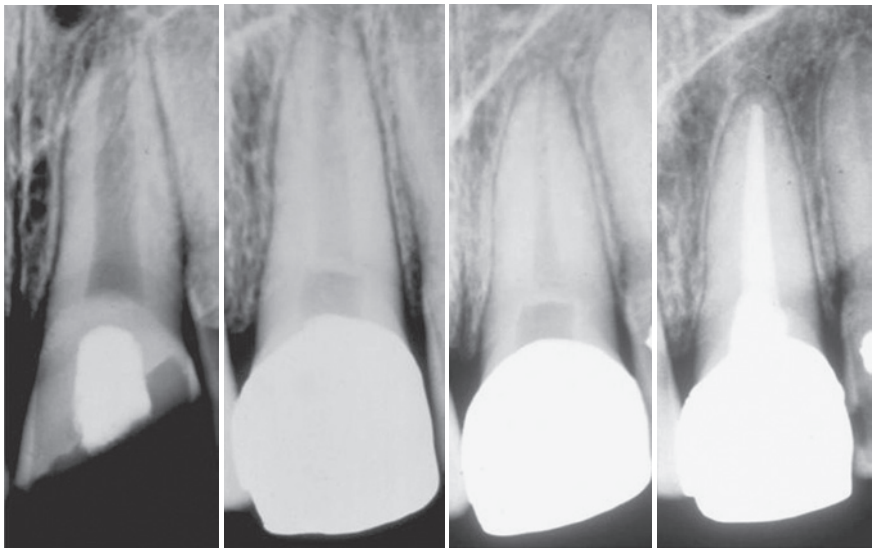

FIG. 20-13 Successful pulpotomy followed by a pulpectomy at 18 months. (Courtesy Dr. Leif Tronstad, Oslo, Norway.)

Pulpectomy

Pulpectomy implies removal of the entire pulp to the level of the apical foramen.

Indications

Pulpectomy is indicated in a complicated crown fracture of mature teeth if conditions are not ideal for vital pulp therapy or if it is foreseeable that restoring the tooth would require placement of a post. This procedure is no different from root canal treatment of a vital nontraumatized tooth.

Treatment of the Nonvital Pulp

Immature Tooth: Apexification

Indications

Apexification should be performed in teeth with open apices and thin dentinal walls in which standard instrumentation techniques cannot create an apical stop to facilitate effective root canal filling.

Biologic Consequences

A nonvital immature tooth presents a number of difficulties for adequate endodontic treatment. The canal is often wider apically than coronally, necessitating the use of a filling technique with softened filling material to mold it to the shape of the apical part of the canal. Because the apex is extremely wide, no barrier exists to stop this softened material from moving into and traumatizing the apical periodontal tissues. Also, the lack of apical stop and extrusion of material through the canal might result in a canal that is underfilled and susceptible to leakage. An additional problem in immature teeth with thin dentinal walls is their susceptibility to fracture, both during and after treatment.[50]

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FIG. 20-14 Creamy mix of calcium hydroxide on a Lentulo spiral instrument ready for placement into the canal.

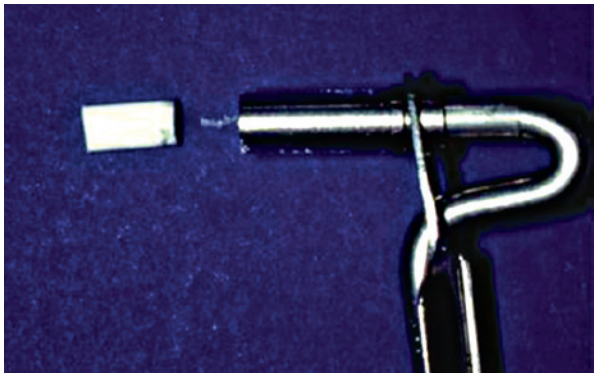

FIG. 20-15 Thick mix of calcium hydroxide.

These problems are overcome by stimulating the formation of a hard tissue barrier to allow for optimal filling of the canal and by reinforcing the weakened root against fracture, both during and after apexification.[101,154]

Technique

Disinfection of the Canal

In most cases nonvital teeth are infected,[29,138] so the first phase of treatment is to disinfect the root canal system to ensure periapical healing.[39,53] The canal length is estimated with a parallel preoperative radiograph, and after access to the canals has been prepared, a file is placed to this estimated length. When the length has been confirmed radiographically, very light filing (because of the thin dentinal walls) is performed with copious irrigation with 0.5% NaOCl.[54,147] A lower strength of NaOCl is used because of the increased danger of placing the agent through the apex in immature teeth. The increased volume of irrigant used compensates for this lower concentration of NaOCl. An irrigation needle that can passively reach close to the apical length is useful in disinfecting the canals of these immature teeth. The canal is dried with paper points and a creamy mix of calcium hydroxide (toothpaste thickness) spun into the canal with a Lentulo spiral instrument (Fig. 20-14). Additional disinfecting action of calcium hydroxide is effective after its application for at least 1 week,[141] so continuation of treatment can take place any time after 1 week. Further treatment should not be delayed more than 1 month because the calcium hydroxide could be washed out by tissue fluids through the open apex, leaving the canal susceptible to reinfection.

Hard Tissue Apical Barrier

Traditional Method

Formation of the hard tissue barrier at the apex requires a similar environment to that required for hard tissue formation in vital pulp therapy: a mild inflammatory stimulus to initiate healing and a bacteria-free environment to ensure that inflammation is not progressive.

As with vital pulp therapy, calcium hydroxide is used for this procedure.[50,83,85] Pure calcium hydroxide powder is mixed with sterile saline (or anesthetic solution) to a thick (powdery) consistency (Fig. 20-15). The calcium hydroxide is packed against the apical soft tissue with a plugger or thick point to initiate hard tissue formation. This step is followed by backfilling with calcium hydroxide to completely fill the canal. The calcium hydroxide is meticulously removed from the access cavity to the level of the root orifices, and a well-sealing, temporary filling is placed in the access cavity. A radiograph is taken; the canal should appear to have become calcified, indicating that the entire canal has been filled with the calcium hydroxide (Fig. 20-16). Because calcium hydroxide washout is evaluated by its relative radiodensity in the canal, it is prudent to use a calcium hydroxide mixture without the addition of a radiopaquer, such as barium sulfate. These additives do not wash out as readily as calcium hydroxide, so if they are present in the canal, evaluation of washout is impossible.

At 3-month intervals, a radiograph is exposed to evaluate whether a hard tissue barrier has formed and if the calcium hydroxide has washed out of the canal. This is assessed to have occurred if the canal can be seen again radiographically. If no washout is evident, the calcium hydroxide can be left intact for another 3 months. Excessive calcium hydroxide dressing changes should be avoided if at all possible because the initial toxicity of the material is thought to delay healing.[104]

When completion of a hard tissue barrier is suspected, the calcium hydroxide should be washed out of the canal with NaOCl. A file of a size that can easily reach the apex can be used to gently probe for a stop at the apex. When a hard tissue barrier is indicated radiographically and can be probed with an instrument, the canal is ready for filling.

Bioceramic Barrier

The creation of a physiologic hard tissue barrier, although quite predictable, takes anywhere from 3 to 18 months with calcium hydroxide. The disadvantages of this long time period are that the patient is required to present for treatment multiple times and the tooth may fracture during treatment before the thin, weak roots can be strengthened. In addition, one study has indicated that long-term treatment with calcium hydroxide may weaken the roots and make them even more susceptible to fracture.[19]

Bioceramic material has been used to create a hard tissue barrier quickly after disinfection of the canal (see earlier in the section Traditional Method) (Fig. 20-17). Calcium sulfate is pushed through the apex to provide a resorbable extraradicular barrier against which to pack the bioceramic material. The material is mixed and placed into the apical 3 to 4 mm of the canal in a manner similar to the placement of calcium hydroxide. A wet cotton pellet should be placed against the bioceramic material and left for at least 6 hours. After the material

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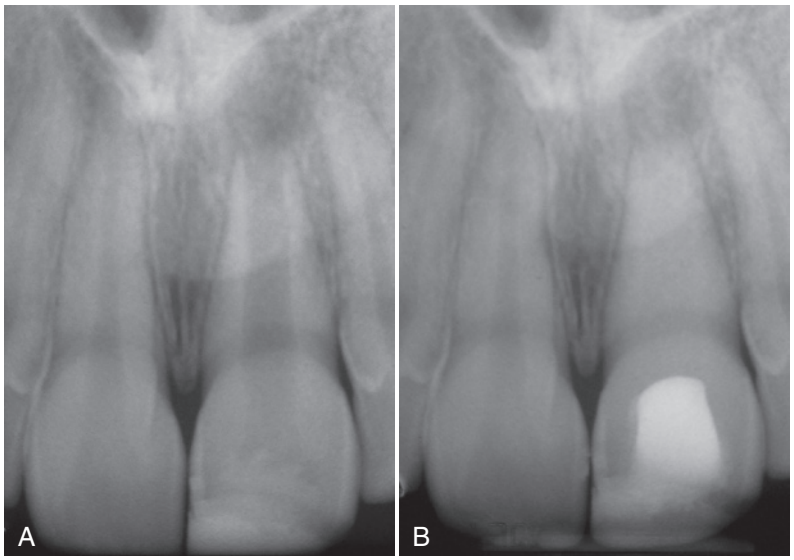

FIG. 20-16 A and B, Root canal that "disappears" after placement of a thick mix of pure calcium hydroxide and then over time washes out again. (Courtesy Dr. Cecilia Bourguignon, Paris, France.)

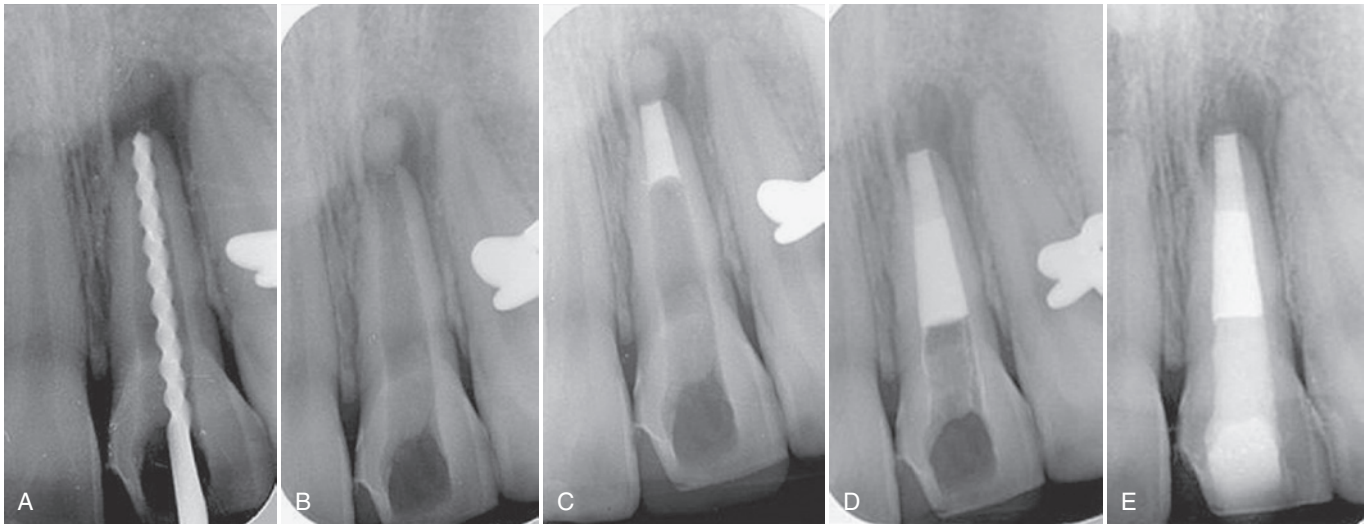

FIG. 20-17 Apexification with mineral trioxide aggregate (MTA). A, The canal is disinfected with light instrumentation, copious irrigation, and a creamy mix of calcium hydroxide for 1 month. B, Calcium sulfate is placed through the apex as a barrier against which the MTA is placed. C, A 4-mm MTA plug is placed at the apex. D, The body of the canal is filled with the Resilon obturation system. E, A bonded resin is placed below the cementoenamel junction (CEJ) to strengthen the root. (Courtesy Dr. Marga Ree. Purmerend, Netherlands)

has fully set, the entire canal is filled with a root-filling material. The cervical canal is then reinforced with composite resin to below the marginal bone level (described later) (see Fig. 20-17).

A number of case reports have been published using this apical barrier technique,[75,87,109] and it has steadily gained popularity with clinicians. Presently, no prospective long-term outcome study is available comparing its success rate with that of the traditional calcium hydroxide technique.

Filling the Root Canal

Because the apical diameter is larger than the coronal diameter of most of these canals, a softened filling technique is indicated in these teeth (see Chapter 7). Care must be taken to avoid excessive lateral force during filling, owing to the thin walls of the root. If the hard tissue barrier was produced by long-term calcium hydroxide therapy, it consists of irregularly arranged layers of coagulated soft tissue, calcified tissue, and cementum-like tissue (Fig. 20-18). Included also are islands of soft connective tissue, giving the barrier a "Swiss cheese" consistency.[33,48] Because of the irregular nature of the barrier, it is not unusual for cement or softened filling material to be pushed through it into the apical tissues during filling (Fig. 20-19). Formation of the hard tissue barrier might be some distance short of the radiographic apex because the barrier forms wherever the calcium hydroxide contacts vital tissue. In teeth with wide, open apices, vital tissue can survive and proliferate from the periodontal ligament a few millimeters into the root canal. Filling should be completed to the level of the hard tissue barrier and not forced toward the radiographic apex.

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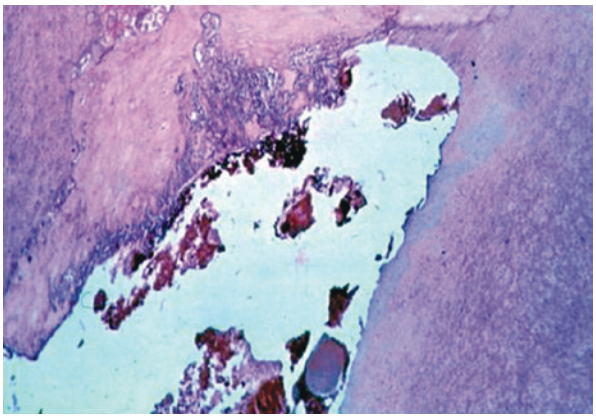

FIG. 20-18 Histologic appearance of hard tissue barrier after calcium hydroxide apexification. The barrier is composed of cementum and bone with soft tissue inclusions.

FIG. 20-19 Root filing with a soft technique after calcium hydroxide apexification. Sealer and softened filling material is expressed through the "Swiss cheese" holes in the barrier.

Reinforcement of Thin Dentinal Walls

The apexification procedure has become a predictably successful procedure (see the section Prognosis),[68] but thin dentinal walls still present a clinical problem. Should secondary injuries occur, teeth with thin dentinal walls are more susceptible to fractures that render them unrestorable.[56,158] It has been reported that approximately 30% of these teeth will fracture during or after endodontic treatment.[101] Consequently, some clinicians have questioned the advisability of the apexification procedure and have opted for more radical treatment procedures, including extraction followed by extensive restorative procedures, such as dental implants. Studies have shown that intracoronal bonded restorations can internally strengthen endodontically treated teeth and increase their resistance to fracture.[76,99] Thus, after root filling, the material should be removed to below the marginal bone level and a bonded resin filling placed (see Fig. 20-17).

Follow-Up

Routine recall evaluation should be performed to determine success in the prevention or treatment of apical periodontitis. Restorative procedures should be assessed to ensure that they in no way promote root fractures.

Prognosis

Periapical healing and the formation of a hard tissue barrier occur predictably with long-term calcium hydroxide treatment (79% to 96%).[50,101] However, long-term survival is jeopardized by the fracture potential of the thin dentinal walls of these teeth. It is expected that the newer techniques of internally strengthening the teeth described earlier will increase their long-term survivability.

Pulp Revitalization

Revitalization of a necrotic pulp has been considered possible only after avulsion of an immature permanent tooth that was reimplanted within 40 minutes (discussed later). The advantages of pulp revascularization lie in the possibility of further root development and reinforcement of dentinal walls by deposition of hard tissue, thus strengthening the root against fracture (see also Chapter 10). After reimplantation of an avulsed immature tooth, a unique set of circumstances exists that allows regeneration to take place. The young tooth has an open apex and is short, which allows new tissue to grow into the pulp space relatively quickly. The pulp is necrotic but usually neither degenerated nor infected, so it acts as a matrix into which the new tissue can grow. It has been experimentally shown that the apical part of a pulp may remain vital and after reimplantation proliferate coronally, replacing the necrotized portion of the pulp.[26,117,145] Because in most cases the crown of the tooth is intact and caries free, bacterial penetration into the pulp space through cracks[106] and defects will be a slow process. The race between new tissue growth and infection of the pulp space favors the new tissue.

Regeneration of pulp tissue in a necrotic infected tooth with apical periodontitis was thought to be impossible until about a decade ago.[114] However, if it is possible to create an environment similar to that described for the avulsed tooth, regeneration will probably occur. If the canal is effectively disinfected, a matrix into which new tissue can grow is provided, and coronal access is effectively sealed. Regeneration should occur as in an avulsed immature tooth.

Banchs and Trope[25] wrote one of the first case reports that suggest it may be possible to replicate the unique circumstances of an avulsed tooth. The case (Fig. 20-20) described the treatment of an immature second mandibular premolar that had radiographic and clinical signs of apical periodontitis, along with a sinus tract. The canal was disinfected without mechanical instrumentation but with copious irrigation with 5.25% NaOCl, followed by placement of an intracanal mixture of antibiotics consisting of equal amounts of ciprofloxacin, metronidazole, and minocycline for 3 weeks.[89]

The antibiotic mixture was rinsed out after 3 weeks, and a blood clot was produced to the level of the cementoenamel junction to provide a matrix for ingrowth of new tissue; this was followed by a deep coronal restoration to provide a bacteria-tight seal. Clinical and radiographic evidence of healing appeared as early as 22 days; the large radiolucency had disappeared within 2 months; and at the 24-month recall, it was obvious that the root walls were thick and development of the root below the restoration was similar to that of the adjacent and contralateral teeth. More recently, published cases and case series reports have further supported that it is possible

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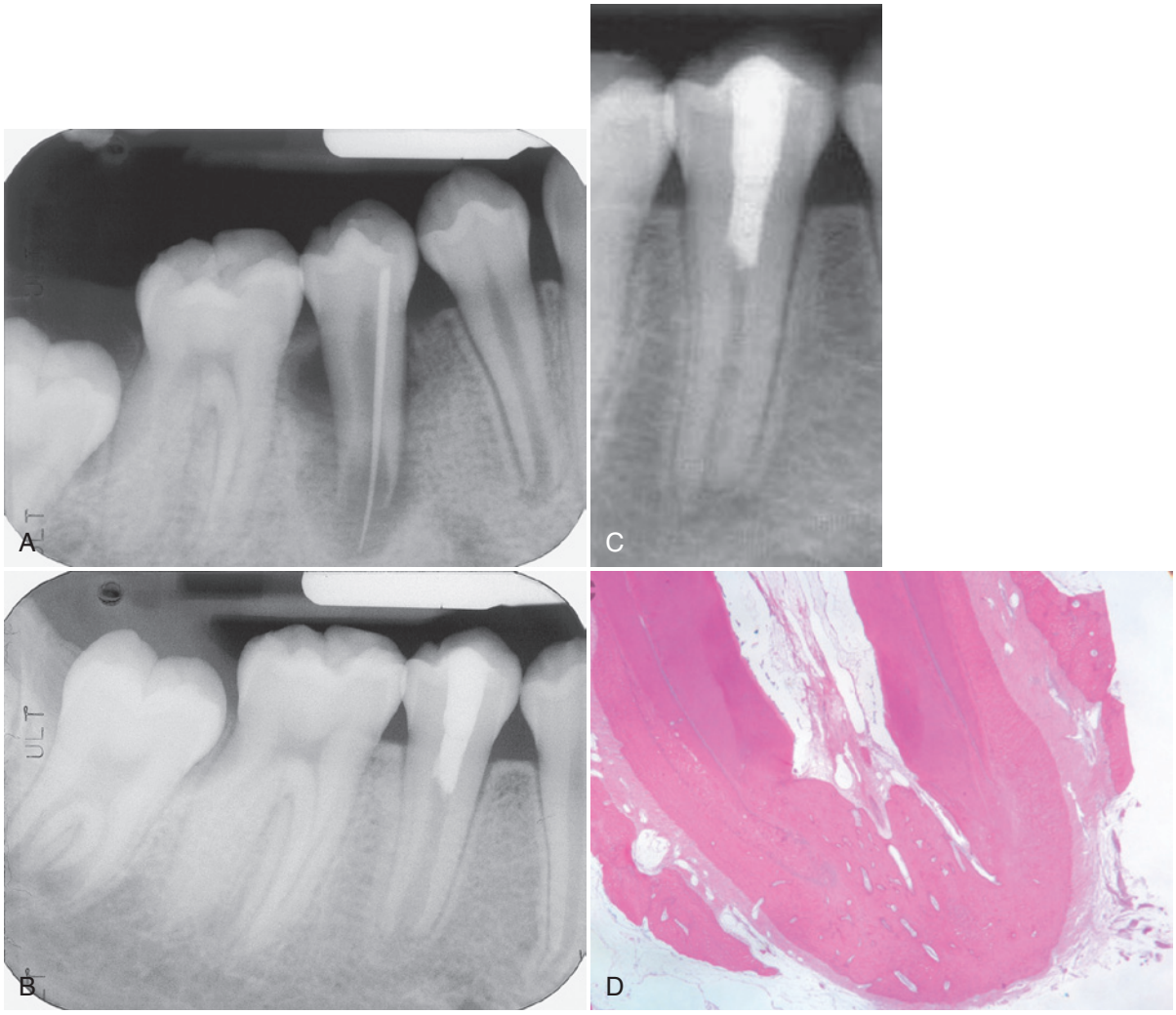

FIG. 20-20 A, Preoperative radiograph showing an immature root with periradicular periodontitis and a traced sinus tract to the apex of the root. B, Radiographic checkup 7 months after treatment that included application of a mixture of ciprofloxacin, metronidazole, and minocycline. C, At 24-month checkup, showing continued root development both in length and width of the root. D, Histologic slide from Thibodeau et al.[155] showing thickened root walls, apical development, and healing of apical periodontitis in a dog tooth where a periapical lesion had been created in an immature tooth.

to have ingrowths of tissue followed by new apposition of dentin and continuation of root formation.[96,155] Studies have also confirmed the potent antibacterial properties of the triantibiotic paste used in these cases.

There has been an explosion of studies in the last few years aimed at making the procedure more predictable both in healing of apical periodontitis and in promoting pulp and not nonspecific connective tissue (see Chapter 10).

The minocycline in the triantibiotic paste causes unacceptable discoloration of the teeth; this has led to many studies on eliminating or replacing this antibiotic in the paste, or eliminating the tripaste. Studies are underway to find a synthetic matrix that can act as a more predictable scaffold for new ingrowth of tissue than the blood clot that was used in these examples.[155,173]

Studies are also underway on stem cells and growth factors aimed at a more predictable production of odontoblasts and thus pulp cells.

The procedure described here can be attempted in most cases, and if after 3 months there are no signs of regeneration, the more traditional treatment methods can be initiated.

Mature Tooth

Conventional endodontic therapy is recommended for mature teeth with closed apex.

CROWN-ROOT FRACTURE

Crown-root fracture is a periodontal rather than an endodontic challenge. The tooth must be treated periodontally to enable a well-sealed coronal restoration. This could be accomplished by simple gingivectomy if the extent of the root component of the fracture is large. Alternatively, the tooth could be orthodontically or surgically extruded such that the exposed surface of the root fracture is treatable. Once the feasibility of the coronal

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restoration is assured, the particular crown fracture is treated as previously described.

ROOT FRACTURE

Root fracture implies fracture of the cementum, dentin, and pulp. These injuries are relatively infrequent, occurring in less than 3% of all dental injuries.[177] Immature teeth with vital pulps rarely sustain horizontal root fractures.[7] When a root fractures horizontally, the coronal segment is displaced to a varying degree, but generally the apical segment is not displaced. Because the apical pulpal circulation is not disrupted, pulp necrosis in the apical segment is extremely rare. Permanent pulpal necrosis of the coronal segment, requiring endodontic treatment, occurs in about 25% of cases.[8,9,93]

Diagnosis and Clinical Presentation

The clinical presentation of a root fracture is similar to that of luxation injuries. The extent of displacement of the coronal segment is usually indicative of the location of the fracture and can vary from none, simulating a concussion injury (apical fracture), to severe, simulating an extrusive luxation (cervical fracture).

Radiographic examination for root fractures is extremely important. Because root fractures are usually oblique (facial to palatal) (Fig. 20-21), one periapical radiograph may easily miss it. It is imperative to take at least three angled radiographs (45°, 90°, and 110°) so that at least at one angulation, the x-ray beam passes directly through the fracture line to make it visible on the radiograph (Fig. 20-22).

Treatment

Emergency treatment involves repositioning the segments into close proximity as much as possible. In the case of severe displacement of the coronal segment, its apical extension is frequently lodged in (if not perforating through) the cortical bone facial to the tooth. Forcing the crown facially is not possible, and the two segments will not be properly aligned. The only way to accomplish reapproximation of the two segments is to release the coronal segment from the bone by gently pulling it slightly downward with finger pressure or extraction forceps and, once it is loose, rotating it back to its original position (Fig. 20-23). The traditionally recommended[124] splinting protocol has been changed from 2 to 4 months with rigid splinting to a semirigid splint to adjacent teeth for 2 to 4 weeks.[18] If a long time has elapsed between the injury and treatment, repositioning of the segments close to their original position probably will not be possible; this compromises the long-term prognosis for the tooth.

FIG. 20-21 Extracted root after a root fracture. Note the oblique angle of the fracture.

Healing Patterns

Investigators[21] have described four types of responses to root fractures.

1. *Healing with calcified tissue.* Radiographically, the fracture line is discernible, but the fragments are in close contact (Fig. 20-24, *A*).
2. *Healing with interproximal connective tissue.* Radiographically, the fragments appear separated by a narrow radiolucent line, and the fractured edges appear rounded (Fig. 20-24, *B*).
3. *Healing with interproximal bone and connective tissue.* Radiographically, the fragments are separated by a distinct bony ridge (Fig. 20-24, *C*).
4. *Interproximal inflammatory tissue without healing.* Radiographically, a widening of the fracture line and/or a developing radiolucency corresponding to the fracture line becomes apparent (Fig. 20-24, *D*).

The first three healing patterns are considered successful. The teeth are usually asymptomatic and respond positively to sensitivity testing. Coronal yellowing is possible because calcific metamorphosis of the coronal segment is not unusual (see Fig. 20-29).[94,177]

The fourth type of root fracture response is typical when the coronal segment loses its vitality. The infective products in the coronal pulp cause an inflammatory response and typical radiolucencies at the fracture line (see Fig. 20-24, *D*).[21]

Treatment of Complications

Coronal Root Fractures

Historically it had been thought that fractures in the cervical segment had a poor prognosis, and extraction of the coronal segment was recommended. Research does not support this treatment; in fact, if these coronal segments are adequately splinted, chances of healing do not differ from those for midroot or apical fractures (Fig. 20-25).[177] However, if the fracture occurs at the level of or coronal to the crest of the alveolar bone, the prognosis is extremely poor.

If reapproximation of the fractured segments is not possible, extraction of the coronal segment is indicated. The level of fracture and length of the remaining root are evaluated for restorability. If the apical root segment is long enough, gentle orthodontic eruption of this segment can be carried out to enable fabrication of a restoration.

Midroot and Apical Root Fractures

Permanent pulpal necrosis occurs in 25% of root fractures.[48,93] Initially it is likely that in many cases the pulp in the coronal segment will become necrotic after the injury; however, because of a very large apical opening in the coronal segment, revascularization is possible if the segments are well reapproximated.

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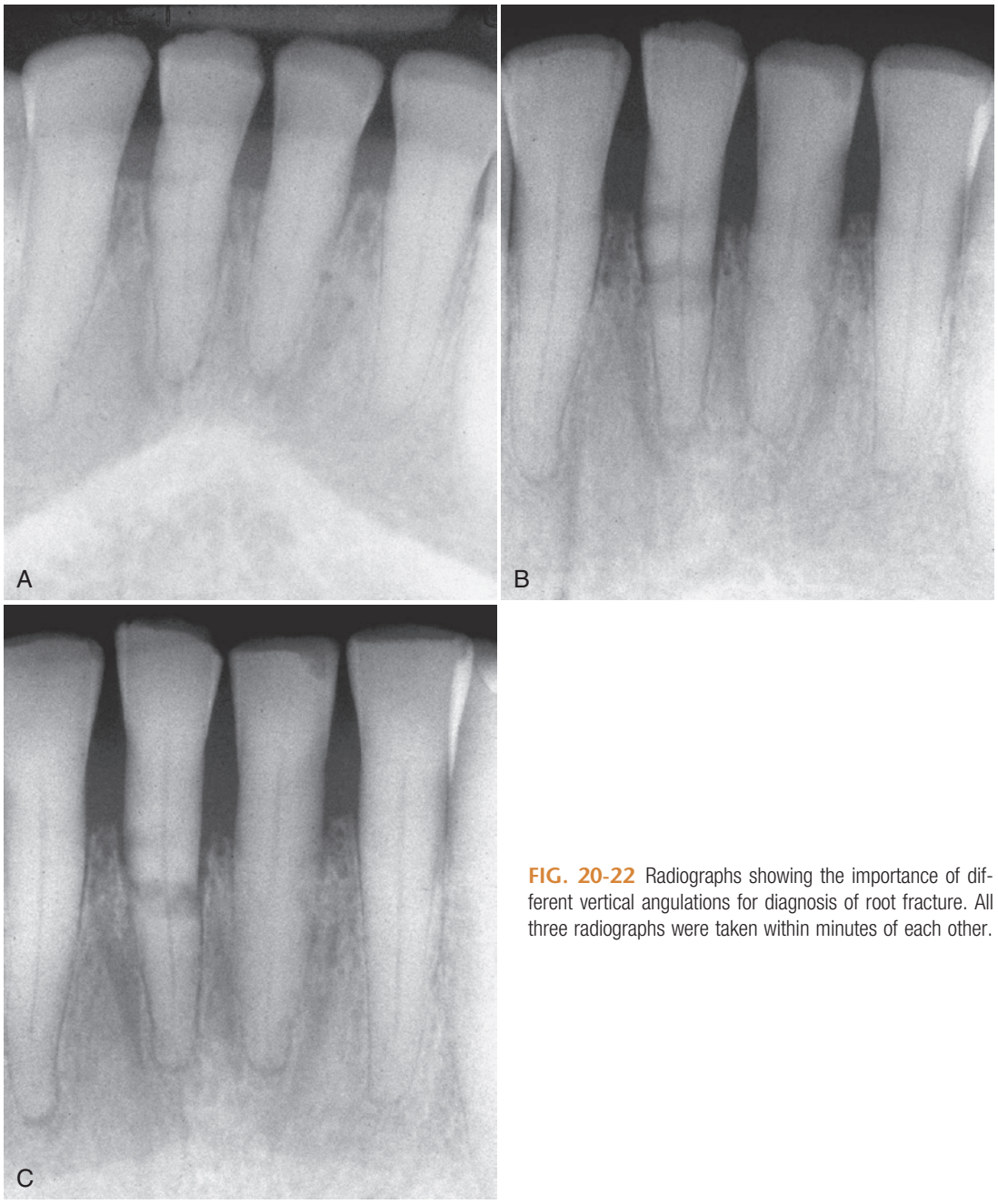

FIG. 20-22 Radiographs showing the importance of different vertical angulations for diagnosis of root fracture. All three radiographs were taken within minutes of each other.

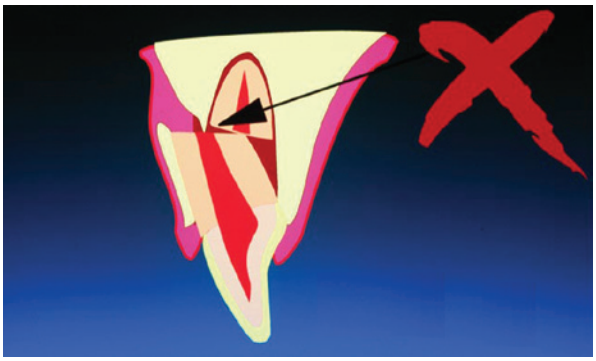

FIG. 20-23 Figure of facial root fracture manipulated back into place.

In most cases, permanent necrosis occurs in the coronal segment, with only the apical segment remaining vital. Therefore, endodontic treatment is indicated in the coronal root segment unless periapical pathology is seen in the apical segment. In most cases the pulpal lumen is wide at the apical extent of the coronal segment, and long-term calcium hydroxide treatment or an MTA apical plug is indicated (see the section Apexification). The coronal segment is filled after a hard tissue barrier has formed apically in the coronal segment and periradicular healing has taken place.

In rare cases in which both the coronal and apical pulp are necrotic, treatment is more complicated. Endodontic treatment through the fracture is extremely difficult. Endodontic manipulations, medicaments, and filling materials all have a

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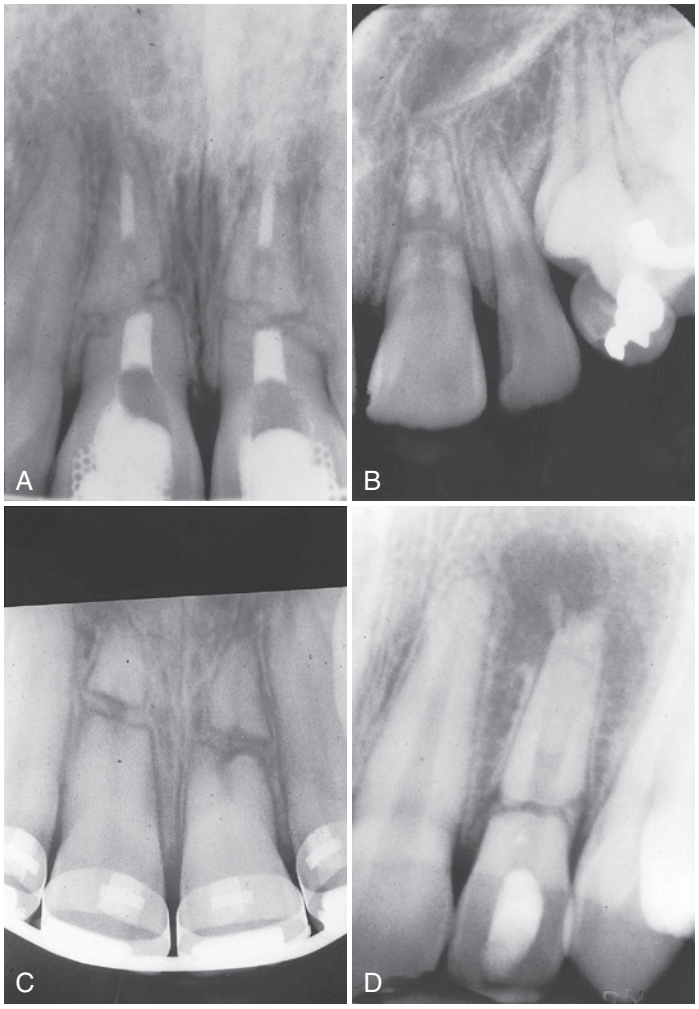

FIG. 20-24 Healing patterns after horizontal root fractures. **A,** Healing with calcified tissue. **B,** Healing with interproximal connective tissue. **C,** Healing with bone and connective tissue. **D,** Interproximal connective tissue without healing.

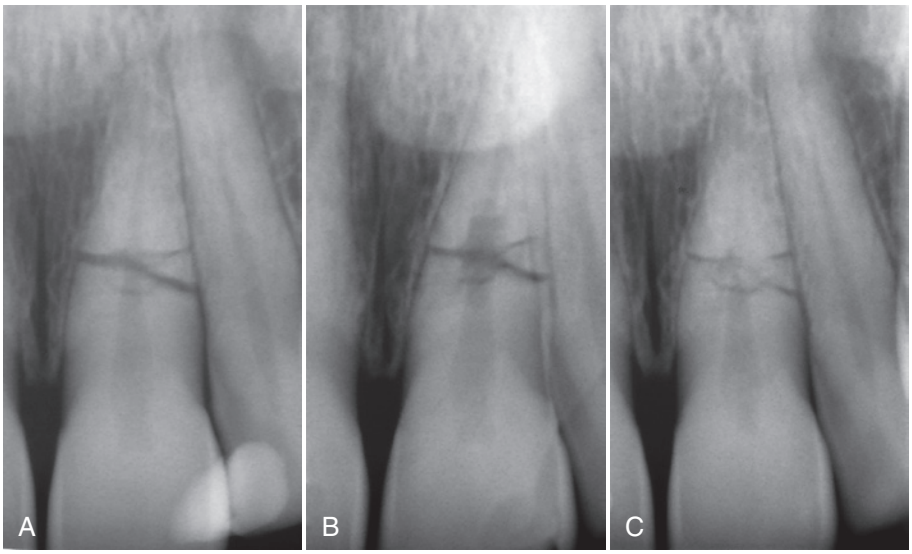

FIG. 20-25 Internal root resorption in the root fracture area; tooth was diagnosed with compound midroot fracture. **A,** Coronal portion was repositioned and then splinted to the lateral incisor with a composite splint. **B,** At 6-month recall, the pulp responded normally to cold, but an internal root-resorptive defect was noted on radiograph. No treatment was indicated, because the pulp did respond normally to vitality tests. **C,** At 42-month recall, the tooth was still asymptomatic, and the pulp still responded to vitality tests. The resorptive defect had healed, and signs of dystrophic calcifications were evident in the apical and coronal segments.

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detrimental effect on healing of the fracture site. If healing of the fracture has been completed and is followed by necrosis of the apical segment, the prognosis is much improved.

In more apical root fractures, necrotic apical segments can be surgically removed. This is a viable treatment if the remaining coronal root segment is long enough to provide adequate periodontal support. Removal of the apical segment in midroot fractures leaves the coronal segment with a compromised attachment; endodontic implants have been used to provide additional support to the tooth.

Follow-Up

After the splinting period is completed, follow-up is as for all dental traumatic injuries: at 3, 6, and 12 months and yearly thereafter.

Prognosis

The following factors influence repair:

1. The degree of dislocation and mobility of the coronal fragment are extremely important in determining outcome.[7,17,94,148,177] Increased dislocation and coronal fragment mobility result in a poorer prognosis.
2. Immature teeth are seldom involved in root fractures, but in the unlikely event they are, the prognosis is good.[17,92]
3. The quality of treatment is vital to successful repair. The prognosis improves with quick treatment, close reduction of the root segments, and semirigid splinting for 2 to 4 weeks.[18]

Complications include pulp necrosis and root canal obliteration. Pulp necrosis can be treated successfully[48,93] by treating the coronal segment with calcium hydroxide to stimulate hard tissue barrier formation. Root canal obliteration is common if the root segment (coronal or apical) remains vital.

LUXATION INJURIES

Definitions

The types of luxation injury can be defined as follows:

1. *Concussion* implies no displacement, normal mobility, and sensitivity to percussion.
2. *Subluxation* implies sensitivity to percussion, increased mobility, and no displacement.
3. *Lateral luxation* implies displacement labially, lingually, distally, or incisally.
4. *Extrusive luxation* implies displacement in a coronal direction.
5. *Intrusive luxation* implies displacement in an apical direction into the alveolus.

Definitions 1 through 5 describe injuries of increasing magnitude in terms of intensity of the injury and subsequent sequelae.

Incidence

Luxation injuries as a group are the most common of all dental injuries, with a reported incidence ranging from 30% to 44%.[55]

Treatment

Teeth that are concussed or subluxated do not need any immediate treatment. Responses to vitality tests should be investigated and noted. Even after mild injury, such as subluxation, the pulp might be unresponsive to vitality tests for several weeks if not months.[144] When the pulp is unresponsive initially after the trauma, patients should be recalled on a regular basis and monitored for any additional signs of infection of the root canal (discussed later).

Teeth with lateral and extrusive luxation should be repositioned as soon as possible. In lateral luxation, the apex might be perforating the facial bone plate, and the tooth must be slightly and gently pulled down to loosen the hold before it is repositioned in its original position. Current IADT guidelines call for 2 weeks of physiologic splinting in cases of extrusion luxation and 4 weeks for lateral luxation. A decision on root canal therapy follows the guidelines for avulsion (discussed later). If the tooth has a fully formed apex and was diagnosed to have moved into (if not through) the cortical plate (apical translocation), there is a good likelihood of the pulp being devitalized; therefore, endodontic treatment should be initiated as early as 2 weeks after the injury. If the apex is still not fully formed, waiting for signs of revascularization is strongly recommended.

Permanent teeth that are intruded are not likely to spontaneously re-erupt, especially if the apex is fully formed.[100] Alternative treatment, such as orthodontic extrusion or immediate surgical repositioning, should be considered. If orthodontic extrusion is planned for an intruded tooth, it should be initiated as soon as possible and should not be delayed longer than 2 to 3 weeks after the trauma. Only a few studies have evaluated the true efficacy of this approach. One study[168] using a dog model indicated that severely intruded teeth showed signs of ankylosis as early as 11 to 13 days after the trauma, despite initiation of orthodontic movement 5 to 7 days after the injury.

For the surgical approach, one study[47] (using a dog model) concluded that "a careful immediate surgical repositioning of a severely intruded permanent tooth with complete root formation has many advantages with few disadvantages." Another investigation,[58] a retrospective study of 58 intruded human teeth, indicated that surgical repositioning resulted in a predictable outcome, with only five of the teeth lost over the observation period. However, it was also observed that less surgical manipulation positively influenced the healing. If an intruded tooth is immediately reimplanted, it should be splinted for at least 4 weeks, but in most cases the splint needs to be left on the tooth longer.

Biologic Consequences

Luxation injuries result in damage to the attachment apparatus (periodontal ligament and cemental layer), the severity of which depends on the type of injury sustained (concussion least, intrusion most). The apical neurovascular supply to the pulp is also affected to varying degrees, resulting in an altered or nonvital tooth.

Healing can be favorable or unfavorable. Favorable healing after a luxation injury occurs if the initial physical damage to the root surface and the resultant inflammatory response to the damaged external root surface are again covered with cementum. An unfavorable response occurs when there is direct attachment of bone to the root, with the root ultimately being replaced by bone.

There are two resorption responses in which the pulp plays an essential role:

1. In *external* inflammatory root resorption, the *necrotic infected pulp* provides the stimulus for periodontal

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inflammation in the ligament space. If the cementum has been damaged, the inflammatory stimulators in the pulp space are able to diffuse through the dentinal tubules and stimulate an inflammatory response over large areas of the periodontal ligament. Because of the lack of cemental protection, the periodontal inflammation includes root resorption in addition to the expected bone resorption.

2. With *internal* inflammatory root resorption, the inflamed pulp is the tissue involved in resorbing the root structure. The pathogenesis of internal root resorption is not completely understood. Here it is thought that coronal necrotic infected pulp provides a stimulus for a pulpal inflammation in the more apical parts of the remaining vital pulp.[7] If the internal root surface has lost its precemental protection during an injury, internal root resorption occurs in the area adjacent to the inflamed pulp. Thus, both the necrotic infected pulp and the inflamed pulp contribute to this type of root resorption.

External Root Resorption

Caused by an Injury (Alone) to the External Root Surface

If an injury harms the attachment, the by-products of this mechanical damage stimulate an inflammatory response. The healing response depends on the extent of the initial damage.

Localized Injury: Healing with Cementum

When the injury is localized (e.g., after concussion or subluxation injury), mechanical damage to the cementum occurs, resulting in a local inflammatory response and a localized area of root resorption. If no further inflammatory stimulus is present, periodontal healing and root surface repair occur within 14 days (Fig. 20-26).[78] The resorption is localized to the area of mechanical damage, and treatment is not required because it is free of symptoms and not even visualized radiographically in most cases. In a minority of cases, however, small radiolucencies can be seen on the root surface if the radiograph is taken at a specific angle. It is important to avoid misinterpreting these cases as progressive. The pulp is not involved. If the pulp responds to sensitivity tests, this is a clue that no treatment should be performed. A wait-and-see attitude can allow spontaneous healing to take place. It is important to realize that in the initial stages, radiolucency could be followed by spontaneous repair, and endodontic treatment should *not* be initiated.

Diffuse Injury: Healing by Osseous Replacement

When the traumatic injury is severe (e.g., intrusive luxation or avulsion with extended dry time), involving diffuse damage on more than 20% of the root surface, an abnormal attachment can occur after healing takes place.[105] The initial reaction, as always, is inflammation in response to the severe mechanical damage to the root surface. After the initial inflammatory response, a diffuse area of root surface devoid of cementum results. Cells in the vicinity of the denuded root now compete to repopulate it. Often cells that are precursors of bone, rather than the slower-moving periodontal ligament cells, move across from the socket wall and populate the damaged root. Bone comes into contact with the root without an intermediate attachment apparatus. This phenomenon is termed *dentoalveolar ankylosis*.[105] However, the ankylosis and osseous replacement that follows cannot be reversed and can be considered a physiologic process because bone resorbs and reforms throughout life. Thus root is resorbed by osteoclasts, but in the reforming stage, bone is laid down instead of dentin, slowly replacing the root with bone. This process is termed *osseous replacement* (Fig. 20-27).[164] The initial inflammation to remove the mechanical debris of the traumatic injury is a pathologic response that in the authors' opinion may be reversed. In these traumatic cases, however, the resorptive phase includes the root (Fig. 20-28).

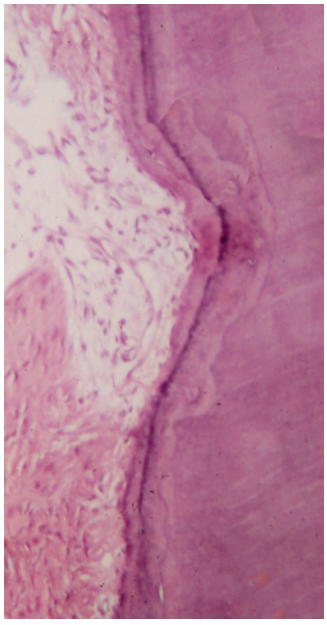

FIG. 20-26 Histologic section showing previous root resorptive defect healed with new cementum and periodontal ligament.

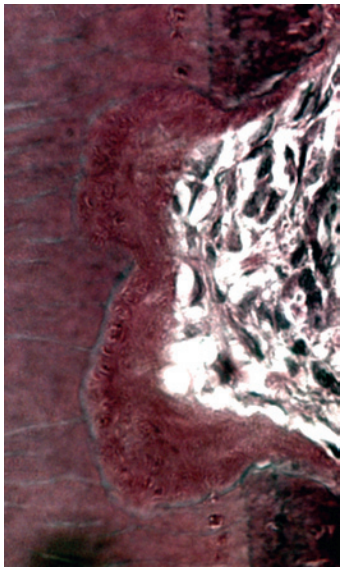

FIG. 20-27 Histologic appearance of osseous replacement. There is direct contact between bone and root structure. Resorptive defects are seen in the bone and root tissue, which is the normal physiologic process of bone turnover. The resorptive defects are filled by new bone and additional areas resorbed. In this way the entire root is replaced by bone at a rate dependent on the metabolic rate of the patient.

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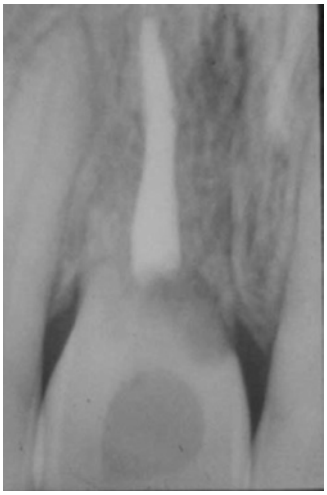

FIG. 20-28 Radiographic appearance of osseous replacement. The root acquires the radiographic appearance of the surrounding bone (without a lamina dura). Note that radiolucencies typical of active inflammation are not present.

Treatment

Treatment strategies for limiting the effect of the traumatic injury to the periodontal structures are outside the scope of this chapter. However, in general they involve minimizing the initial inflammatory response to the injury. The initial inflammation is destructive in nature and increases the surface area of root to be covered in the healing phase.[20] The smaller the surface area to be covered by new cementum, the higher the chances of favorable repair.

A study has indicated that if Ledermix, a drug combining corticosteroid and tetracycline, is placed in the root canal immediately after a severe trauma in which osseous replacement is expected, favorable healing occurs at a very high rate.[38] In a more recent study, it was shown that triamcinolone (the corticosteroid portion of the Ledermix paste) was as effective as Ledermix at inhibiting external root resorption.[41] Both of these studies were done on young dogs and need to be replicated in human studies.

Caused by an Injury to the External Root Surface and Inflammatory Stimulus in the Root Canal

Recognized inflammatory stimuli that cause root resorption are pressure, pulp space infection, and sulcular infection. This chapter focuses on pulp space infection.

Consequences of Apical Neurovascular Supply Damage

Pulp Canal Obliteration (Calcification)

Pulp canal obliteration is common after luxation injuries (Fig. 20-29). The frequency of pulp canal obliteration appears inversely proportional to pulp necrosis. The exact mechanism of pulp canal obliteration is unknown. It has been theorized that the sympathetic/parasympathetic control of blood flow to the odontoblasts is altered, resulting in uncontrolled reparative dentin.[7,15] Another theory is that hemorrhage and blood clot formation in the pulp after injury form a nidus for subsequent calcification if the pulp remains vital.[7,15] One study[11] found that pulp canal obliteration could usually be diagnosed within the first year after injury and was more frequent in teeth with open apices (>0.7 mm radiographically), those with extrusive and lateral luxation injuries, and those that had been rigidly splinted.[11]

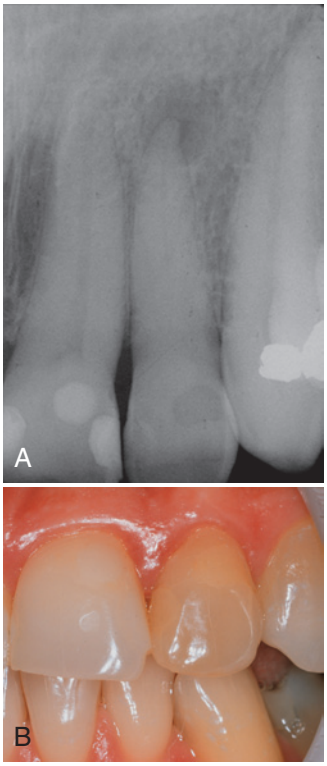

FIG. 20-29 A, Pulp canal calcification after a luxation injury. Radiographic appearance of a calcified maxillary lateral incisor; note that despite calcification, it has now become necrotic and infected, evident by the periapical lesion. B, The typical yellow appearance of the tooth caused by thickened dentin in the pulp chamber.

Pulp Necrosis

The factors most important for the development of pulp necrosis are the type of injury (concussion least, intrusion most) and the stage of root development (mature apex more than an immature apex).[10] Pulp necrosis is most likely to lead to infection of the root canal system, with problematic consequences.

Pulp Space Infection

Pulp space infection in conjunction with damage to the external root surface results in periradicular root and bone resorption and continues in its active state as long as the pulpal stimulus (infection) remains. When the root loses its cemental protection, lateral periodontitis with root resorption can result (Fig. 20-30).

To have pulp space infection, the pulp must first become necrotic. Necrosis occurs after a fairly serious injury in which displacement of the tooth results in severing of the apical blood vessels. In mature teeth, pulp regeneration cannot occur, and usually by 3 weeks, the necrotic pulp becomes infected. (For details of the typical bacterial contents of a traumatized necrotic pulp, the reader is referred to Chapter 14 or to Bergenholtz.[29]) Because a serious injury is required for pulp necrosis, areas of cemental covering of the root usually are also affected, resulting in loss of its protective (insulating) quality. Now bacterial toxins can pass through the dentinal tubules and

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FIG. 20-30 Inflammatory root resorption caused by a pulp space infection. Note the radiolucencies in the root and surrounding bone. (Courtesy Dr. Fred Barnett.)

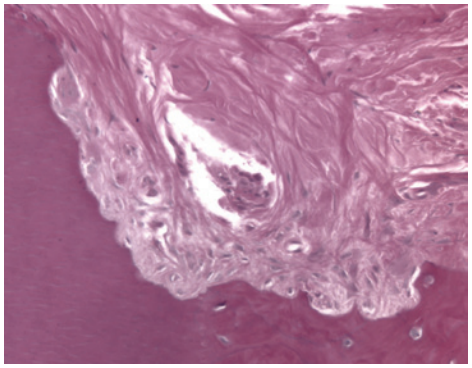

FIG. 20-31 Histologic appearance of multinucleated osteoclasts (dentinoclasts) resorbing the dentin of the root.

stimulate an inflammatory response in the periodontal ligament. The result is resorption of the root and bone. The periodontal infiltrate consists of granulation tissue with lymphocytes, plasma cells, and polymorphonuclear leukocytes. Multinucleated giant cells resorb the denuded root surface, and this continues until the stimulus (pulp space bacteria) is removed (Fig. 20-31).[160] Radiographically the resorption is observed as progressive radiolucent areas of the root and adjacent bone (see Fig. 20-30).

Treatment

Assessing attachment damage caused by the traumatic injury and minimizing the subsequent inflammation should be the focus of the emergency visit. The clinician's attention to pulp space infection should ideally be 7 to 10 days after the injury.[166,167] Root canal disinfection removes the stimulus to the periradicular inflammation, thus the resorption will stop.[78,166,167] In most cases a new attachment will form, but if a large area of root is affected, osseous replacement can result by the mechanism already described. Again, treatment principles include prevention of pulp space infection or elimination of the bacteria if they are present in the pulp space.

1. Prevention of pulp space infection
   a. *Reestablish the vitality of the pulp*. If the pulp stays vital, the canal will be free of bacteria, and external inflammatory root resorption will not occur. In severe injuries in which vitality has been lost, it is possible under some circumstances to promote revascularization of the pulp. Revascularization is possible in young teeth with incompletely formed apices if the teeth are replaced in their original position within 60 minutes of the injury (Fig. 20-32).[51] If the tooth has been avulsed, soaking it in doxycycline for 5 minutes or covering the root with minocycline powder before replantation has been shown to double or triple the revascularization rate.[51,127] Even under the best conditions, however, revascularization fails to occur on many occasions, and a diagnostic dilemma results. If the pulp revascularizes, external root resorption will not occur, and the root will continue to develop and strengthen. If the pulp becomes necrotic and infected, the subsequent external inflammatory root resorption that develops could result in loss of the tooth in a very short time. At present, the diagnostic tools available cannot detect a vital pulp in this situation before approximately 6 months after successful revascularization. This period of time is obviously unacceptable because by that time the teeth that have not revascularized could be lost to the resorption process. Recently the laser Doppler flowmeter or the pulse oximeter have been shown to have diagnostic potential for the detection of revascularization in immature teeth (Fig. 20-33). These devices appear to detect the presence of vital tissue in the pulp space by 4 weeks after the traumatic injury.[176]

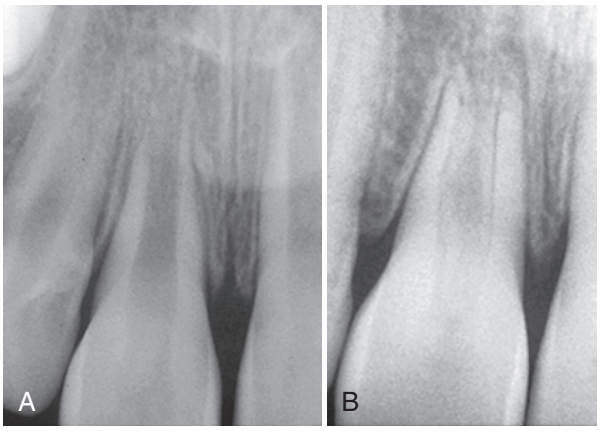

FIG. 20-32 Revascularization of immature root. A tooth with an open apex was replanted soon after the avulsion. The checkup radiograph 12 years later confirms that regrowth has taken place into the pulp chamber. It appears that a bone plug has grown in, and new periodontal ligament has formed, with a lamina dura within the pulp canal. (Courtesy Dr. Cecilia Bourguignon, Paris, France.)

   b. *Prevent root canal infection by initiating root canal treatment at 7 to 10 days*. In teeth with closed apices, revascularization cannot occur. These teeth should be endodontically treated within 7 to 10 days of the injury, before the ischemically necrosed pulp becomes infected.[166,167] Theoretically, treating teeth within this time period can be considered equivalent to treating a tooth with a vital pulp, and the endodontic treatment could be completed in one visit if possible. However, efficient treatment is extremely difficult so soon after a serious traumatic injury, and in the authors' opinion, it is beneficial to start the endodontic treatment with chemomechanical preparation, after which an intracanal

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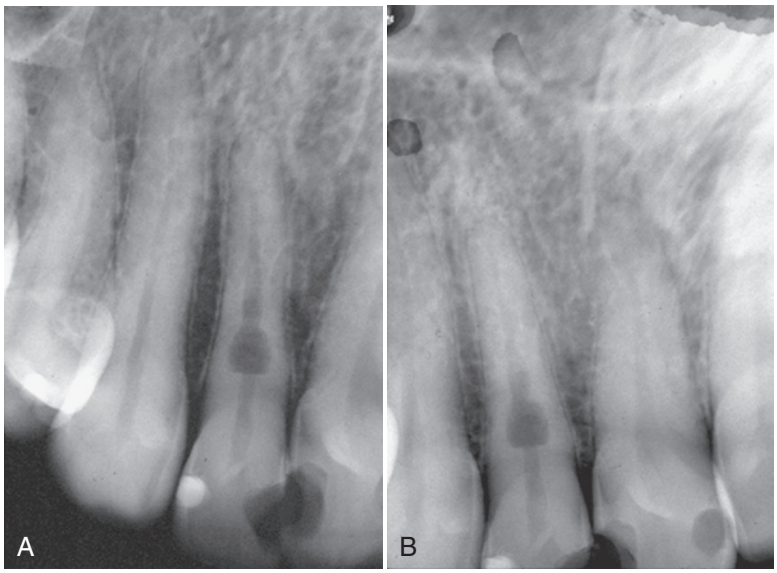

FIG. 20-33 Angled radiographs to show internal resorption. Radiographs from two different horizontal projections depict (A) the lesion within the confines of the root canal on both views and (B) the adjacent bone intact on both views.

dressing with a creamy mix of calcium hydroxide is placed (see Fig. 20-14).[166] Then, the clinician can fill the canal at his or her convenience after periodontal healing of the injury is complete, approximately 1 month after the instrumentation visit. There appears to be no necessity for long-term calcium hydroxide treatment in cases in which the endodontic treatment is started within 10 days of the injury.[166]

2. *Eliminate pulp space infection*. When root canal treatment is initiated later than 10 days after the accident or if active external inflammatory resorption is observed, the preferred antibacterial protocol consists of microbial control followed by long-term dressing with densely packed calcium hydroxide.[167] Calcium hydroxide can effect an alkaline pH in the surrounding dentinal tubules (Fig. 20-34), kill bacteria, and neutralize endotoxin, a potent inflammatory stimulator.

The first visit consists of the microbial control phase, with cleaning and shaping of the canal and the placement of a creamy mix of calcium hydroxide using a Lentulo spiral. The patient is seen in approximately 1 month, at which time the canal is filled with a dense mix of calcium hydroxide. Once filled, the canal should appear radiographically to be calcified because the radiodensity of calcium hydroxide in the canal is usually similar to that of the surrounding dentin (see Fig. 20-16). A radiograph is then exposed at 3-month intervals. At each visit the tooth is tested for symptoms of periodontitis. In addition to stopping the resorptive process, calcium hydroxide washout is assessed. Because the root surface is so radiodense as to make assessment of healing difficult, the adjacent bone healing is assessed. If adjacent bone has healed, it is assumed that the resorptive process has stopped in the root as well; then the canal can be obturated with the permanent root filling material (Fig. 20-35).

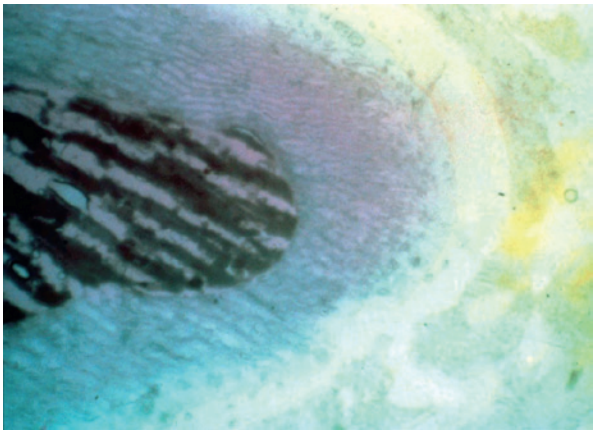

FIG. 20-34 High pH of calcium hydroxide. The root was filled with calcium hydroxide and then cut in cross section. A pH indication shows the high pH in the canal and surrounding root, whereas the surrounding tissue is a neutral pH.

Internal Root Resorption

Internal root resorption is rare in permanent teeth. Internal resorption is characterized by an oval-shaped enlargement of the root canal space.[16] External resorption, which is much more common, is often misdiagnosed as internal resorption.

Etiology

Internal root resorption is characterized by resorption of the internal aspect of the root by multinucleated giant cells adjacent to the granulation tissue in the pulp (Fig. 20-36). Chronic inflammatory tissue is common in the pulp, but only rarely does it result in resorption. There are different theories on the origin of the pulpal granulation tissue involved in internal resorption. The most logical explanation is that it is inflamed pulp tissue caused by an infected coronal pulp space. Communication between the coronal necrotic tissue and the vital pulp is through appropriately oriented dentinal tubules (see Fig. 20-36).[170] One investigator[170] reports that resorption of the

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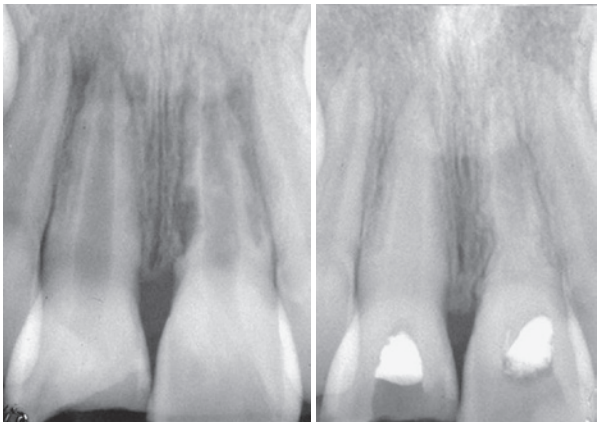

FIG. 20-35 Healing of external inflammatory root resorption after calcium hydroxide treatment. The radiolucencies seen before treatment have disappeared with the reestablishment of the lamina dura. (Courtesy Dr. Fred Barnett.)

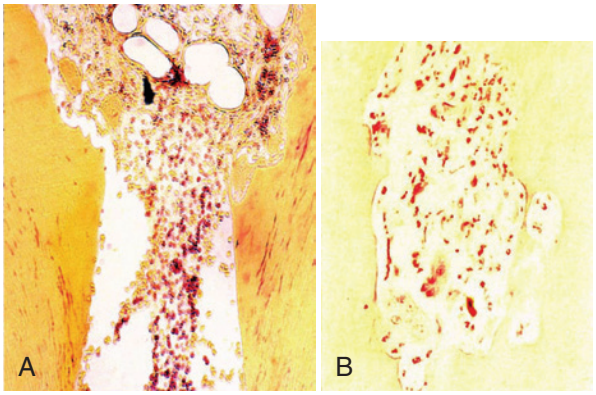

FIG. 20-36 Histologic appearance of internal root resorption. A, Section stained with Brown and Brenn. Bacteria are seen (in the dentinal tubules) communicating between the necrotic coronal segment and the apical granulomatous tissue and resorbing cells. B, An area of active internal root resorption. (Courtesy Dr. Leif Tronstad, Oslo, Norway.)

dentin is frequently associated with deposition of hard tissue resembling bone or cementum and not dentin. He postulates that the resorbing tissue is not of pulpal origin but is "metaplastic" tissue derived from the pulpal invasion of macrophage-like cells. Others[149] concluded that the pulp tissue was replaced by periodontium-like connective tissue when internal resorption was present. In addition to the requirement of the presence of granulation tissue, root resorption takes place only if the odontoblastic layer and predentin are lost or altered.[160]

Reasons for the loss of predentin adjacent to the granulation tissue are not obvious. Trauma frequently has been suggested as a cause.[135,171] Another reason for the loss of predentin might be extreme heat produced when cutting on dentin without an adequate water spray. The heat presumably would destroy the predentin layer, and if later the coronal aspect of the pulp becomes infected, the bacterial products could initiate the typical inflammation in conjunction with resorbing giant cells in the vital pulp adjacent to the denuded root surface. Internal root resorption has been produced experimentally by the application of diathermy.[170]

FIG. 20-37 A maxillary incisor with internal root resorption. Uniform enlargement of the pulp space is apparent. Outline of the canal cannot be seen in the resorptive defect.

Clinical Manifestations

Internal root resorption is usually asymptomatic and is first recognized clinically through routine radiographs. For internal resorption to be active, at least part of the pulp must be vital. The coronal portion of the pulp is often necrotic, whereas the apical pulp that includes the internal resorptive defect can remain vital. Therefore, a negative sensitivity test result does not rule out active internal resorption. It is also possible that the pulp becomes nonvital after a period of active resorption, giving a negative sensitivity test, radiographic signs of internal resorption, and radiographic signs of apical inflammation. Traditionally, the pink tooth has been thought pathognomonic of internal root resorption. The pink color is due to the granulation tissue in the coronal dentin undermining the crown enamel. The pink tooth can also be a feature of subepithelial external inflammatory root resorption, which must be ruled out before a diagnosis of internal root resorption is made.

Radiographic Appearance

The usual radiographic presentation of internal root resorption is a fairly uniform radiolucent enlargement of the pulp canal (Fig. 20-37). Because the resorption is initiated in the root canal, the resorptive defect includes some part of the root canal space, so the original outline of the root canal is distorted.

Histologic Appearance

Like that of other inflammatory resorptive defects, the histologic picture of internal resorption is granulation tissue with multinucleated giant cells (see Fig. 20-35). An area of necrotic pulp is found coronal to the granulation tissue. Dentinal tubules containing microorganisms and communicating between the necrotic zone and the granulation tissue can sometimes be seen (see Fig. 20-36).[140,160,167,170] Unlike external root resorption, the adjacent bone is not affected with internal root resorption.

Treatment

Treatment of internal root resorption is conceptually very easy. Because the resorptive defect is the result of the inflamed pulp and the blood supply to the tissue is through the apical

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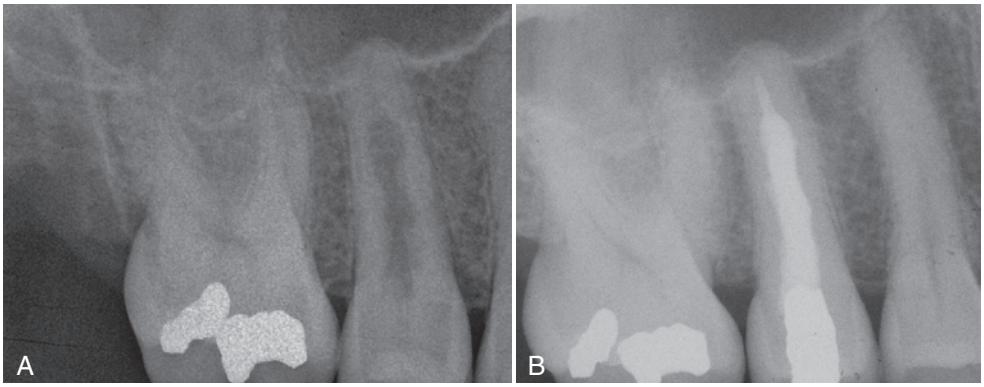

FIG. 20-38 A, Internal root resorption in a maxillary premolar with a history of trauma 7 years before the diagnosis (patient's head slammed against side window during an automobile accident). B, Three-year follow-up radiograph after endodontic treatment.

foramina, endodontic treatment that effectively removes the blood supply to the resorbing cells is the treatment approach. After adequate anesthesia has been obtained, the canal apical to the internal defect is explored, and a working length short of the radiographic apex is used. The apical canal is thoroughly cleaned and shaped to ensure that the blood supply to the tissue resorbing the root is cut off.

By completion of the root canal instrumentation, it should be possible to obtain a blood-free and dry canal with paper points. Calcium hydroxide is spun into the canal to facilitate the removal of the tissue in the irregular defect at the next visit. At the second visit, the tooth and defect are filled with a warm gutta-percha technique (Fig. 20-38).

Diagnostic Features of External Versus Internal Root Resorption

It is often very difficult to distinguish external from internal root resorption, so misdiagnosis and incorrect treatment may result. The following sections present a list of typical diagnostic features of each resorptive type.

Radiographic Features

A change of angulation of x-rays should give a fairly good indication of whether a resorptive defect is internal or external. A lesion of internal origin appears close to the canal, whatever the angle of the x-ray (see Fig. 20-37). A defect on the external aspect of the root moves away from the canal as the angulation changes (Fig. 20-39). By using the buccal object rule, it is usually possible to distinguish whether the external root defect is buccal or lingual-palatal.

With internal resorption, the outline of the root canal is usually distorted, and the root canal and radiolucent resorptive defect appear contiguous (see Fig. 20-37). When the defect is external, the root canal outline appears normal and can usually be seen "running through" the radiolucent defect (Fig. 20-40).

External inflammatory root resorption is always accompanied by resorption of the bone in addition to the root (Fig. 20-41); radiolucencies will be apparent in the root and the adjacent bone. Internal root resorption does not involve the bone, and as a rule the radiolucency is confined to the root (see Fig. 20-37). On rare occasions, if the internal defect perforates the root, the bone adjacent to it is resorbed and appears radiolucent on the radiograph.

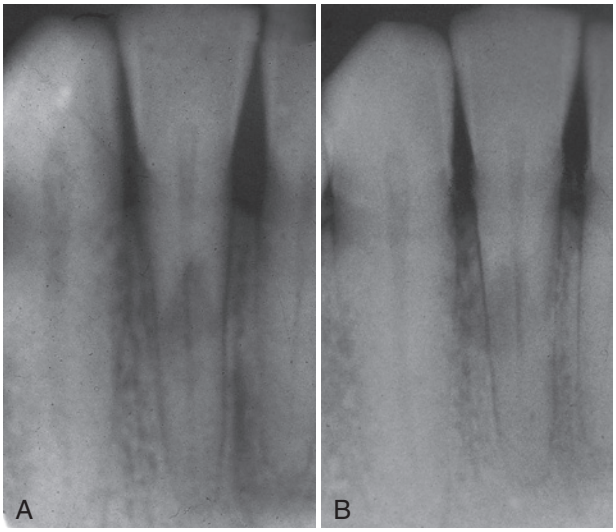

FIG. 20-39 External root resorption. Radiographs from two different horizontal projections depict movement of the lesion to outside the confines of the root canal.

FIG. 20-40 External root resorption on a mandibular premolar 6 years after completing orthodontic treatment. Note the mottled appearance of the resorptive defect and the outline of the root canal within the defect.

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FIG. 20-41 Mandibular molar with subepithelial external inflammatory resorption on its mesial aspect. Note the small opening into the root and the extensive resorption in the dentin; however, the pulp is not exposed. Also note that a resorptive defect is present in the adjacent bone, appearing on the radiograph as similar to an infrabony pocket.

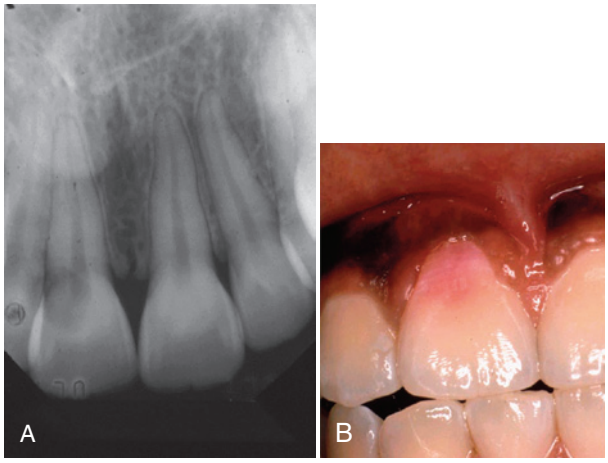

FIG. 20-42 Pink spot of subepithelial external inflammatory root resorption. A, Radiographic appearance. B, Clinical appearance.

Vitality Testing

External inflammatory resorption in the apical and lateral aspects of the root involves an infected pulp space, so no response to sensitivity tests supports the diagnosis. However, because subepithelial external root resorption does not involve the pulp (the bacteria are thought to originate in the sulcus of the tooth), a normal response to sensitivity testing is usually associated with this type of resorption. Internal root resorption usually occurs in teeth with vital pulps and responses to sensitivity testing. In teeth that exhibit internal root resorption, it is common to register a no-response to sensitivity testing because often the coronal pulp has been removed or is necrotic, and the active resorbing cells are more apical in the canal. Also, the pulp might have become necrotic after active resorption took place.

Pink Spot

With apical and lateral external root resorption, the pulp is nonvital, so the granulation tissue that produces the pink spot is not present in these cases. For subepithelial external (Fig. 20-42) and internal root resorption, the pink spot due to the granulation tissue undermining the enamel is a possible sign.

Summary of Possible Diagnostic Features

- External inflammatory root resorption due to pulp infection.
  - *Apical:* No response of the pulp to thermal or electric stimuli, with or without a history of trauma.
  - *Lateral:* History of trauma, no response of the pulp to thermal or electric stimuli, lesion moves on angled x-rays, root canal visualized radiographically overlying the defect, bony radiolucency also apparent.
- Subepithelial external inflammatory root resorption due to sulcular infection. History of trauma (often forgotten, or its long-term risks not appreciated by the patient); positive pulp sensitivity test; lesion located at the attachment level of the tooth; lesion moves on angled x-rays; root canal outline is undistorted and can be visualized radiographically; crestal bony defect associated with the lesion; pink spot possible.
- Internal root resorption. History of trauma, crown preparation, or pulpotomy; responsive pulp to thermal or electric stimuli likely, may occur at any location along the root canal (not only attachment level); lesion stays associated with the root canal on angled x-rays, radiolucency contained in the root without an adjacent bony defect; pink spot possible.

Most misdiagnoses of resorptive defects are made between subepithelial external and internal root resorptions. The diagnosis should always be confirmed as treatment proceeds. If root canal therapy is the treatment of choice for an apparent internal root resorption, the bleeding within the canal should cease quickly after pulp extirpation because the blood supply of the granulation tissue is the apical blood vessels. If bleeding continues during treatment, and particularly if it is still present at the second visit, the source of the blood supply is external, and treatment for perforating external resorption should be carried out. On obturation, it should be possible to fill the entire canal from within in internal resorption. Failure to achieve this should make the clinician suspicious of an external lesion that perforates the root. Finally, if the blood supply of an internal resorption defect is removed on pulp extirpation, any continuation of the resorptive process on recall radiographs should alert the clinician to the possibility that an external resorptive defect was misdiagnosed.

CLINICAL MANAGEMENT OF THE AVULSED TOOTH

Favorable healing after an avulsion injury requires quick emergency intervention followed by evaluation and possible treatment at decisive times during the healing phase. The urgency of the emergency visit and the multidisciplinary nature of follow-up evaluations require that both the public and clinicians from many dental disciplines be knowledgeable about the treatment strategies involved.

Consequences of Tooth Avulsion

Tooth avulsion results in attachment damage and pulp necrosis. The tooth is "separated" from the socket due mainly to tearing of the periodontal ligament that leaves viable periodontal ligament cells on most of the root surface. In addition, small and localized cemental damage occurs from the crushing of the tooth against the socket.

If the periodontal ligament left attached to the root surface does not dry out, the consequences of tooth avulsion are

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usually minimal.[14,146] The hydrated periodontal ligament cells will maintain their viability and repair after replantation, with minimal destructive inflammation as a by-product. Because the areas of the crushing injury are localized, inflammation stimulated by the damaged tissues will be correspondingly limited, and favorable healing with new replacement cementum is likely to occur after the initial inflammation subsides (see Fig. 20-26).

If excessive drying occurs before replantation, the damaged periodontal ligament cells elicit a severe inflammatory response over a diffuse area on the root surface. Unlike the situation described earlier, in which the area to be repaired after the initial inflammatory response is small, here a large area of root surface is affected that must be repaired by new tissue. The slower moving cementoblasts cannot cover the entire root surface in time, and it is likely that in certain areas, bone will attach directly onto the root surface. In time, through physiologic bone recontouring, the entire root will be replaced by bone. As earlier noted, this has been termed *osseous replacement* or *replacement resorption* (see Figs. 20-27 and 20-28).[20,163]

Pulpal necrosis always occurs after an avulsion injury. Although a necrotic pulp itself is not of consequence, the necrotic tissue is extremely susceptible to bacterial contamination. If revascularization does not occur or effective endodontic therapy is not carried out, the pulp space inevitably becomes infected. The combination of bacteria in the root canal and cemental damage on the external surface of the root results in an external inflammatory resorption that can be very serious and can lead to rapid loss of the tooth (see Fig. 20-30).[160]

The consequences after tooth avulsion appear to be directly related to the severity and surface area of the inflammation on the root surface and resultant damaged root surface that must be repaired. Treatment strategies should always be considered in the context of limiting the extent of the periradicular inflammation, thus tipping the balance toward favorable responses (cemental) rather than unfavorable ones (osseous replacement or inflammatory resorption).

Treatment Objectives

Treatment is directed at avoiding or minimizing resultant inflammation due to the two main consequences of the avulsed tooth: attachment damage and pulpal infection.

Attachment damage as a direct result of the avulsion injury cannot be avoided. However, considerable additional damage can occur to the periodontal ligament in the time that the tooth is out of the mouth (primarily because of drying). Treatment is directed at minimizing this damage (and the resultant inflammation) so that the fewest possible complications result. When severe additional damage cannot be avoided and osseous replacement of the root is considered certain, steps are taken to slow the replacement of the root by bone to maintain the tooth in the mouth for as long as possible.

In the open apex tooth, all efforts are made to promote revascularization of the pulp, thus avoiding pulp space infection. When revascularization fails (in the open apex tooth) or is not possible (in the closed apex tooth), all treatment efforts are made to prevent or eliminate toxins from the root canal space.

Clinical Management

Emergency Treatment at the Accident Site

Replant if possible or place in an appropriate storage medium. As mentioned, damage to the attachment apparatus that occurred during the initial injury is unavoidable but usually minimal. However, all efforts must be made to minimize necrosis of the remaining periodontal ligament while the tooth is out of the mouth. Pulpal sequelae are not a concern initially and are dealt with at a later stage of treatment.

The single most important factor to assure a favorable outcome after replantation is the speed with which the tooth is replanted.[16,22] Of utmost importance is the prevention of drying, which causes loss of normal physiologic metabolism and morphology of the periodontal ligament cells.[22,146] Every effort should be made to replant the tooth within the first 15 to 20 minutes.[27] This usually requires emergency personnel at the site of the injury with some knowledge of treatment protocol. The clinician should communicate clearly with the person at the site of the accident. Ideally this information should have been given at an earlier time; for example, as an educational offering to school nurses or athletic trainers. Failing this, the information can be given over the phone. The aim is to replant a clean tooth with an undamaged root surface as gently as possible, after which the patient should be brought to the office immediately. If doubt exists that the tooth can be replanted adequately, the tooth should quickly be stored in an appropriate medium until the patient can get to the dental office for replantation. Suggested storage media, in order of preference, are milk, saliva (either in the vestibule of the mouth or in a container into which the patient expectorates), physiologic saline, and water.[86] Water is the least desirable storage medium because the hypotonic environment causes rapid cell lysis and increased inflammation on replantation.[35,36]

Cell culture media in specialized transport containers, such as Hank's Balanced Salt Solution (HBSS), have shown superior ability to maintain the viability of the periodontal ligament fibers for extended periods.[165] Presently they are considered impractical because they need to be present at the accident site before the injury occurs. However, if we consider that more than 60% of avulsion injuries occur close to home or school,[81] it seems reasonable to assume that it would be beneficial to have these media available in emergency kits at these sites. It would also be advantageous to have them in ambulances and in the kits of emergency response personnel who are likely to treat the more serious injuries in which teeth might otherwise be sacrificed to a more serious life-threatening situation.

Management in the Dental Office

Emergency Visit

Prepare socket, prepare root, replant, construct a functional splint, and administer local and systemic antibiotics.

Recognizing that a dental injury might be secondary to a more serious injury is essential. The attending dental clinician is likely to be the first health care provider the patient sees after a head injury, so ruling out any injuries to the brain (e.g., concussion) and/or central nervous system (CNS) in general is paramount. If on examination a CNS injury is suspected, immediate referral to the appropriate expert is the first priority, above and beyond the dental injury. Once a CNS injury has been ruled out, the focus of the emergency visit is the attachment apparatus. The aim is to replant the tooth with a minimum of irreversibly damaged cells (that will cause inflammation) and the maximal number of periodontal ligament cells that have the potential to regenerate and repair the damaged root surface.

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Diagnosis and Treatment Planning

If the tooth was replanted at the site of injury, a complete history is taken to assess the likelihood of a favorable outcome. The position of the replanted tooth is assessed and adjusted if necessary. On rare occasions, the tooth may be gently removed to prepare the root to increase the chances of a favorable outcome (discussed later).

If the patient presents with the tooth out of the mouth, the storage medium should be evaluated and the tooth placed in a more appropriate medium if required. HBSS is presently considered the best medium for this purpose. Milk or physiologic saline is also appropriate for storage purposes.

The medical and accident histories are taken, and a clinical exam is carried out, with emphasis on questions about when, how, and where the injury occurred.

The clinical examination should include an examination of the socket to ascertain whether it is intact and suitable for replantation. The socket is gently rinsed with saline, and when it has been cleared of the clot and debris, its walls are examined directly for the presence, absence, or collapse of the socket wall. The socket and surrounding areas, including the soft tissues, should be radiographed. Three vertical angulations are required for diagnosis of the presence of a horizontal root fracture in adjacent teeth.[16] The remaining teeth in both the upper and lower jaws should be examined for injuries, such as crown fractures. Any soft tissue lacerations should be noted and, if tooth fragments are missing, explored.

PREPARATION OF THE ROOT

Preparation of the root depends on the maturity of the tooth (open versus closed apex) and on the dry time of the tooth before it was placed in a storage medium. A dry time of 60 minutes is considered the point where survival of root periodontal ligament cells is unlikely.

Extraoral Dry Time Less Than 60 Minutes

Closed Apex

The root should be rinsed of debris with water or saline and replanted in as gentle a fashion as possible.

If the tooth has a closed apex, revascularization is not possible,[51] but because the tooth was dry for less than 60 minutes (replanted or placed in appropriate medium), the chance for periodontal healing exists. Most important, the chance of a severe inflammatory response at the time of replantation is lessened. A dry time of less than 15 to 20 minutes is considered optimal, and periodontal healing would be expected.[14,27,146]

A continuing challenge is the treatment of the tooth that has been dry for more than 20 minutes (periodontal cell survival is assured) but less than 60 minutes (periodontal cell survival unlikely). In these cases, logic suggests that the root surface consists of some cells with the potential to regenerate and some that will act as inflammatory stimulators.

Open Apex

Gently rinse off debris, soak in doxycycline for 5 minutes or cover with minocycline, replant.

In an open apex tooth, revascularization of the pulp and continued root development are possible (see Fig. 20-32). Investigators[51] found in monkeys that soaking the tooth in doxycycline (1 mg in approximately 20 mL of physiologic saline) for 5 minutes before replantation significantly enhanced complete revascularization. This result was confirmed later in dogs by other investigators.[127,176] A study found that covering the root with minocycline (Arestin, OraPharma, Warminster, Pennsylvania), which attaches to the root for approximately 15 days, further increased the revascularization rate in dogs.[127] Although animal studies do not provide us with a prediction of the rate of revascularization in humans, it is reasonable to expect that the same enhancement of revascularization that occurred in two animal species also will occur in humans. As for a closed apex tooth, the open apex tooth is gently rinsed and replanted.

Extraoral Dry Time More Than 60 Minutes

Closed Apex

Remove the periodontal ligament by placing in acid for 5 minutes, soak in fluoride, replant.

When the root has been dry for 60 minutes or more, survival of the periodontal ligament cells is not expected.[22,146] In such cases the root should be prepared to be as resistant to resorption as possible (attempting to slow the osseous replacement process). These teeth should be soaked in acid for 5 minutes to remove all remaining periodontal ligament and thus remove the tissue that would initiate the inflammatory response on replantation. The tooth should then be soaked in 2% stannous fluoride for 5 minutes and replanted.[34,137] A few years ago, studies were published that indicated that an enamel matrix protein, Emdogain (Straumann USA, Andover, Massachusetts), could be beneficial in teeth with extended extraoral dry times, not only to make the root more resistant to resorption, but also, possibly, to stimulate the formation of new periodontal ligament from the socket (see Fig. 20-8).[64,90] Unfortunately, more recent studies have shown that the positive effect of Emdogain is only temporary, and most of these teeth start to resorb after a few years.[132]

If the tooth has been dry for more than 60 minutes and no consideration is given to preserving the periodontal ligament, the endodontics may be performed extraorally. In the case of a tooth with a closed apex, no advantage exists to this additional step at the emergency visit. However, in a tooth with an open apex, endodontic treatment performed after replantation involves a long-term apexification procedure. In these cases, completing the root canal treatment extraorally, in which a seal in the blunderbuss apex is easier to achieve, may be advantageous. When endodontic treatment is performed extraorally, it must be performed aseptically with the utmost care to achieve a root canal system that is thoroughly disinfected.

Open Apex

Replant? If yes, treat as with closed apex tooth. Endodontic treatment may be performed out of the mouth.

Because these teeth are in young patients in whom facial development is usually incomplete, many pediatric clinicians consider the prognosis to be so poor and the potential complications of an ankylosed tooth so severe, they recommend that these teeth not be replanted. Considerable debate exists as to whether it would be beneficial to replant the root even though it will inevitably be lost due to osseous replacement. If the patients are followed carefully and the root submerged by decoronation procedure at the appropriate time,[6,63,67] the height, and more important, the width of the alveolar bone will

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be maintained, allowing for easier permanent restoration at the appropriate time when the child's facial development is complete.

PREPARATION OF THE SOCKET

The socket should be left undisturbed before replantation.[16] Emphasis is placed on removal of obstacles in the socket to facilitate replacement of the tooth into the socket.[78] It should be lightly aspirated if a blood clot is present. If the alveolar bone has collapsed or may interfere with replantation, a blunt instrument should be inserted carefully into the socket in an attempt to reposition the wall.

SPLINTING

A splint that allows for physiologic movement of the tooth during healing and that is in place for a minimal period results in a decreased incidence of ankylosis.[3,12,78] Semirigid (physiologic) fixation for 1 to 2 weeks is recommended.[3,5,67] The splint should allow movement of the tooth, should have no memory (so the tooth is not moved during healing), and should not impinge on the gingiva and/or prevent maintenance of oral hygiene in the area. Many splints satisfy the requirements of an acceptable device. A new titanium trauma splint (TTS) has recently been shown to be particularly effective and easy to use (Fig. 20-43).[169] After the splint is in place, a radiograph should be exposed to verify the positioning of the tooth and as a preoperative reference for further treatment and follow-up. When the tooth is in the best possible position, adjusting the bite to ensure that it has not been splinted in a position causing traumatic occlusion is important. One week is sufficient to create periodontal support to maintain the avulsed tooth in position.[164] Therefore, the splint should be removed after 1 to 2 weeks. The only exception is with avulsion in conjunction with alveolar fractures, for which 4 to 8 weeks is the suggested time of splinting.[164]

MANAGEMENT OF THE SOFT TISSUES

Soft tissue lacerations of the socket gingiva should be tightly sutured. Lacerations of the lip are fairly common with these types of injuries. The clinician should approach lip lacerations with some caution; a plastic surgery consult might be prudent.

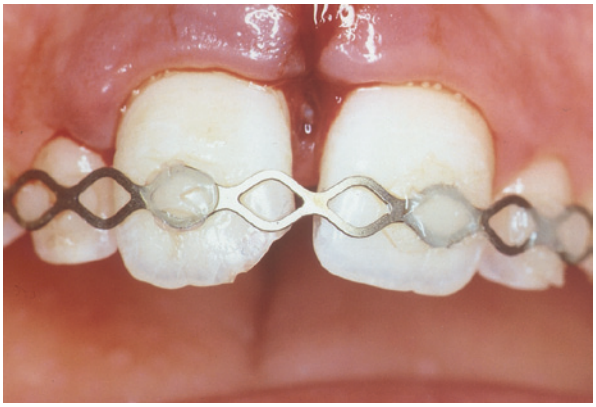

FIG. 20-43 Titanium trauma splint (TTS) in place.

If these lacerations are sutured, care must be taken to clean the wound thoroughly beforehand because dirt or even minute tooth fragments left in the wound affect healing and the aesthetic result.

ADJUNCTIVE THERAPY

Systemic antibiotics given at the time of replantation and before endodontic treatment are effective in preventing bacterial invasion of the necrotic pulp and therefore subsequent inflammatory resorption.[79] Tetracycline has the additional benefit of decreasing root resorption by affecting the motility of the osteoclasts and reducing the effectiveness of collagenase.[128] The administration of systemic antibiotics is recommended, beginning at the emergency visit and continuing until the splint is removed.[79] For patients not susceptible to tetracycline staining, the antibiotic of choice is doxycycline twice daily for 7 days at the appropriate dosage for patient age and weight.[128,129] Penicillin V 1000 mg as a loading dose, followed by 500 mg 4 times daily for 7 days, has also been shown to be beneficial. The bacterial content of the sulcus also should be controlled during the healing phase. In addition to stressing to the patient the need for adequate oral hygiene, the use of chlorhexidine rinses for 7 to 10 days is helpful.

As stated previously, a recent series of studies by our research group found great benefit in removal of the pulp contents at the emergency visit and placing Ledermix or corticosteroid into the root canal.[38,41] Apparently the use of the medicament was able to shut down the inflammatory response after replantation to allow for more favorable healing compared to those teeth that did not have the medicament.

The need for analgesics should be assessed on an individual case basis. The use of pain medication stronger than nonprescription nonsteroidal antiinflammatory drugs (NSAIDs) is unusual. The patient should be sent to a physician for consultation regarding a tetanus booster within 48 hours of the initial visit.

Second Visit

The second visit should take place 1 to 2 weeks after the trauma. At the emergency visit, emphasis was placed on the preservation and healing of the attachment apparatus. The focus of the second visit is the prevention or elimination of potential irritants from the root canal space. These irritants, if present, provide the stimulus for the progression of the inflammatory response, bone and root resorption. Also at this visit, the course of systemic antibiotics is completed; the chlorhexidine rinses can be stopped. At this appointment the splint is removed; the tooth might still have class I or class II mobility after splint removal, but all indications are that it will continue to heal better without the splint.[3]

ENDODONTIC TREATMENT

Extraoral Time Less Than 60 Minutes

Closed Apex

Initiate endodontic treatment after 1 to 2 weeks. When endodontic treatment is delayed or signs of resorption are present, provide long-term calcium hydroxide treatment before obturation.

No chance exists for revascularization of teeth with closed apices; therefore, endodontic treatment should be initiated at

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the second visit 7 to 10 days later.[13,51] If therapy is initiated at this optimum time, the pulp should be necrotic without (or with minimal) infection.[108,166] Endodontic therapy with an effective interappointment antibacterial agent[166] over a relatively short period (1 to 2 weeks) is sufficient to ensure effective disinfection of the canal.[141] Long-term calcium hydroxide treatment should always be considered if the injury occurred more than 2 weeks before initiation of the endodontic treatment or especially if radiographic evidence of resorption is present.[166]

The root canal is thoroughly cleaned and shaped, irrigated, and then filled with a thick (powdery) mix of calcium hydroxide and sterile saline (anesthetic solution is also an acceptable vehicle) (see Fig. 20-15). The canal is obturated when a radiographically intact periodontal membrane can be demonstrated around the root (see Fig. 20-35). Calcium hydroxide is an effective antibacterial agent[39,141] and favorably influences the local environment at the resorption site, theoretically promoting healing.[161] It also changes the environment in the dentin to a more alkaline pH, which may slow the action of the resorptive cells and promote hard tissue formation.[161] However, changing of the calcium hydroxide should be kept to a minimum (not more than every 3 months) because it has a necrotizing effect on the cells attempting to repopulate the damaged root surface.[104]

Calcium hydroxide is considered the drug of choice in the prevention and treatment of inflammatory root resorption, but it is not the only medicament recommended in these cases. Some attempts have been made not only to remove the stimulus for the resorbing cells, but also to affect them directly. The antibiotic-corticosteroid paste Ledermix is effective in treating inflammatory root resorption by inhibiting the spread of dentinoclasts[122] without damaging the periodontal ligament; however, its ability to diffuse through the human tooth root has been demonstrated,[1] and its release and diffusion are enhanced when it is used in combination with calcium hydroxide paste.[2]

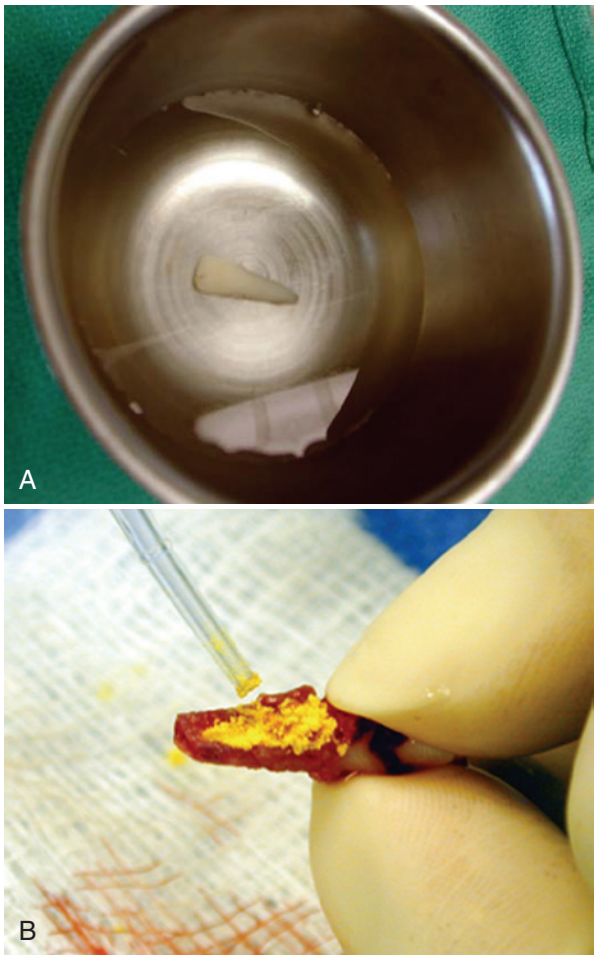

FIG. 20-44 **A**, Avulsed tooth soaking in doxycycline. **B**, Minocycline powder placed on the root surface before replantation.

Open Apex

Avoid endodontic treatment and look for signs of revascularization. At the first sign of an infected pulp, initiate apexification procedure.

Teeth with open apices have the potential to revascularize and continue root development; initial treatment is directed toward reestablishing the blood supply (Fig. 20-44).[51,127,175] The initiation of endodontic treatment is avoided if at all possible, unless definite signs of pulp necrosis are present (e.g., periradicular inflammation). An accurate diagnosis of pulp vitality is extremely challenging in these cases. After trauma, a diagnosis of necrotic pulp is particularly undesirable because infection in these teeth is potentially more harmful due to cemental damage accompanying the traumatic injury. External inflammatory root resorption can be extremely rapid in these young teeth because the tubules are wide and allow irritants to move freely to the external surface of the root.[51,175]

Patients are recalled every 3 to 4 weeks for pulp vitality testing. Studies indicate that thermal tests with carbon dioxide snow (−78° C) or dichlorodifluoromethane (−40° C) placed at the incisal edge or pulp horn are the best methods of sensitivity testing, particularly in young permanent teeth.[71,72,117] One of these two tests must be included in the pulp vitality testing. Recent reports confirm the superiority of the laser Doppler flowmeter in the diagnosis of revascularization of traumatized immature teeth[176]; however, the cost of such an instrument precludes its use in the average dental office. Radiographic signs (apical breakdown and/or signs of lateral root resorption) and clinical signs (pain on percussion and palpation) of pulp pathosis are carefully assessed. At the first sign of pathosis, endodontic treatment should be initiated, and after disinfection of the root canal space, an apexification procedure should be carried out.

Extraoral Time More Than 60 Minutes

Closed Apex

Teeth with closed apices are treated endodontically in the same way as teeth that had an extraoral time of less than 60 minutes.

Open Apex (if Replanted)

If endodontic treatment was not performed out of the mouth, initiate apexification procedure.

The chance of revascularization in these teeth is extremely poor,[163,167] so no attempt is made to revitalize them. An apexification procedure is initiated at the second visit if root canal treatment was not performed at the emergency visit. If endodontic treatment was performed at the emergency visit, the second visit is a recall visit to assess initial healing only.

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TEMPORARY RESTORATION

Effectively sealing the coronal access is essential to prevent infection of the canal between visits. Recommended temporary restorations are reinforced zinc oxide eugenol cement, acid etch composite resin, or glass ionomer cement. The depth of the temporary restoration is critical to its sealability. A depth of at least 4 mm is recommended, so a cotton pellet should not be placed; the temporary restoration is placed directly onto the calcium hydroxide in the access cavity. Calcium hydroxide should be removed from the walls of the access cavity before the temporary restoration is placed because calcium hydroxide is soluble and washes out when it comes into contact with saliva, leaving a defective temporary restoration.

After initiation of the root canal treatment, the splint is removed. If time does not permit complete removal of the splint at this visit, the resin tacks are smoothed so as not to irritate the soft tissues; the remaining resin is removed at a later appointment.

At this appointment, healing is usually sufficient to allow a detailed clinical examination of the teeth surrounding the avulsed tooth. Pulp vitality tests, reaction to percussion and palpation, and periodontal probing measurements should be carefully recorded for reference at follow-up visits.

Root Filling Visit

This visit should take place at the clinician's convenience or after long-term calcium hydroxide therapy, when an intact lamina dura is traced.

If the endodontic treatment was initiated 1 to 2 weeks after the avulsion and a thorough examination confirms normality, filling of the root canal at this visit is acceptable. Long-term use of calcium hydroxide is also a proven option for these cases. If endodontic treatment was initiated more than 2 weeks after the avulsion or active resorption is visible, the pulp space must first be disinfected before root filling. Traditionally, the reestablishment of a lamina dura (see Fig. 20-35) is a radiographic sign that the canal bacteria have been controlled. When an intact lamina dura can be traced throughout, root filling can take place.

The canal is cleaned, shaped, and irrigated under strict asepsis (i.e., a rubber dam). After completion of cleaning and shaping, the canal can be filled.

PERMANENT RESTORATION

Much evidence exists that coronal leakage caused by defective temporary and permanent restorations results in a clinically relevant amount of bacterial contamination of the root canal after root filling.[126] Therefore, the tooth should receive a permanent restoration as soon as possible. The depth of restoration is important for a tight seal, so the deepest restoration possible should be made. A post should be avoided if possible. Because most avulsions occur in the anterior region of the mouth where aesthetics is important, composite resins combined with dentin bonding agents are recommended in these cases.

Follow-Up Care

Follow-up evaluations should take place at 3 months, 6 months, and yearly for at least 5 years. If osseous replacement is identified (see Fig. 20-28), a more closely monitored follow-up schedule is indicated. In the case of inflammatory root resorption (see Fig. 20-30), a new attempt at disinfection of the root canal space by standard retreatment might reverse the process. Teeth adjacent to and surrounding the avulsed tooth or teeth may show pathologic changes long after the initial accident, so these teeth, too, should be tested at follow-up visits and the results compared to those collected soon after the accident.

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CHAPTER 21

Cracks and Fractures

ZVI METZGER | LOUIS H. BERMAN | AVIAD TAMSE

CHAPTER OUTLINE

Unobserved Trauma
Diagnostic Challenge
Fracture Mechanics
Cracked and Fractured Cusps
Definition
Diagnosis
Etiology
Treatment Planning
Cracked and Split Teeth
Definition
Diagnosis
Etiology
Treatment Planning
Vertical Root Fracture (VRF)
Definition
Diagnosis
Etiology
Treatment Planning
Summary

Root cracks and fractures can be two of the most frustrating aspects of endodontic and restorative dentistry. The diagnosis can be difficult; the symptoms can be either vague or specific, yet they are often insufficient for a definitive diagnosis; the radiographic evaluation can be evasive. Clinical management of the crack or fracture depends on its extent. Prevention of a potential crack or fracture is a fundamental principle, and early detection is imperative.

UNOBSERVED TRAUMA

Tooth fracture is commonly associated with impact trauma. A car accident, a fall from a bicycle, and an accidental blow to the face are among the common causes. These types of traumatic fractures mainly occur in the anterior segment of the mouth and are covered elsewhere in this book (see Chapter 20). In contrast, the cracks and fractures described in this chapter are often not associated with a traumatic event that the patient can remember. These cracks and fractures are frequently the result of an accumulating, unobserved trauma resulting from either normal or excessive occlusal forces that are repetitively applied[22,34,45,47,70,71,90] without the patient's awareness.

DIAGNOSTIC CHALLENGE

Three categories of cracks and fractures are discussed in this chapter: cracked and fractured cusps, cracked and split teeth, and vertical root fractures. Each is often undiagnosed or misdiagnosed for a relatively long time.[16,19,80] The astute clinician can usually diagnose a pulpitis, apical periodontitis, or an abscess after appropriate diagnostic tests and clinical assessments are performed. However, because of the wide variety of clinical presentations from cracks and fractures, the diagnosis is less straightforward.[16,18,78] To complicate the diagnosis, not one of the three entities necessarily exhibits a radiographic manifestation in the early stages, depriving the dentist of one of the most objective diagnostic tools.

Symptoms from one of these conditions may be present for *several months* before an accurate diagnosis is made,[5,16,19] which may be frustrating for both the patient and the dentist, and often causes the patient to develop a subsequent loss of trust and confidence in the dentist.

The final diagnosis is typically reached at a relatively late stage of these conditions, often after complications have already occurred. Complications may include a catastrophic fracture of the tooth or cusp or significant periradicular bone loss associated with a vertical root fracture as seen by radiographic examination (see also Chapter 2). For this reason, this chapter emphasizes the *early diagnosis* of these conditions. The collection of signs and symptoms associated with each of the three categories of cracks and fractures may be confusing unless the clinician considers these indicators as representing a continuous process examined at a given time point.

FRACTURE MECHANICS

Fracture mechanics is the field of biomechanics concerned with the propagation of *cracks* in a given material until the formation of the final catastrophic *fracture*.[7,52] Many terms have been loosely used in the dental literature to describe the clinical entities that are the subject of this chapter.[5,9,18,35] Here, the term

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FIG. 21-1 Complete vertical root fracture. A cross section of a mesial root of a mandibular molar with a complete vertical root fracture. The fracture is in the buccolingual plane and extends from the buccal convexity of the root to its lingual convexity.

crack will be used in the biomechanical sense: a *partial discontinuity* in a material that may propagate and eventually lead to a complete discontinuity, also known as a *fracture*.[7,52] In this context, a cusp may be described as *cracked*, a condition leading with time to a *fractured cusp*. A *cracked tooth* may be so called until a final fracture occurs that separates the tooth into two parts, a condition termed a *split tooth*. Similarly, microcracks may appear in the radicular dentin of an endodontically treated tooth, and these cracks may propagate with time until a *vertical root fracture* occurs, at which point the full thickness of the dentinal wall shows discontinuity: a through-and-through fracture. Such a fracture may be *incomplete*, involving one wall of the root, or *complete*, separating the root into two parts[80] (Fig. 21-1).

CRACKED AND FRACTURED CUSPS

Definition

A *cracked cusp* is characterized by a crack between a cusp and the rest of the tooth structure, to the extent of allowing microscopic flexure upon mastication. This crack typically does not involve the pulp. With time, the crack may propagate, eventually resulting in a *fractured cusp*.[5]

Diagnosis

Patient History

In the case of a cracked cusp, the patient history is the most important tool for making a diagnosis. The patient will likely complain about pain when chewing, to the extent of not being able to chew on the side on which the crack occurred.[18,35,69] The patient will also often state that the condition existed for a relatively long time and that his or her dentist could not find the source or glean any information from radiography.[3,16,47] When asked whether the pain is sharp or dull, patients will typically report a sharp pain that makes them immediately stop chewing on that side. The diagnostic challenge is attempting to determine which cusp of which tooth is involved, as patients often have difficulties determining the specific location of the discomfort.[16,18,35,69] Because the pain has a pulpal origin, the patient's proprioception may not be accurate, as no periodontal ligament is involved. Occasionally, the pain upon chewing may radiate to nondental locations on the same side of the face,[16,47,71] as also described in Chapters 4 and 17.

Clinical Manifestation

Early Manifestation

The typical characteristic of cracked cusps is a sharp pain upon chewing, although the affected tooth may not be sensitive, or only selectively sensitive, to percussion.[70] The tooth is vital, and its response to a cold stimulus may be normal; but with time, this response may resemble a pulpitis, which may be either localized or referred to other odontogenic or nonodontogenic locations.[16,47,71]

Cracked cusps are often associated with extensive occlusal restorations,[3,29,71,78] which may undermine and weaken the cusp and predispose it to initiating or perpetuating a crack from occlusal forces. Nevertheless, cracked cusps may also be present in intact teeth or teeth with smaller restorations.[13,70]

Late Manifestation

With time, a crack may propagate and result in a fractured cusp. If the fracture line occurs coronally to the periodontal ligament the fractured portion will simply separate from the tooth. However, if the fracture line extends subgingivally, gingival fibers or the periodontal ligament will often retain the fractured cusp. Initially, it may be possible to move the cusp by wedging a sharp explorer into the fracture line, making the fractured cusp more visible. Often, from continued mastication, a localized and more acute type of pain may emerge secondary to the movement of the fractured fragment in the coronal PDL. The pulpal pain that is typical at an earlier stage (the cracked cusp) will typically resolve once a complete fracture occurs.

Diagnosis

A cracked cusp may be diagnosed, to a large extent, on the basis of the patient history. To locate the affected tooth, a *biting test* should be performed using a Tooth Slooth (Professional Results, Laguna Niguel, CA) or a similar device[9,12] (Fig. 21-2). The device is composed of a small pyramid with a flattened top that is placed on a selective cusp, while the wider part of the device is applied to several opposing teeth while the patient occludes (see Fig. 21-2). The application of these forces to a cracked cusp will generate a sharp pain, which may occur upon pressure or released.[3,34,43] The patient will typically state that this sensation has reproduced the sensation of the chief complaint.

Magnification with such devices as loupes or an operating microscope can be helpful when looking for a crack. If the tooth does not have an extensive intracoronal restoration, *transillumination* may also assist in revealing the crack line. If the tooth has a large restoration, the removal of the restoration may facilitate the effective use of this diagnostic tool (Fig. 21-3). The light source should be intense but with small dimensions (Fig. 21-4); it is applied to the tooth at the area of the suspected cusp fracture, with the lights of the dental unit, microscope, and room extinguished. The light penetrates the tooth structure up to the crack, leaving the part beyond the crack relatively dark (see Fig. 21-3). However, when large intracoronal restorations are present, this type of examination may be less effective.

Once the crack propagates, resulting in a fractured cusp, the diagnosis becomes more straightforward: the fractured cusp will either be missing or moved by wedging an explorer into the fracture line (Fig. 21-5).

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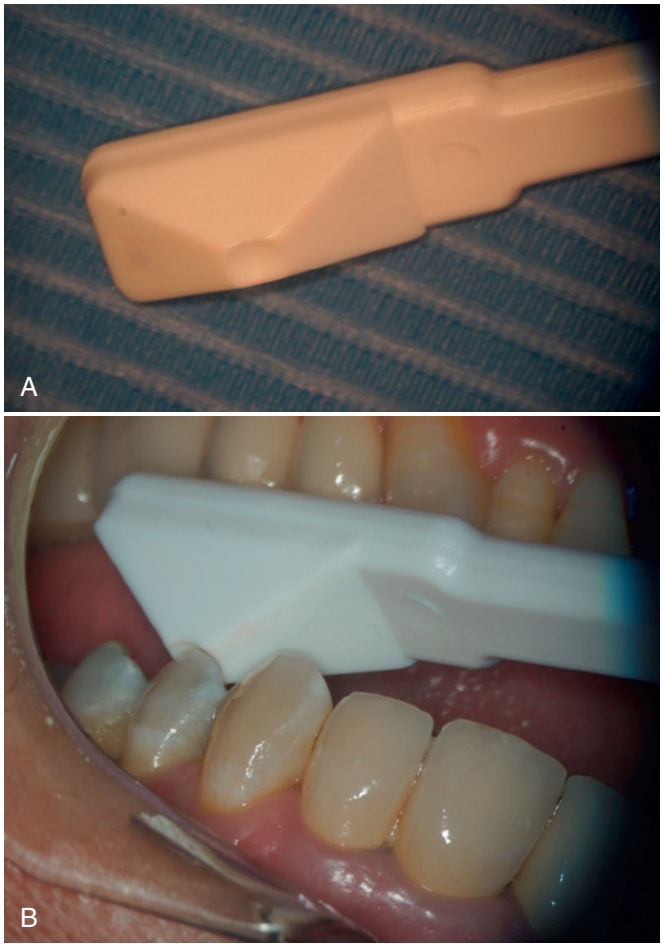

FIG. 21-2 **A Tooth Slooth device. A,** The Tooth Slooth device. **B,** Application for a bite test: the tip of the pyramid is touching the tested cusp while the wide base is supported by multiple contacts.

Etiology

Extensive intracoronal restorations may be a predisposing factor for cracked and fractured cusps.[3,29,71,78] Otherwise, the etiology of these conditions is similar to that of cracked teeth (see Cracked and Split Teeth, Etiology).

Treatment Planning

Cracked Cusp

Treatment should consist of protecting the affected cusp from occlusal forces, both to prevent pain while chewing and to prevent the propagation of the crack into a full fracture. A full-coverage crown or onlay is recommended,[5,16,39] although bonded composite restorations have also been proposed.[28,64] One should keep in mind that if the cracked cusp is not protected, the tooth may eventually fracture. If the fracture plane extends apically into the root, the tooth will be potentially nonrestorable.[5,9,12] Endodontic treatment is indicated only if signs and symptoms of pulpal pathosis are observed. In addition, if the removal of the cracked cusp and associated restoration will result in little or no remaining coronal tooth structure, then elective root canal treatment may be necessary for prosthetic reasons. When such a treatment plan is selected, one should also perform an occlusal reduction of the tooth as soon as possible to remove the tooth from active occlusion. The patient should be instructed to be careful when chewing until the tooth is restored with a crown.

Fractured Cusp

The treatment of a fractured cusp depends on the amount of tooth structure remaining. If the missing part is limited in size, then the conservative restoration of a bonded composite resin may be indicated to cover the exposed dentin. In contrast, when a larger fragment has fractured and is either removed or missing, a full crown or an onlay may be necessary.

In certain cases, when cracked cusps are found in intact teeth or in teeth with no extensive restoration, it is difficult to predict the direction in which the crack is propagating. Therefore, in these cases, when considering endodontic and restorative treatment, the patient should be advised as to the potential decrease in prognosis, as described later.

CRACKED AND SPLIT TEETH

Definition

A *cracked tooth* exhibits a crack that incompletely separates the tooth crown into two parts. If the crack is allowed to propagate longitudinally, the tooth will eventually fracture into two fragments, resulting in a *split tooth*.

Diagnosis

Patient History

In cases of a cracked tooth, the patient history may be similar to that for a cracked cusp—namely, sharp pain upon mastication and prolonged failure of the dentist to diagnose the source of the pain.[16,19] Similar to a cracked cusp, the diagnosis of a cracked tooth is often made on the basis of the patient history alone. Often it is challenging for the practitioner to determine the location of the offending tooth. With time, the patient may report that he or she *used to have* a sharp pain and now experiences great sensitivity to cold stimuli; the patient may even report, at a later stage, that the pain has subsided. These observations are consistent with pulpitis or pulp necrosis, which may develop in the affected tooth with time.[13]

Clinical Manifestation

Early Manifestation

Cracked teeth may have extensive restorations with a weakened crown, or they may have minimal or no restorations. A cracked tooth begins with a crack in the clinical crown, which may gradually propagate in an apical direction.[3,5,28,47] Such cracks typically run in the mesiodistal direction, often splitting the crown into the buccal and lingual fragments. In the early stages, the tooth may be vital and painful to mastication. The pain may be sharp, to the extent that the patient is unable to chew on the affected side. This condition may persist for an extended period of time.[16,18,19] The pain may be localized or referred to any tooth, maxillary or mandibular, on the same side of the mouth.[16,47,71] No radiographic manifestations are present at these early stages, as the crack is microscopic and runs perpendicular to the x-ray beam. The affected tooth may or may not be sensitive to percussion at this point, and pulp testing may be normal or indicative of increased sensitivity to cold stimuli.

Late Manifestation

The late manifestation of a cracked tooth may include *pulp involvement* and eventually the *loss of pulp vitality*[13] or apical

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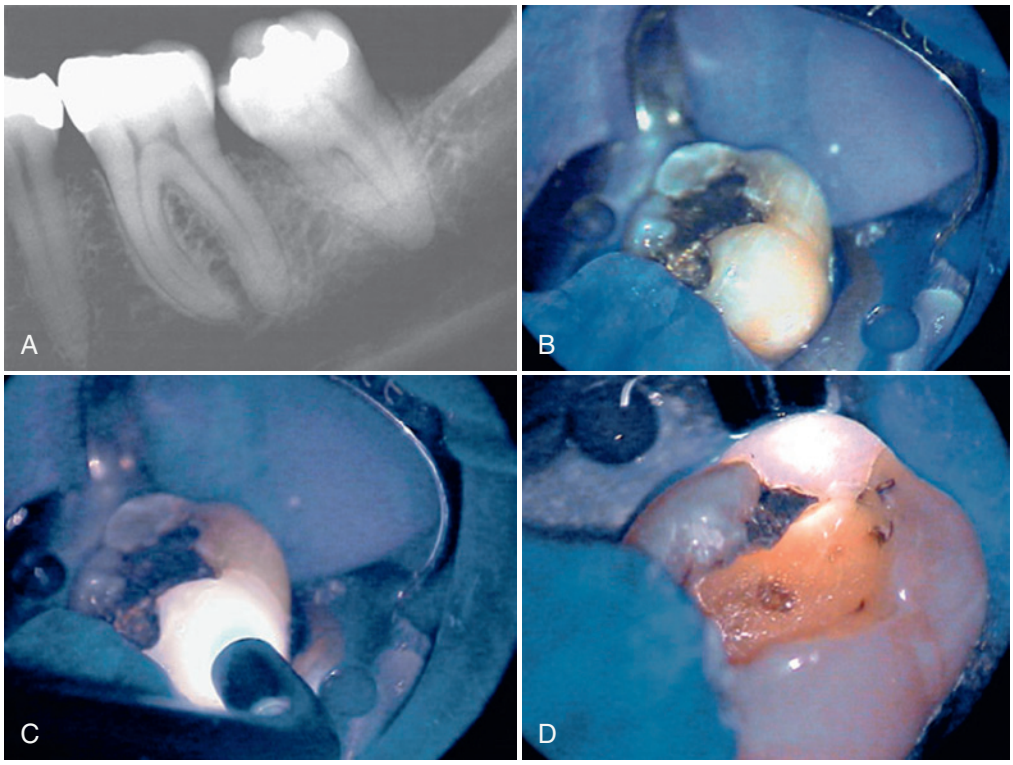

FIG. 21-3 Transillumination for the detection of a cracked cusp/tooth. An intense but small light source is applied to the suspect tooth, preferably in relative darkness. The light is transmitted through the tooth structure but is reflected from the crack plane, leaving the area behind the crack in darkness.

FIG. 21-4 Light source for transillumination. The light source should be intense yet with small dimensions, such as the simple battery-operated device manufactured by Yeti Dental (Engen, Germany).

propagation of the fracture, resulting in a *split tooth*. In one investigation,[13] 27 nonvital molar and premolar teeth that had minimal or no restorations or caries were studied. Upon extraction, these teeth were examined under a surgical operating microscope or using micro–computed tomography (CT) analysis. In each of these teeth, a longitudinal crack was observed, which extended to the pulp. Although the study had a limited sample size, the clinician should understand the potentially poor prognosis from cracked teeth, especially when the crack is suspected to be the cause of pulp necrosis.[13]

Pulp involvement occurs more often in cases of centrally located cracks (i.e., extending from marginal ridge to marginal ridge through the central fossa) than in cracks with a more buccal or lingual location.[18,90] These centrally located cracks commonly affect the roof of the pulp chamber at a later stage. Consequently, pulp vitality may be compromised and later lost due to bacterial penetration through the crack. The pulp may first become reversibly or irreversibly inflamed and later necrotic and infected. The sharp pain upon mastication that is typical of the early stage may disappear once pulp vitality is lost. Moreover, apical periodontitis in an apparently intact molar may be a late manifestation of an untreated case of a cracked tooth.[13] When pulp necrosis occurs, the radiographic manifestation may be an apical radiolucency, which is undistinguishable from that of apical periodontitis (Fig. 21-6).

A crack may propagate with time through the pulp chamber and into the root, resulting in a complete fracture that separates the tooth into two parts, a condition termed *split tooth*. When this split occurs, the resulting parts of the tooth may be movable by wedging a sharp explorer into the fissure.[5,9] Later, more evident movement of the parts may be observed. The radiographic presentation at such a late stage may eventually develop into a diffuse radiolucency surrounding the root. At this late stage, narrow isolated deep periodontal pockets may be present.[9] However, such pockets are typically located

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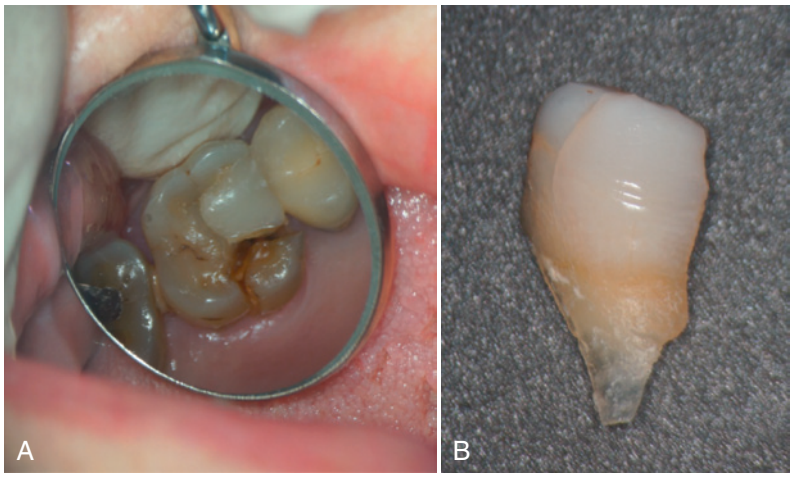

FIG. 21-5 A fractured cusp. A, The mesiopalatal cusp of a maxillary first right molar was fractured. The fractured cusp was movable and was retained by the periodontal ligament. **B,** The cusp was removed, and the tooth was considered restorable. It was treated with root canal treatment and a crown.

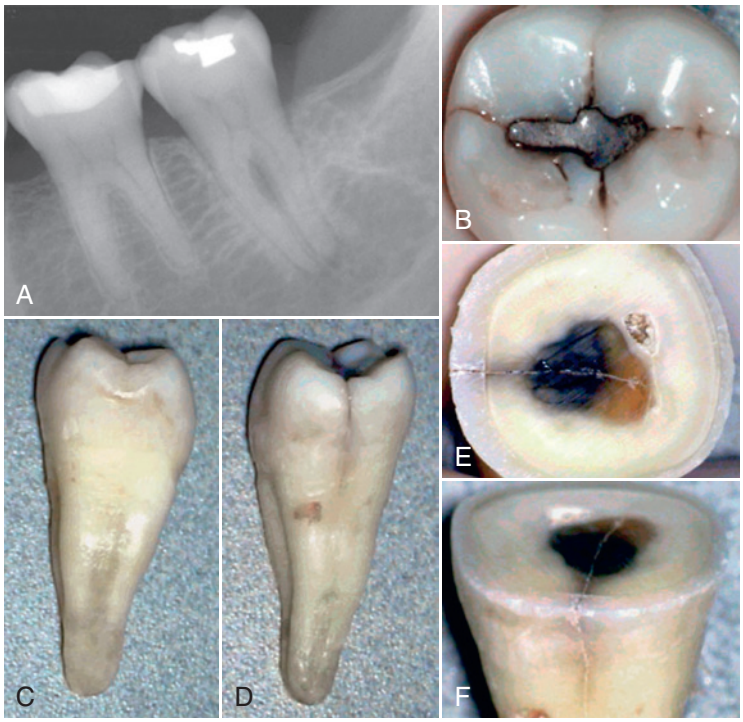

FIG. 21-6 A case of fracture-induced necrosis. A tooth with minimal or no restoration or caries is unlikely to become nonvital. **A,** This radiograph of a mandibular second molar shows a restoration that is distant from the pulp chamber, yet the tooth is nonvital and symptomatic. **B,** On occlusal examination, a slight crack is observed on the distal marginal ridge. **C,** After extraction, the mesial aspect of the crown and root shows no indication of a fracture. **D,** However, the distal aspect of the crown and coronal root shows the fracture. **E, F,** When the crown is sectioned, the crack can be observed to extend well into the pulp chamber.

mesially or distally and, if adjacent teeth are present, they will be difficult, if not impossible, to detect.

Cracked and split teeth may present with a large and variable collection of signs and symptoms that are potentially confusing.[16,18,78] Only by being aware of the *process* leading from early to late manifestations can clinicians interpret these signs and symptoms and identify the specific point they are encountering on the potential timeline of this process.

A definitive combination of factors, signs, and symptoms that, when collectively observed, allows the clinician to conclude the existence of a specific disease state is termed a *syndrome*. However, given the multitude of signs and symptoms that cracked roots can present with, it is often difficult to achieve an objective definitive diagnosis. For this reason, the terminology of *cracked tooth syndrome*[18] should be avoided.

Diagnosis

As in the case of a cracked cusp, early detection is imperative in order to resolve the patient's symptoms as well as increasing the prognosis. However, the use of the Tooth Slooth device (see Fig. 21-2) may or may not provide as clear a result for a symmetrically cracked tooth, as each of the parts of the tooth may be rather stable. Asking the patient to chew on a cotton roll[9] or on the tip of a cotton-tip applicator placed at a particular site may reproduce the pain. Nevertheless this method may not indicate whether the source is a maxillary or mandibular tooth and need further measures to pinpoint the involved tooth. *Magnification* using either loupes or an operating microscope can be helpful for detecting a fracture line. In addition, dyes, such as methylene blue or tincture of iodine, which are applied either to the outer surface of the crown or to the dentin after the removal of an existing intracoronal restoration, can be

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helpful for visualizing the crack (see Fig. 21-3). Transillumination can also be applied to the suspected tooth and, if the tooth has no restorations, this method may yield an impressively straightforward diagnosis (see Fig. 21-3). Anesthetizing the suspected tooth, followed by asking the patient to chew again on the cotton roll, may further confirm the diagnosis and finally differentiate the origin as a mandibular or maxillary tooth. At a later stage, when splitting of the tooth has occurred, wedging of a sharp explorer into the fracture line will provide a clear diagnosis of a split tooth.

Generally speaking, the diagnosis of a crack in a tooth can be difficult. When there are signs and symptoms of a pulpitis or necrosis, it is incumbent upon the clinician to determine the source that initiated the signs and symptoms. In the case of a problematic tooth with no apparent reason for the pulpitis or necrosis, like a tooth with minimal or no caries, restoration, or trauma, a crack or fracture must be considered.[13] In some cases, an objective diagnosis may not be possible; however, with the possibility of a crack or fracture, the patient should be advised of a potential decrease in the endodontic or restorative prognosis.[5,12]

Etiology

Masticatory forces are the cause of cracked teeth.[22,34,45,47,70,71,90] Thus, the dietary habit of chewing on coarse food has been proposed as a contributing factor.[22,90] Bruxism or clenching of the teeth as well as occlusal prematurities are also frequent causes of cracked teeth.[18,22,34,45,47,70,71,90] For this reason, certain teeth may be more prone to developing cracks, such as mandibular second molars and maxillary premolars.[18] Masticatory habits such as chewing ice may also predispose teeth to cracks. The term *fatigue root fracture,* proposed by Yeh,[90] encompasses all of these causes.

In certain cases, traumatic injuries, such as a severe upward blow to the mandible (e.g., during a car or sports accident) can also cause a tooth crack or fracture. Another potential cause is the unexpected chewing of a hard object (e.g., a cherry pit or an unpopped corn kernel in popcorn). The occlusal forces applied by the first molars are as high as 90 kg,[45] which, when fully applied unexpectedly, may damage the tooth structure. However, in most cases, a tooth crack can be attributed to no specific cause other than normal or excessive masticatory forces.[22,34,45,47,70,71,90]

Treatment Planning

Cracked Tooth

When a cracked tooth is either suspected or determined, the patient should be informed that the prognosis is reduced and sometimes questionable.[5,9,12] Protecting a tooth from the propagation of a crack and improving comfort while chewing are the principal goals in the treatment of cracked teeth. Both goals can often be immediately achieved by placing an orthodontic band around the tooth[3,9,16] or by placing a provisional crown. These procedures allow the clinician to evaluate the extent of pulp involvement by checking whether the pulpal symptoms subside in response to the intervention.[3,9,16]

Protecting the tooth from further splitting forces by the placement of a permanent crown is essential in these cases.[3,5,9,16,39] Unfortunately, a crown alone is often not sufficient to resolve symptoms, and endodontic treatment may be considered prior to the placement of the permanent crown, depending on the pulpal symptoms.[9]

A root canal treatment followed by a permanent crown has the benefit of immediately eliminating the long-lasting, painful symptoms as well as early protection from occlusal forces that may cause propagation of a cracked tooth to become a split tooth. Nevertheless, when 127 cracked teeth with reversible pulpitis were treated with a crown alone, 20% of these teeth converted to irreversible pulpitis within 6 months and required root canal treatment.[51] In contrast, none of the other teeth needed a root canal over a 6-year evaluation period.[51] However, it should also be understood that varying percentages of crowned teeth, cracked or noncracked, may also require endodontic treatment, merely from the trauma of the crown preparation. By comparison, it has been shown that when suspected cracked teeth were restored with resin bonded restorations, only 7% required subsequent endodontic treatment or extraction.[64]

After removal of an intracoronal restoration or when penetrating the dentin to prepare for endodontic access, one may observe discoloration along the crack in the dentin. Once the access cavity has been completed, the floor and the distal and mesial walls of the pulp chamber should be carefully inspected to check for the presence and extent of any cracks. Upon evaluating 245 restored teeth, cracks were observed preoperatively in 23.3% of the teeth; however, when the restorations were removed, 60% of these teeth were found to have cracks.[1] Methylene blue dye may be helpful in this type of examination. If a crack is found that reaches from the mesial wall, through the floor of the pulp chamber and into the distal wall, then the prognosis for the tooth is poor, and extraction should be considered (see Fig. 21-6).[5,9,12] If the crack does not reach the pulp chamber or is limited to the coronal parts of the mesial or distal wall, the subsequent protection of the tooth with a crown may save the tooth. Nevertheless, as mentioned previously, the patient should be advised that the treatment success may be compromised and that long-term follow-up will be required.[5,9,12]

Given that such cracks occasionally occur in teeth with minimal or no restorations and that all of the pain may cease once the pulp is removed, it may be tempting to limit the final restoration to an intracoronal amalgam or composite restoration. This temptation should be resisted by all means, as the forces that caused the crack are still present, and the apical propagation of the crack and loss of the tooth is still likely.

Split Tooth

When the tooth is split either through its whole length or diagonally (Fig. 21-7), extraction is typically the only treatment option.[5,9] However, if the fracture line is such that the split results in large and small segments, and if the removal of the small fragment preserves enough tooth structure that is restorable, then retention and restoration of the tooth may be considered.[5]

VERTICAL ROOT FRACTURE (VRF)

Definition

A *vertical root fracture* (VRF) is a longitudinally oriented complete or incomplete fracture initiated in the root at any level and is usually directed buccolingually[5] (Figs. 21-1 and 21-8 through 21-11). By definition, these types of fractures do not arise from the propagation of a fracture that originated in the crown. This definition separates a VRF from a split tooth,

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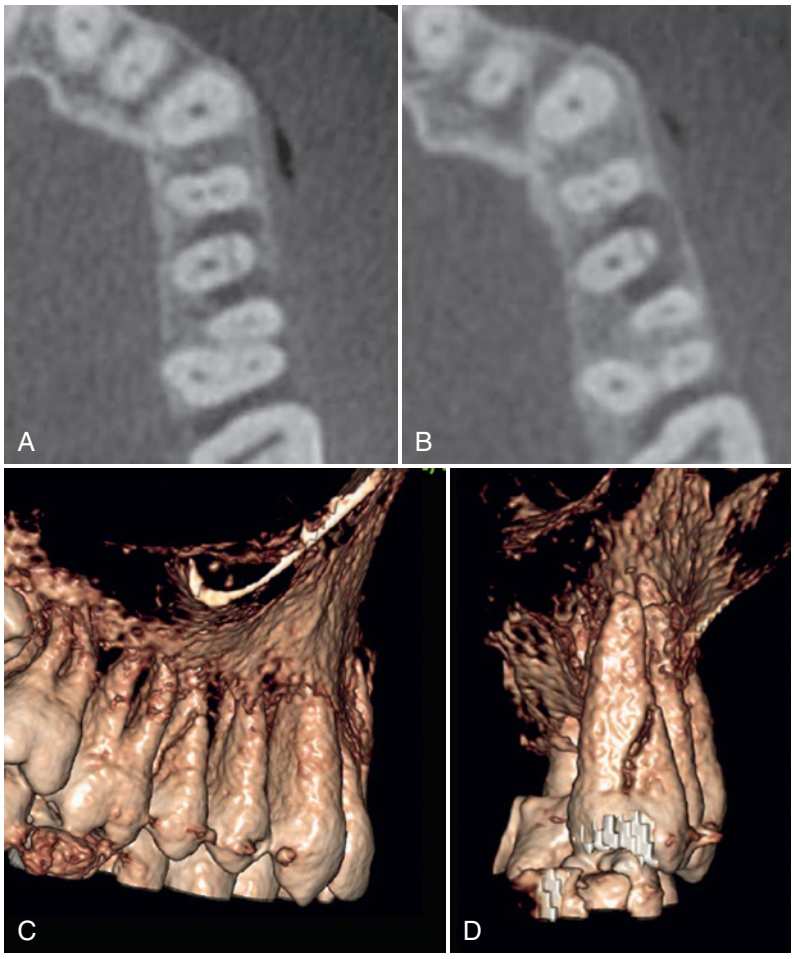

FIG. 21-7 Diagonally split tooth. A maxillary right second premolar presented with a mesiodistal coronal fracture. **A, B,** CBCT axial views revealing a mesiodistal fracture at two different levels, with associated mesial and distal bone loss. The more coronal view has mesial and buccal bone loss **(A)**. A more apical view reveals mesial, distal, and buccal bone loss **(B)**. **C, D,** the three-dimensional reconstruction clearly reveals the nature and direction of the fracture and defined the tooth as unrestorable. Both bone loss and the thick dimensions of the fracture line indicate a longstanding case in which bone resorption allowed opening of the fracture to dimensions that could be detected by CBCT. It is unlikely that such clear demonstration of the fracture line could appear at earlier stages of its formation. (Courtesy Dr. Anda Kfir, Tel Aviv, Israel.)

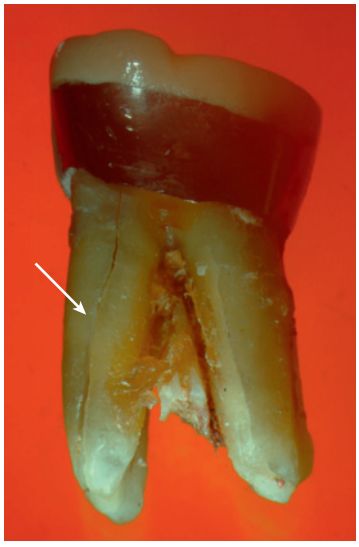

FIG. 21-8 Diagonal VRF in a molar. A lingual view of a right first mandibular molar revealing a diagonal VRF in the mesial root.

which begins with a crack of the crown that propagates apically into the root as a longitudinal fracture. Although both are catastrophic longitudinal factures, a vertical root fracture should be clearly differentiated from a split tooth in that the causes, origins, and typical planes of fracture are substantially different.

Diagnosis

Patient History

In the case of a VRF, a patient may complain of pain or sensitivity related or adjacent to a given tooth. Sensitivity and discomfort while chewing are also common complaints.[80] Swelling may occasionally occur in the area. There is often a long history of failing to diagnose the cause of the pain and discomfort. A history of repeated clinical and radiographic examinations that revealed no cause for the pain is also common in VRF cases. After recent endodontic retreatment, if the symptoms remain and the dentist is unable to determine the cause of the symptoms, the patient may lose confidence in the dentist. Often retreatment or surgical retreatment may have

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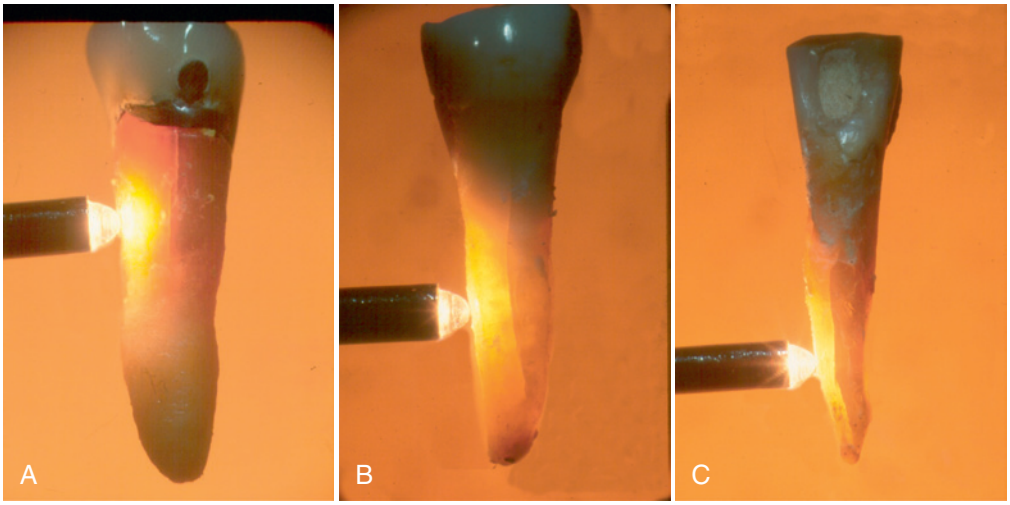

FIG. 21-9 Three types of VRFs. **A,** A coronally located VRF extending apically as far as one third of the root. **B,** A midroot VRF extending along the middle third of the root. **C,** An apically located VRF extending coronally as far as the apical two thirds of the root.

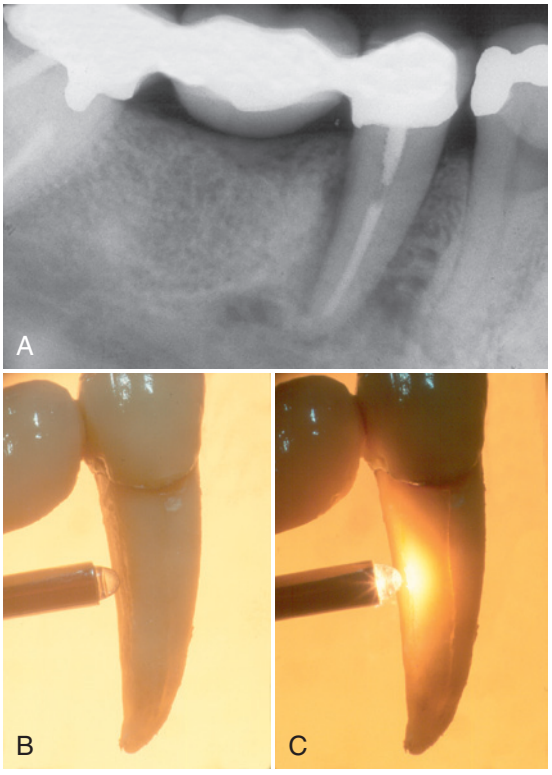

FIG. 21-10 Radiographic presentation of VRF-associated bone loss. A second right mandibular bicuspid was endodontically treated and restored using a short post. Two years later the patient started experiencing discomfort and sensitivity upon chewing. Initially no radiographic signs were present. This lasted for 9 months until a radiograph **(A)** revealed an extensive bone resorption and the tooth extracted. **A,** A large radiolucent lesion was present along the root on the distal side. **B,** The tooth was extracted, but the buccal VRF was not readily evident. **C,** Transillumination with an intense but small light source was used after extraction to clearly demonstrate the VRF. (Courtesy Dr. Ramy Levi, Israel.)

been attempted to reveal the accurate diagnosis.[1] Unfortunately, such ineffective treatment attempts may only worsen the dentist-patient relationship.

Clinical Manifestations

Susceptible Teeth and VRF Location

Vertical root fractures are commonly associated with endodontically treated teeth with or without a post.[5] Nevertheless, VRFs can also occur in teeth with no previous root canal treatment.[22] The most susceptible sites and tooth groups are the maxillary and mandibular premolars, the mesial roots of the mandibular molars, the mesiobuccal roots of the maxillary molars, and the mandibular incisors.[81] However, VRFs may occasionally occur in other teeth and roots as well.

Vertical root fractures may progress in the buccolingual direction in these teeth and roots, which are typically narrow mesiodistally and wide buccolingually.[40] However, VRFs may also propagate diagonally, thus affecting the mesial or distal aspect of the root (see Fig. 21-8). VRFs may be initiated at any root level.[5] They may be initiated at the apical part of the root and propagate coronally (see Fig. 21-9, *C*). Nevertheless, certain VRFs originate at the coronal, cervical part of the root and extend apically (see Fig. 21-9, *A*), and in other cases, a VRF may be initiated as a midroot fracture (see Figs. 21-9, *B*; 21-10, *C*; and 21-11, *D*).

It is commonly believed that VRFs begin as microcracks at the root canal surface of the radicular dentin and gradually propagate outward until the full thickness of the radicular dentin is fractured.[5,10,21,56,87] Studies[14,17,50,76,91] indicate that microcracks can also be initiated at the outer surface of the root and propagate inward. Therefore, the correlation between microcracks in the radicular dentin and the formation of VRFs should be further investigated.

Early Manifestation

In the early stages of a VRF, there may be pain or discomfort on the affected side of the tooth. In particular, the tooth may feel uncomfortable and sensitive upon chewing, although this pain is often of a dull nature, as opposed to the sharp pain typical of

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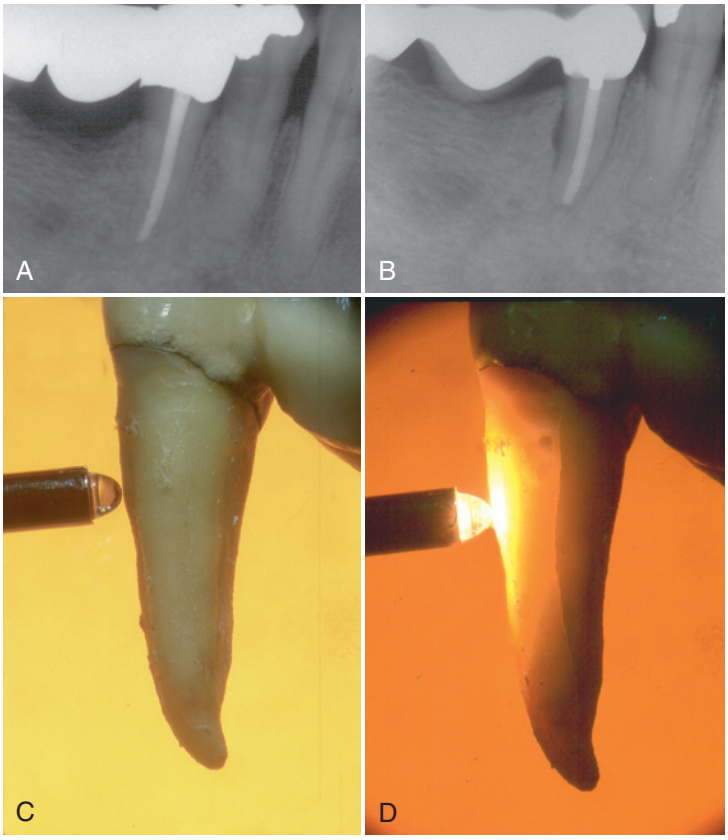

FIG. 21-11 Small isolated radiographic presentation of VRF-associated bone loss. A second right mandibular bicuspid was endodontically treated and restored with no dowel. A year later the patient started complaining about sensitivity on the lingual side. Three months later the radiograph **(A)** was taken and the tooth extracted. A small radiolucent lesion was present along the root on the distal side. The tooth was extracted, but the lingual VRF was not readily evident **(B)**. Transillumination with an intense but small light source was used after extraction to clearly demonstrate the VRF **(C, D)**. (Courtesy Dr. Ramy Levi, Israel.)

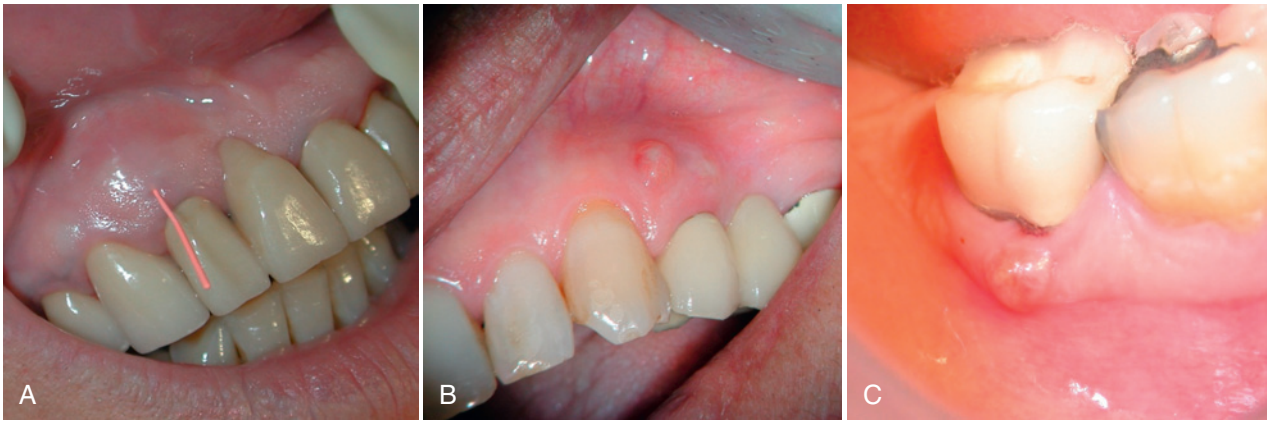

FIG. 21-12 Coronally located sinus tracts. A, A maxillary right lateral incisor with a sinus tract located at the attached gingiva, an uncommon location for sinus tract drainage from a chronic periapical abscess. Maxillary lateral incisors are *not* among the teeth with high prevalence of VRFs. **B,** A draining sinus tract at a coronal location originating from a buccal VRF in the first left maxillary premolar. **C,** A draining sinus tract at the gingival margin of a right first mandibular molar with a buccal VRF in the mesial root. (*A* and *B,* Courtesy Dr. Russ Paul, Zichron Yaakov, Israel.)

a cracked cusp or tooth with a vital pulp. As the fracture and subsequent infection progresses, swelling often occurs, and a sinus tract may be present at a location more coronal than a sinus tract associated with a case of chronic apical abscess[79,81] (Fig. 21-12). These signs and symptoms are frequently similar to those encountered from nonhealing root canal treatment.[57,86] In the early stages, radiographic findings are unlikely because (1) the root canal filling may obstruct the detection of the fracture (Fig. 21-13, *A*), and (2) the bone destruction (which still has limited mesiodistal dimensions) may be obstructed by the superimposed root structure (Fig. 21-14).

A deep, narrow, and isolated periodontal pocket may be associated with the root, which often cannot be explained by, as it is inconsistent with, the surrounding periodontal examination.[79,81,85] This specific type of periodontal defect occurs secondary to the bony dehiscence caused by the vertical root fracture. It is substantially different from the pockets caused by advanced periodontitis (discussed later).

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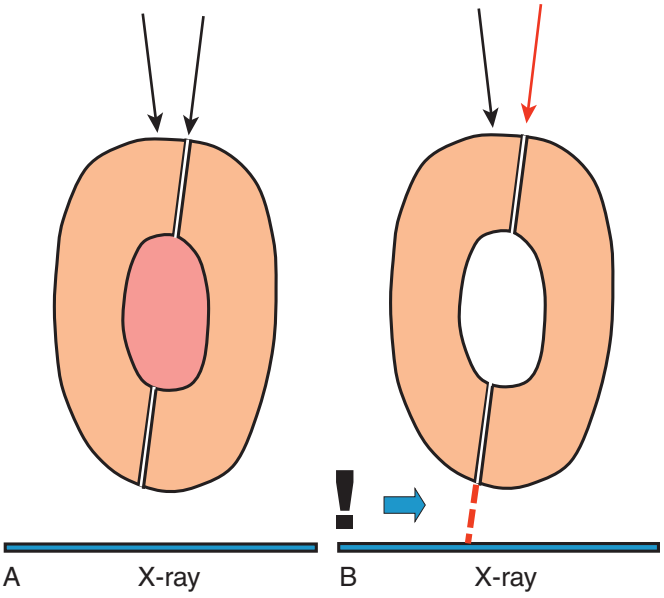

FIG. 21-13 Radiographic examination of filled versus empty canals. **A,** The buccolingual projection of a filled root will fail to detect a VRF at an early stage. **B,** The removal of the root filling and use of radiography at different mesiodistal angulations may reveal the VRF.

Late Manifestation

A longstanding vertical root fracture is easier to detect. The major destruction of the alveolar bone adjacent to the root has already occurred, allowing the VRF to be more likely revealed in a periapical radiograph (see Fig. 21-7). One of the most typical signs is the *J-shaped* or *halo radiolucency*, which is a combination of periapical and periradicular radiolucency (i.e., bone loss apically and along the side of the root, extending coronally)[82,83] (Fig. 21-15). In addition, the pocket along the fracture, which was initially tight and narrow, may become wider and easier to detect. In longstanding cases in which the bone destruction is extensive, the segments of the root may also separate, resulting in a radiograph that clearly reveals an objective root fracture (Fig. 21-16).

Diagnosis

Importance of Early Diagnosis

Accurate and timely diagnosis is crucial in VRF cases, allowing the extraction of the tooth or root before extensive damage to the alveolar bone can occur. Early diagnosis is particularly important when implants are a potential part of the future restorative procedure; when an extraction is performed at an early stage, the uncomplicated placement of an implant is likely. When the tooth is extracted after extensive damage has already occurred (see Fig. 21-14), bone regeneration procedures may be required,[42] adding cost and time to the restoration procedure.

The American Association of Endodontists stated in 2008[5] that a sinus tract and a narrow, isolated periodontal probing defect associated with a tooth that has undergone a root canal treatment, with or without post placement, can be considered pathognomonic for the presence of a VRF.

However, the combination of the following two factors makes the early diagnosis difficult: (1) many of the clinical symptoms associated with VRFs mimic apical periodontitis or periodontal disease, and (2) the narrow and tight pocket associated with early stages of VRF is difficult to detect using rigid probes (see the VRF Pocket section). Consequently, a delay in the accurate diagnosis or a misdiagnosis of a VRF may often occur.

Misdiagnosis of VRFs

Certain cases of longstanding VRFs are so discernible that no dentist can miss the diagnosis (see Fig. 21-16). Nevertheless, two retrospective case series, one by Fuss and colleagues[32] and the other by Chan and associates,[22] reported that general practitioners often misdiagnose VRFs. The teeth that were extracted in these studies had often been diagnosed as endodontic failures or refractive periodontal pockets, only to realize after extraction that in some of them the actual cause was a VRF.

VRF Pockets

The pockets that are typical of the early stages of VRFs differ substantially from the deep pockets associated with advanced periodontal disease. The deep pockets associated with periodontal disease develop as a result of the bacterial biofilm that initially accumulates at the cervical areas of the tooth and the destructive host response to these bacteria.[38] Therefore, deep periodontal pockets are typically wider coronally and relatively loose. This pocket structure allows the easy insertion of rigid periodontal probes (Fig. 21-17). These types of periodontal pockets typically present with the deeper part of a pocket at the mesial or distal aspects of the tooth. Periodontal disease often affects groups of teeth rather than an isolated location of a single tooth.

The pockets associated with VRFs develop due to bacterial penetration into the fracture, triggering a destructive host response that occurs in the periodontal ligament along the entire length of the fracture. These bacteria may leak from an infected root canal[87]; however, when the VRF extends to the cervically exposed root, the microbes in the fracture may also originate from the oral cavity. In the early stages, the periodontal ligament is affected and destroyed along the longitudinal opening of the fracture, initially with a limited resorption to the adjacent bone. This permits the penetration of a periodontal probe. The pocket associated with a VRF is typically isolated and present only in a limited area adjacent to the affected tooth. This pocket is often located at the buccal or lingual convexity of the tooth. In the early stages, the pocket is deep but has a narrow coronal opening (see Fig. 21-17). The insertion of a probe first requires the detection of the coronal opening; often, light pressure is necessary for the insertion of the probe. Because the pocket is narrow, probe insertion may result in the blanching of the surrounding tissue (Fig. 21-18). This is specially the case when a plastic probe is used, as its coronal part is thicker than an equivalent metal probe (Figs. 21-18 and 21-19).

The pocket associated with the early stages of a VRF is quite different from a common periodontal pocket. This difference has been widely recognized, and terms such as *osseous defect*[28] and *probing defect*[5] have been used to emphasize the point. Nevertheless, these pockets do possess enough unique features to justify them being specifically termed a *VRF pocket*.

Rigid metal periodontal probes may be ineffective in probing VRF pockets in the early stages of a VRF. Given that the pocket is deep, narrow, and tight, the bulge of the tooth's crown may prevent the insertion of a metal probe into the pocket (see Fig. 21-19). A flexible probe should be used instead, such as a probe available from Premier Dental Products (Plymouth

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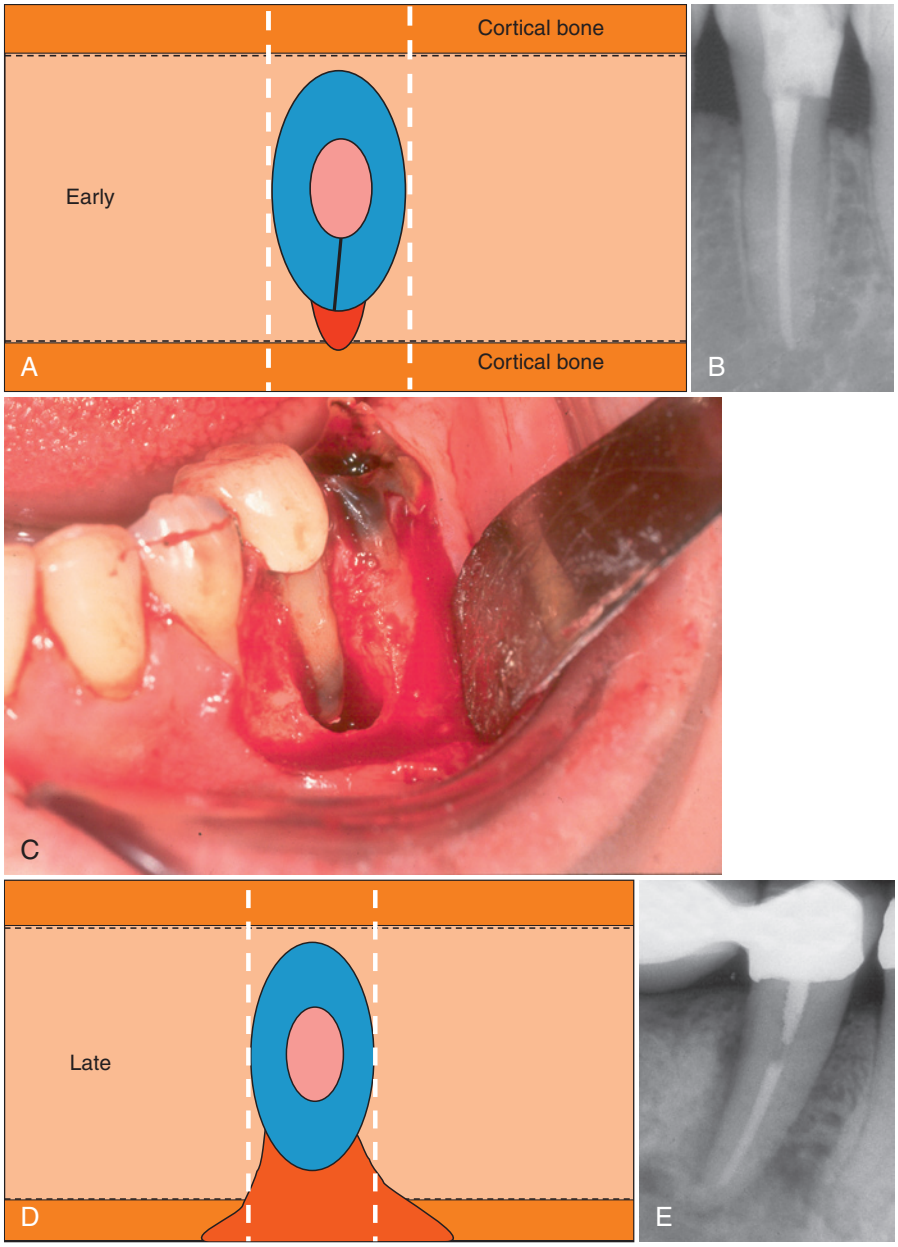

FIG. 21-14 Early versus late radiographic presentation of a VRF-associated bone defect. At an early stage, a bone defect *(red)* is not likely to be detected in a periapical radiograph, as the root will overlap with the defect **(A, B).** At later stages, when major damage has occurred to the cortical plate **(C),** the bone defect may be large enough to extend beyond the silhouette of the root **(C, D)** and appear as a radiolucent defect along the root **(E).** (Surgical image courtesy Dr. Devora Schwartz-Arad, Ramat-Hasharon, Israel.)

Meeting, Pennsylvania) (see Fig. 21-19) or a similar device. This type of flexible probe should be included on every endodontic examination tray and is an essential tool when checking for potential VRF pockets.

A typical VRF pocket was observed in 67% of the VRF cases reported by Tamse and colleagues.[81] However, because the early detection of such pockets is technique sensitive and because traditional metal probes were used in the aforementioned study, the incidence of these pockets may in fact be higher than reported. When a typical VRF pocket is located on the convex flank of the root on the buccal or lingual side, it is likely that the root has a VRF. In contrast, when such a pocket is located at the furcation of a molar, the pocket may indicate either a VRF or a sinus tract from an apical abscess that found a point of least resistance at the furcation area. In cases with a furcation pocket, when a VRF diagnosis cannot be conclusively determined, a positive healing response to the elimination of infection by initiating root canal retreatment may differentiate between these two types of pathoses.

Coronally Located Sinus Tract

Sinus tracts that originate from a chronic apical abscess are typically detected at the site of least bone resistance, against the apical part of the root or in the area of the junction of the

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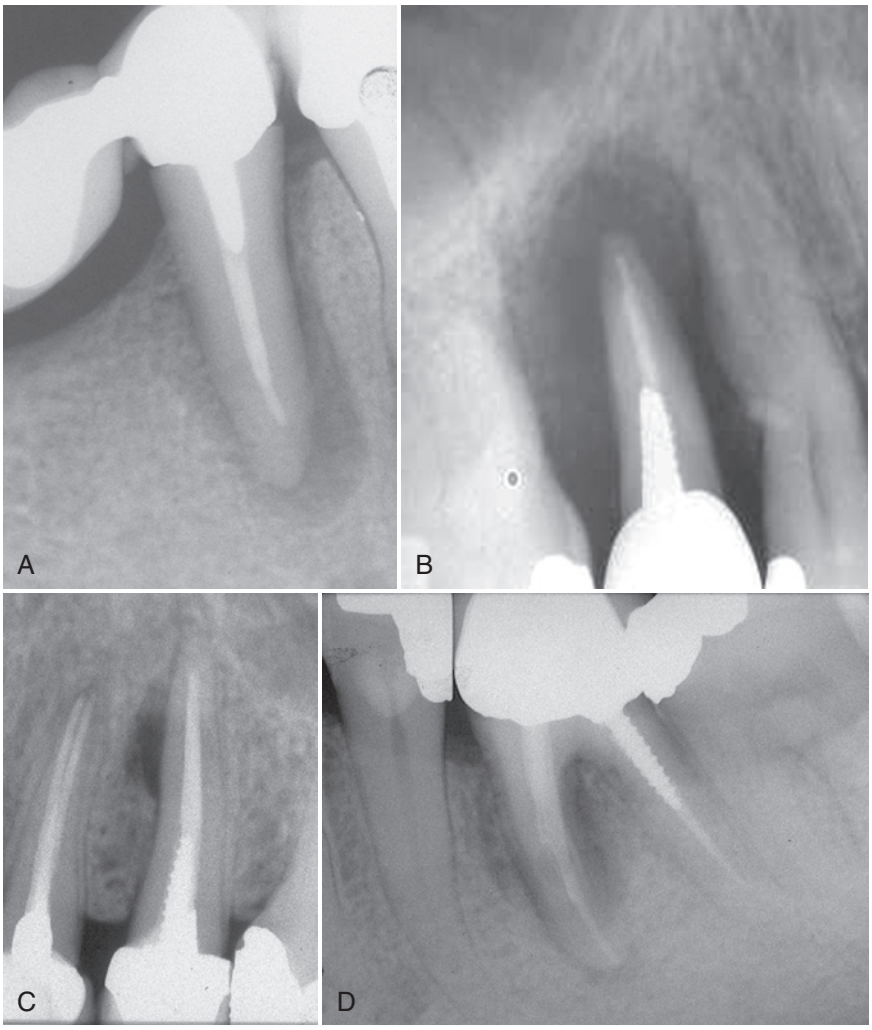

FIG. 21-15 Radiographic presentations of longstanding VRFs. **A**, J-shaped "halo" associated with a VRF in a second right mandibular premolar. **B**, Extensive bone damage associated with a complete VRF in a second right maxillary premolar. **C**, Limited bone damage associated with a midroot VRF in a second left maxillary premolar. **D**, Bone damage associated with a VRF in the mesial root of a first left mandibular molar. All such presentations are typical of longstanding VRFs.

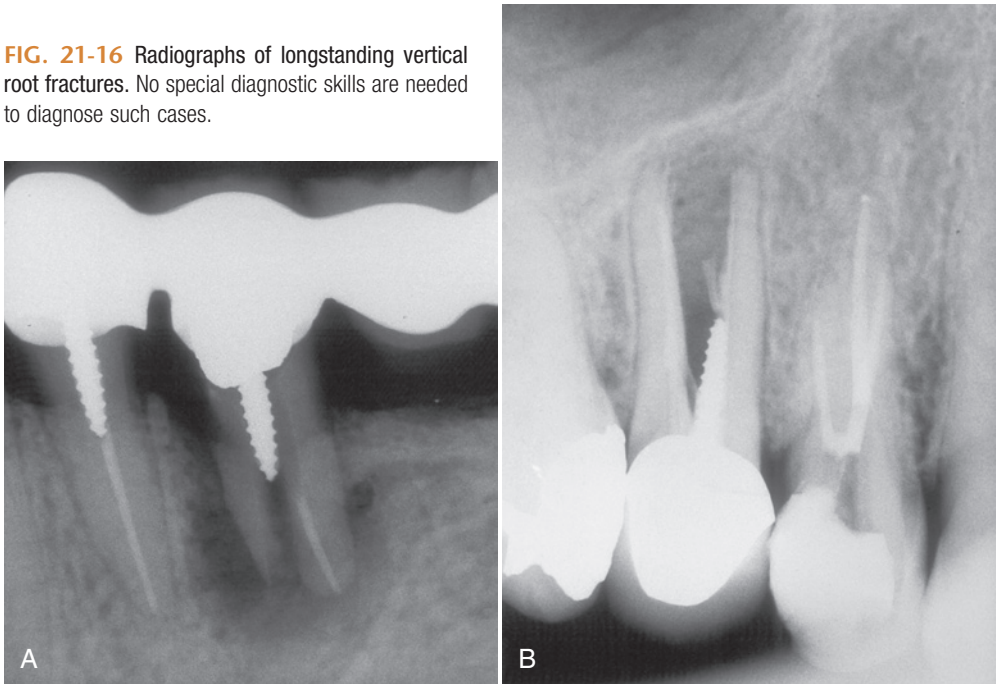

FIG. 21-16 Radiographs of longstanding vertical root fractures. No special diagnostic skills are needed to diagnose such cases.

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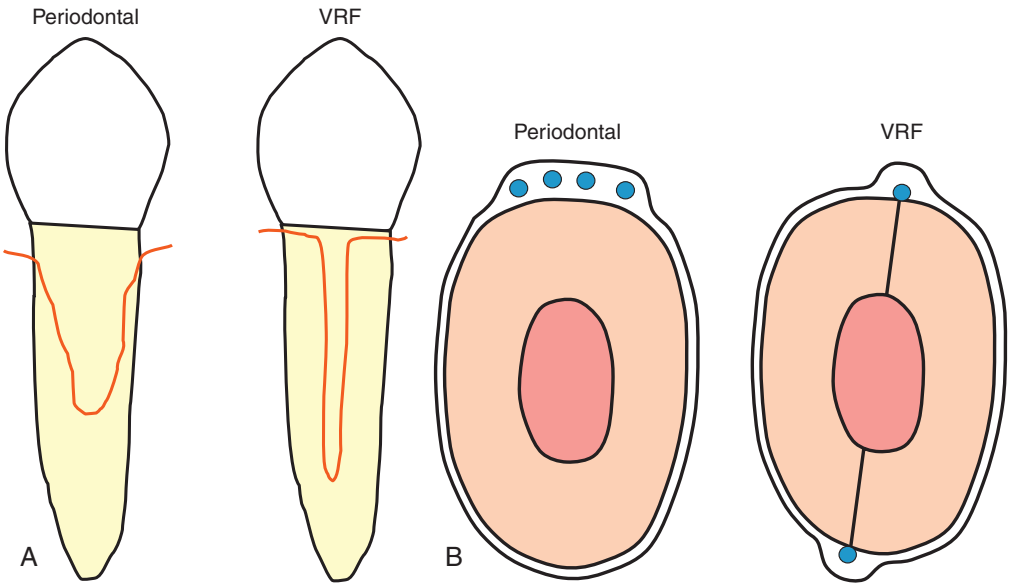

FIG. 21-17 VRF pocket. A, Periodontal pockets *(left)* are wide coronally, whereas VRF pockets *(right)* are narrow and deep. **B,** Periodontal pockets *(left)* are loose and allow probing at various sites, whereas VRF pockets *(right)* are narrow and tight. If not checked carefully at every millimeter of the sulcus, an early VRF pocket can easily be missed. Please note that periodontal pockets appear more commonly in the proximal sides of the root, whereas VRF pockets are more common on the buccal or lingual sides.

attached gingiva and the oral mucosa. Sinus tracts that are associated with a VRF pocket are often found in a more coronal position, as the source is not from a periapical lesion[80] (see Fig. 21-12).

In four clinical retrospective case series, coronally located sinus tracts were found in 13% to 35% of these cases.[58,79,81,85] As in the case of a VRF pocket, if the sinus tract is located at the furcation of a molar, this observation does not necessarily indicate a VRF, as periapical abscesses from a failing root canal treatment can also drain in this coronal location.

Radiographic Features

A definitive VRF diagnosis can sometimes be made based on the radiographic appearance of a thin radiolucent line extending longitudinally down the root.[72] Such lines, however, are difficult to detect and are commonly not seen in routine orthoradial, periapical radiographs because either the root canal filling has "masked" the fracture line or the angulation of the radiograph is not optimal for discerning the fracture (see Fig. 21-13) (Figs. 21-20 and 21-21). Rud and Omnell[72] claimed that it was possible to observe fracture lines in 35.7% of cases, but many of these cases were not true VRF instances. In clinical practice, it is still rare to observe a VRF on a radiograph, especially when only a single periapical radiograph is taken. Such an observation requires the x-ray beam to align with the plane of the fracture as well as the fracture line not being superimposed over the radiopaque root filling (Fig. 21-20, *A*). Therefore, two or three periapical radiographs should be exposed from different horizontal angulations when a fracture is suspected[80] (see Figs. 21-21 and 21-22).

In most VRF cases, the clinician must make *interpretations* or *predictions* based on the various patterns of periradicular bone destruction, which, unfortunately, are also shared by other periodontal and endodontic-like lesions.[58,75,88]

In the early stages of a VRF, no radiolucent bone lesions may be observed[20] (see Fig. 21-14), which may be the reason why VRFs often remain undetected, delaying diagnosis and treatment. Rud and Omnell[72] correlated the direction of the fracture, the degree of bone destruction, and the radiographic appearance, and emphasized that the extent of bone destruction around a fractured root depends on the location of the root fracture and the time elapsed since the inception of the fracture. The significance of time was confirmed by Meister and colleagues,[58] who demonstrated that immediate radiographic detection is difficult due to the time required for bone resorption to occur or for the fractured segments to separate and be radiographically visible. In a study of the patterns of bone resorption in 110 VRF cases, Lustig and associates[57] found that in 72% of patients with either chronic signs and symptoms (i.e., pertaining to a sinus tract, osseous defect, or mobility) or acute exacerbations, there was greater bone loss recorded compared to patients for whom a VRF diagnosis was made at an early stage.[57]

Despite the difficulty of diagnosing *early stage* VRFs in endodontically treated teeth, there are often several radiographic signs associated with *later stages* that are strong indicators of VRF.

The J-shaped or halo appearance, a combination of periapical and periradicular radiolucencies, was associated with a high probability of a VRF in a double-blind radiographic study involving 102 endodontically treated maxillary premolars[82] (see Fig. 21-15). An angular resorption of the crestal bone along the root on one or both sides, without the involvement of the periapical area, mimicking a "periodontal radiolucency" (see Fig. 21-15), was found in 14% of the cases. Tamse and coworkers[83] also reported the radiographic appearance of "halo" (see Fig. 21-10) and "periodontal" radiolucencies (see Fig. 21-10) in vertically fractured mesial roots of

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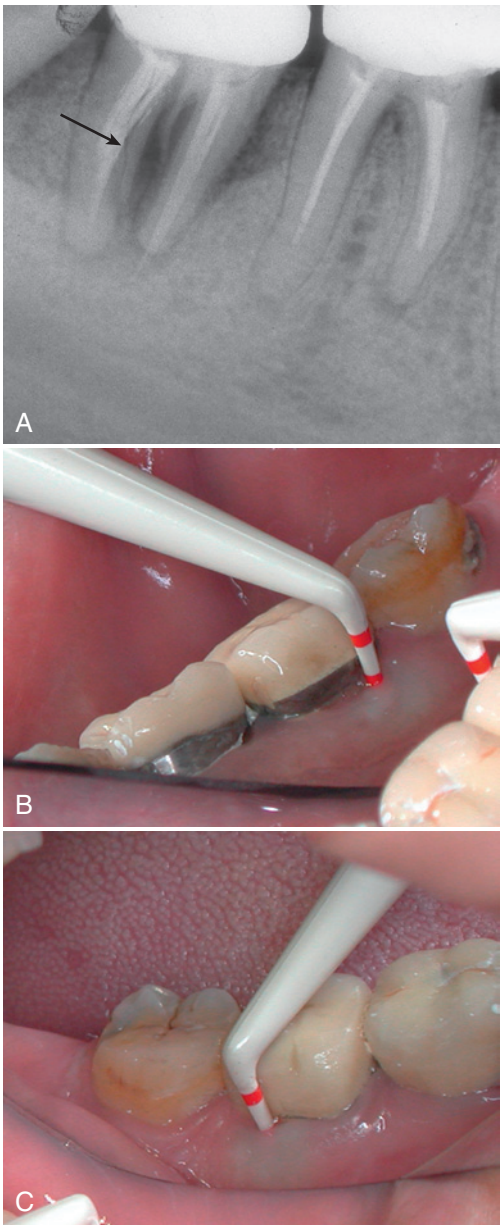

FIG. 21-18 **Tight VRF pockets. A,** The distal root of a second right mandibular molar with a VRF. VRF pockets were found on both the lingual **(B)** and the buccal **(C)** sides. VRF pockets are tight, so inserting a probe into these pockets causes pressure blanching of the surrounding tissues.

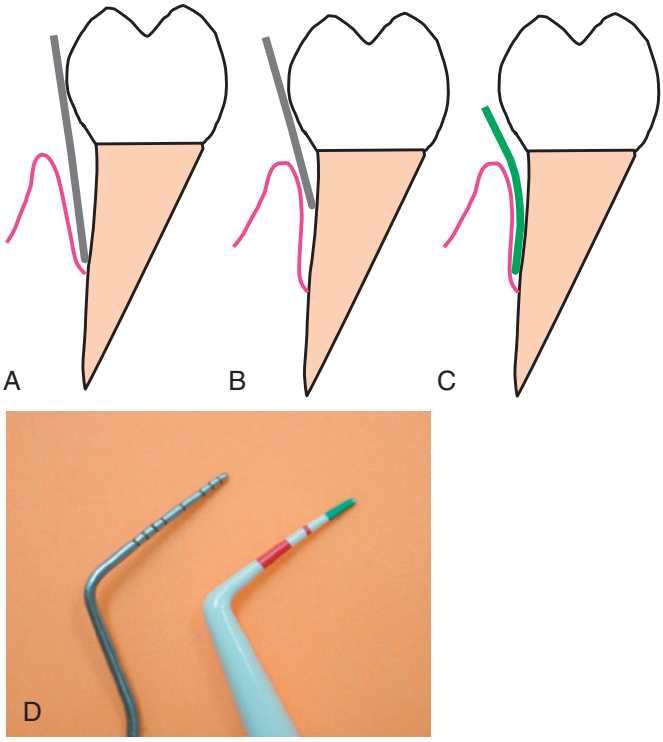

FIG. 21-19 **Rigid versus flexible probes. A,** In a loose periodontal pocket, a rigid metal probe can easily reach the depth of the pocket. **B,** In a tight, early-stage VRF pocket, a rigid probe may be of limited value, as the bulge of the crown often prevents the insertion of the probe into the tight, deep pocket. **C,** A flexible probe **(D)** is more likely to detect VRF pockets at an early stage.

mandibular molars (37% and 29%, respectively). In that study, the use of these two variables, combined with bifurcation involvement (63%) and the presence of an amalgam dowel (67%), predicted fracture in 78% of the cases. Others have reported similar findings.[24,62] Despite the different sample sizes, study designs, and objectives, the most common radiographic feature in these studies was a lateral radiolucency appearing longitudinally along the root and a halo appearance.

Radiolucency in the Bone Along Root

The type of periradicular radiolucency associated with a vertical root fracture is not and should not be interpreted as a thickening of the PDL. Instead, it represents a substantial destruction of the cortical plate of the alveolar bone[57] (see Fig. 21-14). In the case of a VRF in the buccolingual plane, often the bone resorption is limited at early stages, and any associated radiolucency may be obscured by the superimposition of the root (see Fig. 21-14). As the bone loss increases, the radiolucency becomes greater than the dimensions of the root, allowing it to be detected more clearly in the above-mentioned manner (see Fig. 21-14). As the VRF progresses to an intermediate stage, radiographs taken at different horizontal angulations may detect bone resorption (see Fig. 21-22), whereas a conventional orthoradial radiograph may not (see Figs. 21-14 and 21-22). This radiographic feature should be differentiated from a split tooth, in which the fracture plane is typically mesiodistal, with the bone resorption occurring in the earlier stages on the mesial or distal aspects of the root.

Radiograph of Empty Canal

As mentioned previously, the direct clinical detection of an early stage VRF from a periapical radiograph is unlikely, especially when there has been endodontic treatment. Because most VRFs are in the buccolingual plane, the radiopaque obturation often obstructs the view of the hairline radiolucency of the fracture (see Figs. 21-13 and 21-20). When a VRF is suspected, one may initiate root canal retreatment, removing the root obturation, and taking radiographs at two or three different horizontal angulations. The detection of a hairline radiolucency may provide a more definitive diagnosis of a VRF[80] (see Figs. 21-20 and 21-21).

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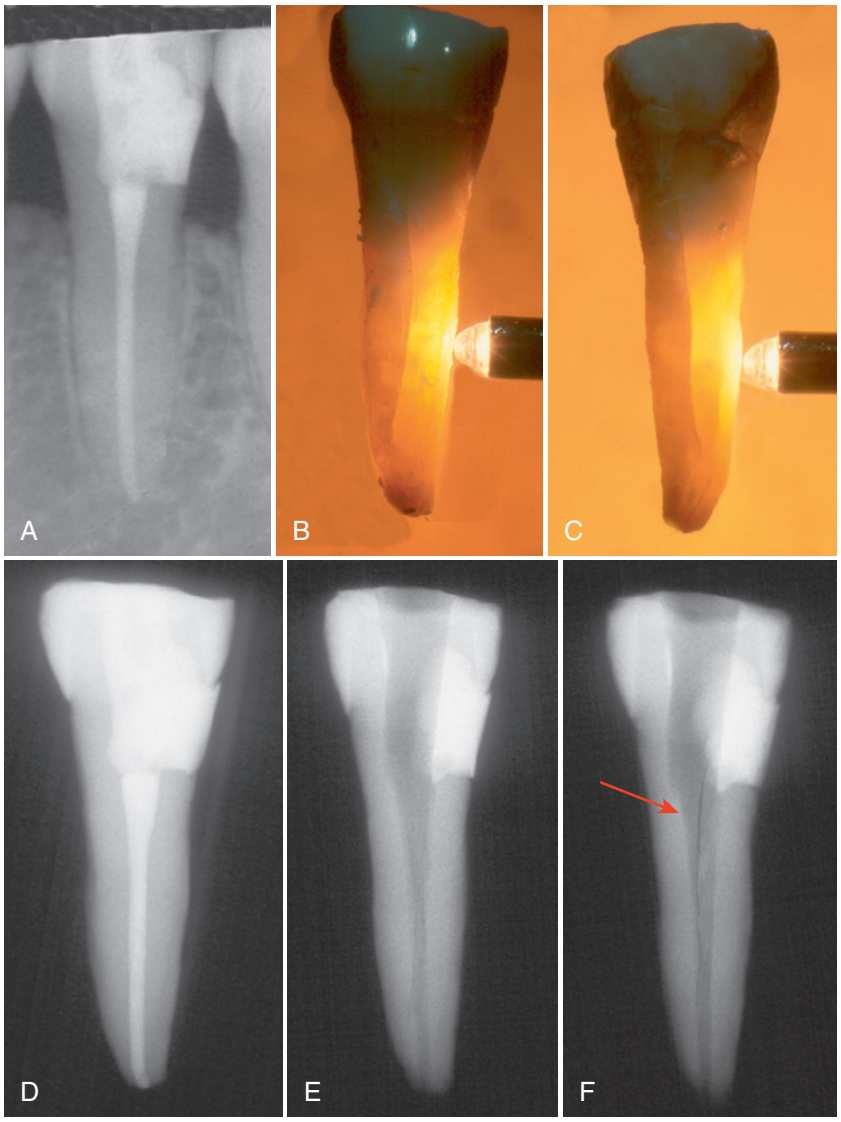

FIG. 21-20 Radiography of an ex vivo empty canal. The second left mandibular bicuspid underwent a root canal treatment, with no post required. Thirteen months later, the patient complained about pain and sensitivity to palpation on the lingual side. A radiograph provided no further information **(A)**. An isolated, 7-mm pocket was detected on the lingual side, and the tooth was extracted. Midroot VRFs were visible on the buccal **(B)** and lingual sides **(C)**. An ex vivo radiograph of the same tooth with no findings **(D)**. The root filling was removed, and radiographs were taken from different mesiodistal angulations **(E, F)**. One of the radiographs clearly revealed the VRF **(F)**.

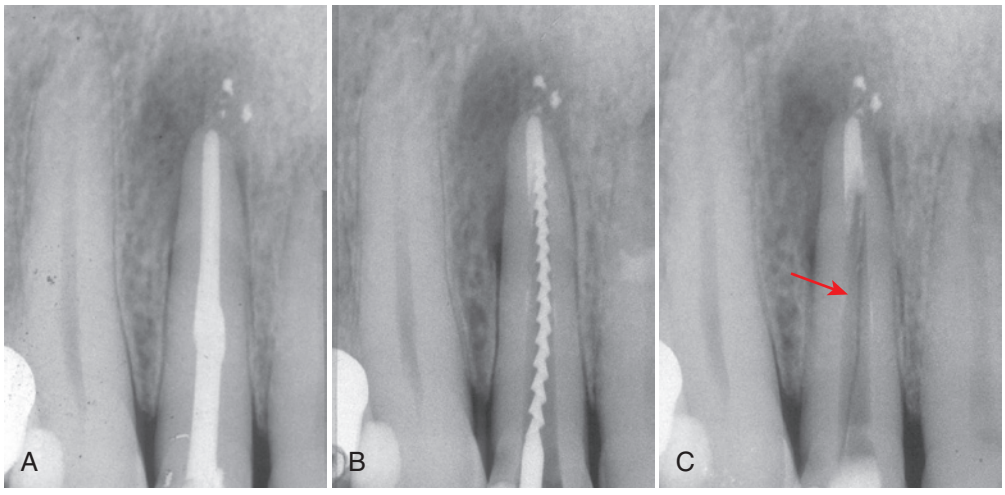

FIG. 21-21 Radiography of an empty canal: a clinical case. The right maxillary lateral incisor underwent a root canal treatment a few years ago. The patient complained about occasional pain on the palatal side. The tooth was sensitive to percussion and palpation on the palatal side. Radiography revealed a periapical radiolucency **(A)**. An isolated, deep, and narrow pocket was found on the palatal side of the root. Both the patient and the referring dentist were reluctant to extract the tooth, assuming that the pocket could potentially be a sinus tract, and decided on retreatment. A radiograph taken with a file during the process **(B)** could have missed essential information that was obscured by the file, which was an evident VRF **(C)**.

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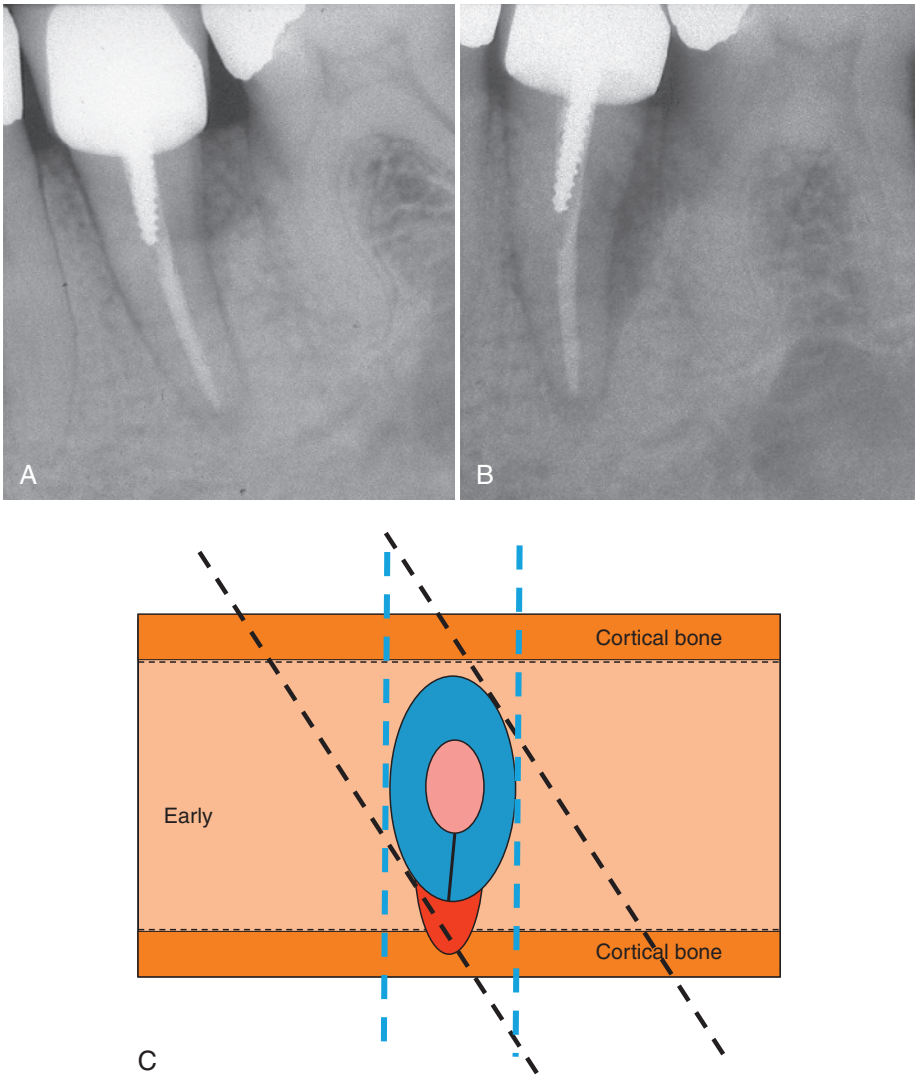

FIG. 21-22 Diagonal radiographs for detection of VRF. A, Orthoradial radiograph: very limited bone loss is seen. **B,** A different horizontal angulation reveals a radiolucent lesion along the root. **C,** Schematic presentation. Although a radiograph in an orthoradial direction *(blue lines)* cannot pick up the radiolucency along the root, diagonal angulation *(black lines)* may do so, as in **B.**

Cone-Beam Computed Tomography in VRF Diagnosis

Modern cone-beam computed tomography (CBCT) has a much smaller radiation dose compared to traditional medical spiral CT imaging, thus rendering CBCT a reasonable diagnostic tool for the use in selected endodontic cases.[6,31,31a,65]

One of the unique features of CBCT is its ability to study the suspected tooth and associated bone in an axial plane. Axial views may provide detailed information regarding the cross-sectional appearance of the tooth and its surrounding bone (Fig. 21-23). With the CBCT devices currently available, the width of an unseparated fracture may be too small and undetectable (see Fig. 21-23) (SEDENTEXCT guidelines[31]). Traditional planar, periapical radiographs are also of limited value for the *early* detection of VRFs. More specifically, bone damage or separation of the fragments is only radiographically evident at a relatively late stage. Several studies suggested that the detection of early-stage VRFs by a CBCT scan set to an axial view may be possible.[41,42,59] Yet such detection may greatly depend on the resolution of the machine (i.e., the voxel size). At a voxel size of 0.3 mm, the detection of early, unseparated VRFs is not reliable; however, when smaller voxel sizes were used in these in vitro studies, the reliability greatly increased.[41,42,59] Although the detection level of a fracture is thought to be twice the voxel size of CBCT imaging, there is presently no literature available to support this theory. Therefore, given that the smallest voxel size currently available for a CBCT device is about 0.075 mm, CBCT imaging would not be able to visualize a root fracture unless the fracture width was greater than 0.15 mm. It should also be noted that the intracanal presence of gutta-percha or a metal post often causes artifacts that make it extremely difficult to differentiate a VRF from such artificial lines.[59]

Although early VRFs may still be below the detection level of many CBCT machines, the *early destruction of the bone* along the suspected fracture may be visible in the cancellous bone (i.e., with an axial view) at relatively early stages, whereas this early bone destruction would not be detectable in traditional planar, periapical radiographs; such bone resorption may help to establish a VRF diagnosis (see Fig. 21-23).

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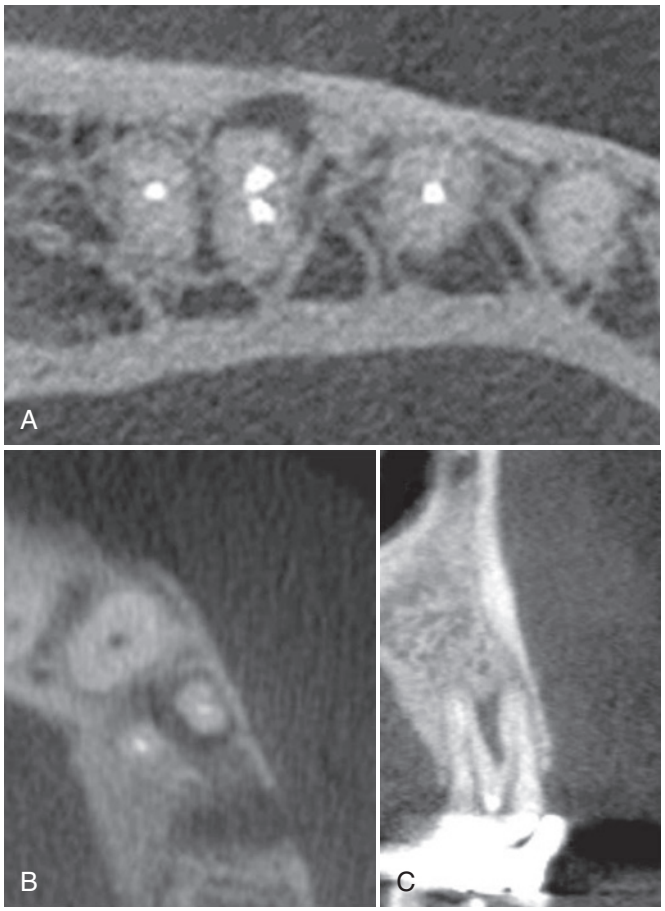

FIG. 21-23 "Invisible" bone destruction associated with early VRFs. **A,** Buccal VRF was present in the mesial root of a first left mandibular molar. At this early stage a typical VRF pocket was evident, yet no associated bone destruction was observed in a periapical radiograph. The VRF-associated bone destruction was evident in an axial plane of a CBCT. **B,** A VRF was present in the palatal side of the buccal root of the first maxillary premolar. A draining sinus tract was present with no radiographic signs to explain it. **C,** CBCT revealed a radiolucent lesion on the palatal side of the buccal root, which was verified, after extraction, as caused by VRF. Please note that in neither of these cases could the actual VRF fracture line be seen in the CBCT scan. (*B,* Courtesy Dr. Anda Kfir, Tel Aviv.)

With likely increased resolution in the near future, CBCT may become an important diagnostic tool for the detection of VRFs. For the present, neither the most updated Joint Position Statement of the American Association of Endodontists and American Academy of Oral and Maxillofacial Radiology (2010), nor the European Society of Endodontology position statement on the use of CBCT in endodontics (2014) recommend the use of CBCT for diagnosis of VRF.[6,31a]

Improvements of CBCT imaging—such as achieving a better signal-to-noise ratio, obtaining a smaller voxel size, and by applying advanced algorithms to segment fracture lines—may promise the potential to enhance the ability to detect early-stage VRFs in the future.

Exploratory Surgery

When clinical and radiographic evaluations are equivocal in detecting a suspected vertical root fracture, exploratory surgery may be indicated. When a full-thickness flap is raised and the granulation tissue is removed, a VRF may often be directly visualized[80] (see Figs. 21-14 and 21-24). The bone resorption pattern associated with a VRF is mostly seen as a bony dehiscence, with the greater bone destruction being present on the buccal cortical plate located over the offending root. In a small percentage of the cases, fenestration can also be seen.[57] Furthermore, it has been shown that the longer a VRF-related infection persists, the greater the resulting periradicular bone destruction.[57]

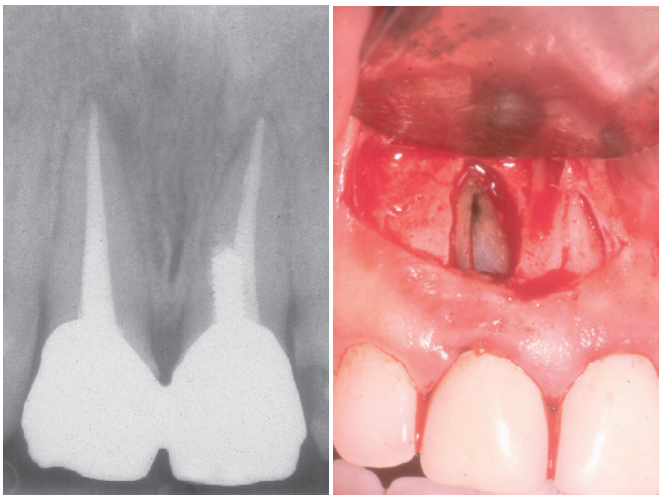

FIG. 21-24 An apical VRF with no pockets. The right maxillary central incisor was symptomatic despite an apparently adequate obturation. A radiolucent area was present around the apex, but no pockets were detected. Apical surgery was attempted, only to reveal a VRF at the apical part of the root that did not (yet) extend into the gingival margin and thus did not present with a typical VRF pocket. A semilunar flap design was used in this case, which was intended for apicoectomy. If surgical exploration for suspected VRF was initially intended, a conventional full flap would have been used. (Courtesy Dr. Ram Zeev, Rehovot, Israel.)

Etiology

Vertical root fractures may arise from a series of factors, some of which are natural whereas others are iatrogenic, arising from dental procedures such as endodontic treatment and the restorative procedures that follow it. The most common dental procedure contributing to vertical root fractures is endodontic treatment.[11]

Most vertical root fractures occur in endodontically treated teeth.[11,24] VRFs usually do not occur during the actual obturation of the root canal, but rather they occur long after the procedure has been completed.[81]

The etiology of VRFs is multifactorial.[33,79] It is likely that in the presence of one or an accumulation of more predisposing factors, the repeated functional or parafunctional occlusal loads may eventually lead, over months or even years, to the development of a VRF. Predisposing factors may include natural ones, such as the anatomy of the root, or iatrogenic ones, such as the excessive forces during root canal instrumentation, excessive tooth structure removal, or excessive obturation pressure.

Natural Predisposing Factors

Shape of Root Cross Section

One of the common anatomic features shared by teeth that typically develop VRFs is an oval cross section of the root, with

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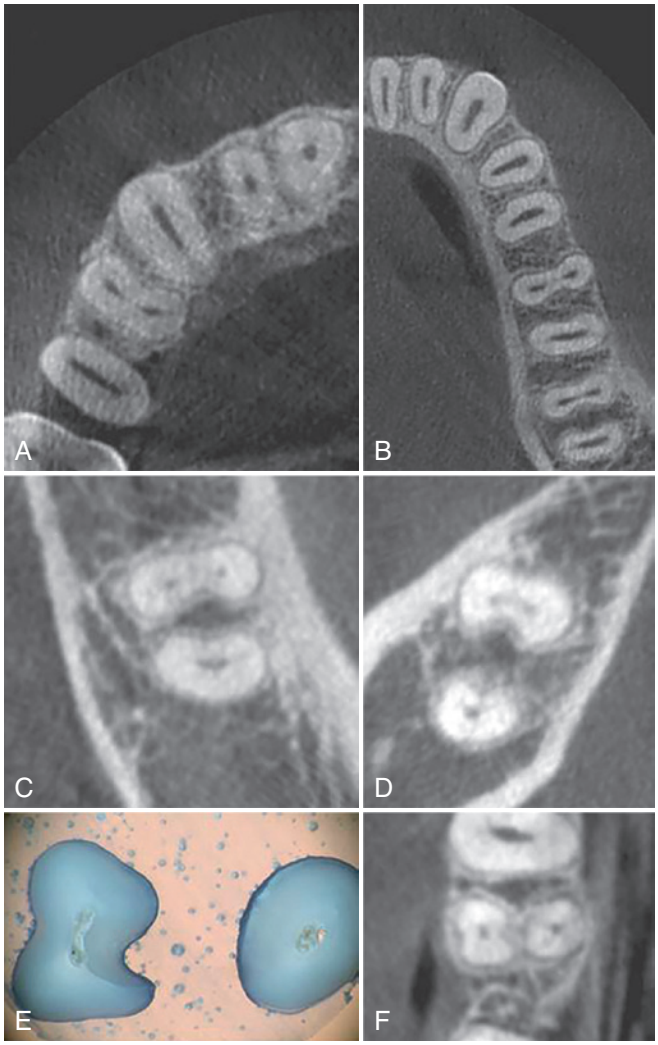

FIG. 21-25 Anatomic predisposing factors. A, An axial view of CBCT scan of a maxilla, revealing oval canals in the maxillary canine and second premolar. **B,** An axial view of a mandible, revealing oval canals in incisors, canine, premolars, and distal roots of the mandibular molars. Oval anatomy combined with endodontic treatment has been associated with a higher incidence of VRFs. **C, D,** Concavities on the distal aspect of the mesial roots of mandibular molar may establish a "danger zone" in which excessive instrumentation, combined with straightening of the canal, may result in a thinner dentin wall that may allow strain concentration. **E, F,** Concavities in the palatal side of the buccal root of maxillary first premolar (*E,* sections; *F,* axial view from a CBCT). These depressions may also represent a potential danger zone. Neither the concavity in **C** and **D** nor the concavity in **E** and **F** would be evident in a planar periapical radiograph. It should be noted that CBCT scans should not be used for routine screening but should be limited to the indications delineated in the joint statement of the American Association of Endodontists (AAE) and the American Academy of Oral and Maxillofacial Radiology (AAOMR).[5,6]

a buccolingual diameter being larger than the mesiodistal diameter.[36,40] These teeth include the maxillary and mandibular premolars, the mesial roots of the mandibular molars, and the mandibular incisors (Fig. 21-25, *A* and *B*). Such anatomy is easily observed in the axial plane of a CBCT scan (see Fig. 21-25, *A* and *B*). The fracture in these teeth typically starts in the buccolingual plane, specifically at the highest convexity of the oval root[21,80] (see Fig 21-25, *A* and *B*). This conclusion,

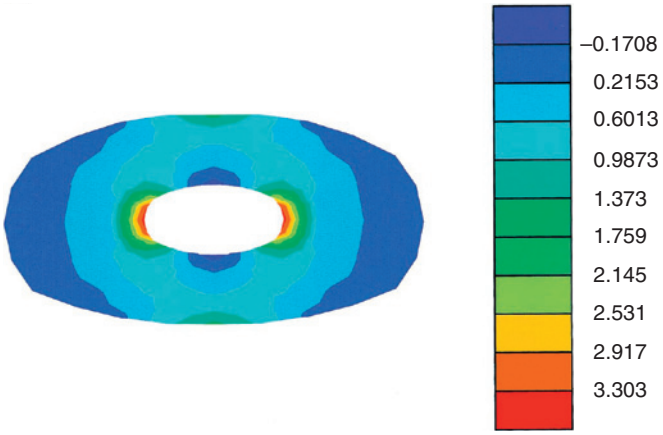

FIG. 21-26 Finite element analysis of the strain distribution in an oval root. Note the strain concentration on the inner side of the highest convexity of the remaining dentin wall. Red and orange represent areas of higher strains than blue areas. (From Lertchirakarn V, Palamara J, Messer HH: Patterns of vertical fractures: factors affecting stress distribution in the root canal, *J Endod* 29:523, 2003.)

derived from large case series, is also supported by finite element analysis.[64] Such analysis clearly demonstrated strain concentration on the inner side of the remaining dentin wall at the highest convexity point (i.e., the buccal and lingual sides of the oval roots)[54,55] (Fig. 21-26).

Occlusal Factors

Excessive occlusal loads or concentration of such loads may be another natural predisposing factor. Load concentrations, such as those caused by occlusal prematurities in maxillary premolars, and excessive occlusal forces, specifically in the case of mandibular second molars, are examples.[18] In combination with other natural and iatrogenic predisposing factors, excessive occlusal loads may, over time, lead to VRFs.

Preexisting Microcracks

Preexisting microcracks may be present in the radicular dentin, likely resulting from repeated forces of mastication or occlusal parafunction.[15,63] Such fractures were also recently reported by Barreto and colleagues,[10] who have found these microcracks in 40% of intact maxillary incisors and canines.

Iatrogenic Predisposing Factors

Root Canal Treatment

VRFs mostly appear in endodontically treated teeth[5,80]; therefore, endodontic treatment per se may be considered an iatrogenic predisposing factor. Teeth were once thought to be more susceptible to fracturing after endodontic treatment because of a decrease in hydration.[43] However, later studies found no difference in the properties of dentin, as a material, after endodontic procedures.[46,75]

Although the physical characteristics of the dentin, as a material, may not be compromised by endodontic treatment, the radicular dentin, as a structure, may be compromised by the accumulative or combined effect of several natural or iatrogenic factors associated with the endodontic treatment and the restoration of endodontically treated teeth. This may be the reason for the often-reported association of VRF with endodontically treated teeth.

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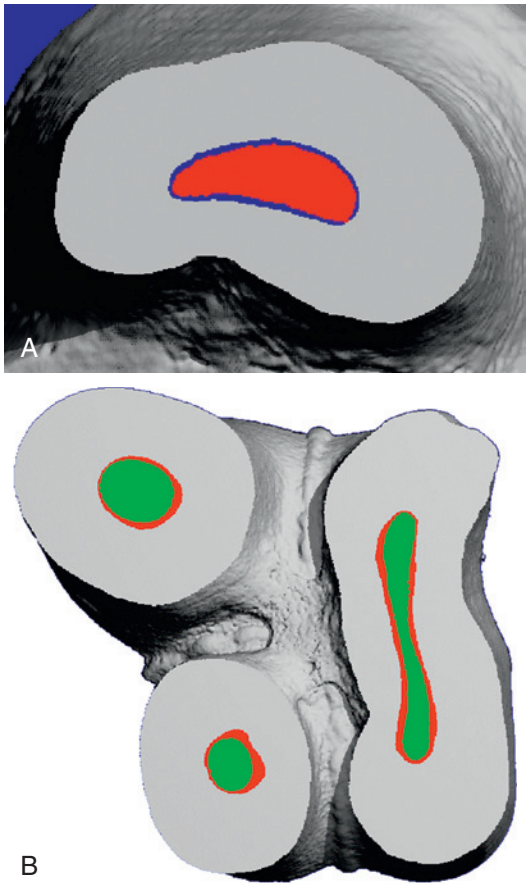

FIG. 21-27 Minimally invasive endodontic preparation. In this preparation, a uniform layer of dentin was removed from all around the root canal wall using a self-adjusting file, as opposed to shaping the canal into a circular cross section using rotary endodontic files. (*A,* From Metzger Z, Teperovich E, Zary R, et al: The self-adjusting file (SAF). Part 1: respecting the root canal anatomy—a new concept of endodontic files and its implementation, *J Endod* 36:679, 2010. *B,* From Solomonov M: Eight months of clinical experience with the self-adjusting file system, *J Endod* 37:881, 2011. Image from ongoing study by Solomonov and Paque F.)

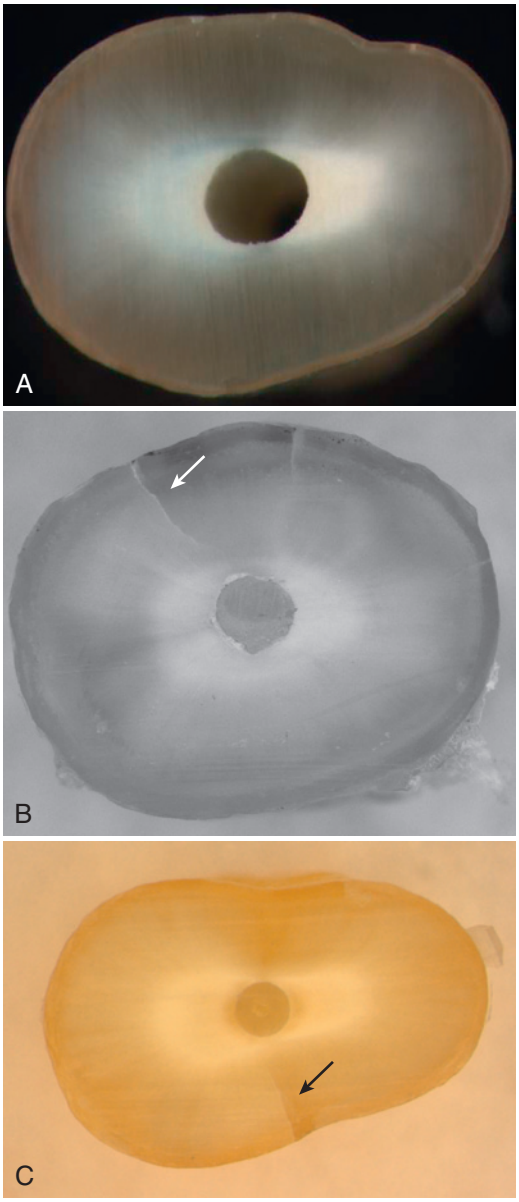

FIG. 21-28 Microcracks generated in single-root teeth by rotary nickel-titanium files. A, Control: hand instrumentation, generating no microcracks. **B, C,** Rotary instrumentation: microcracks generated in the remaining radicular dentin *(arrows).* (Courtesy Dr. H. Shemesh, Amsterdam, Holland.)

It is incumbent for the clinician to recognize this effect and take efforts to minimize any steps that may contribute to the development of a root fracture during endodontic treatment.

Excessive Root Canal Preparation

Excessive root canal preparation may be a predisposing factor for VRF development.[89] In one study, cracks detected by transillumination were more frequent when the same teeth were subjected to a gradually increasing endodontic canal preparation.[89] To reduce the risk of VRFs, less invasive methods may be considered, such as minimally invasive endodontic instrumentation[60,66] (Fig. 21-27; see also Chapter 6).

Microcracks Caused by Rotary Instrumentation

Shemesh and colleagues[76] and others[2,10,14,17,44,77,91] observed that root canal preparation using nickel-titanium rotary and reciprocating files often results in microcracks in the remaining radicular dentin (Fig. 21-28). This finding, which was originally noted for single-root teeth, has been further supported by Yoldas and coworkers,[91] who studied microcrack formation from rotary files in the mesial roots of mandibular molars. Each of the rotary file systems examined in this study caused frequent microcracks in the dentin, whereas both hand instrumentation with files and the self-adjusting file (see Chapter 6) did not cause such cracks (Fig. 21-29).

A finite element analysis by Kim and colleagues[50] supports and may explain these findings. These researchers reported that rotary files induce strain on the dentin, as measured in the surface layers of the root dentin, which likely exceeds the elasticity of the dentin, causing subsequent microcracks, as also reported by Shemesh, Bier, Adorno, Yoldas, and Bürklein, along with their research associates as well as by others.[2,10,14,17,44,76,77,91] Additional stress, by either root obturation with lateral compaction[10,76] or by retreatment[77] that was applied to roots that were previously instrumented with rotary files, caused some of the microcracks to propagate and become

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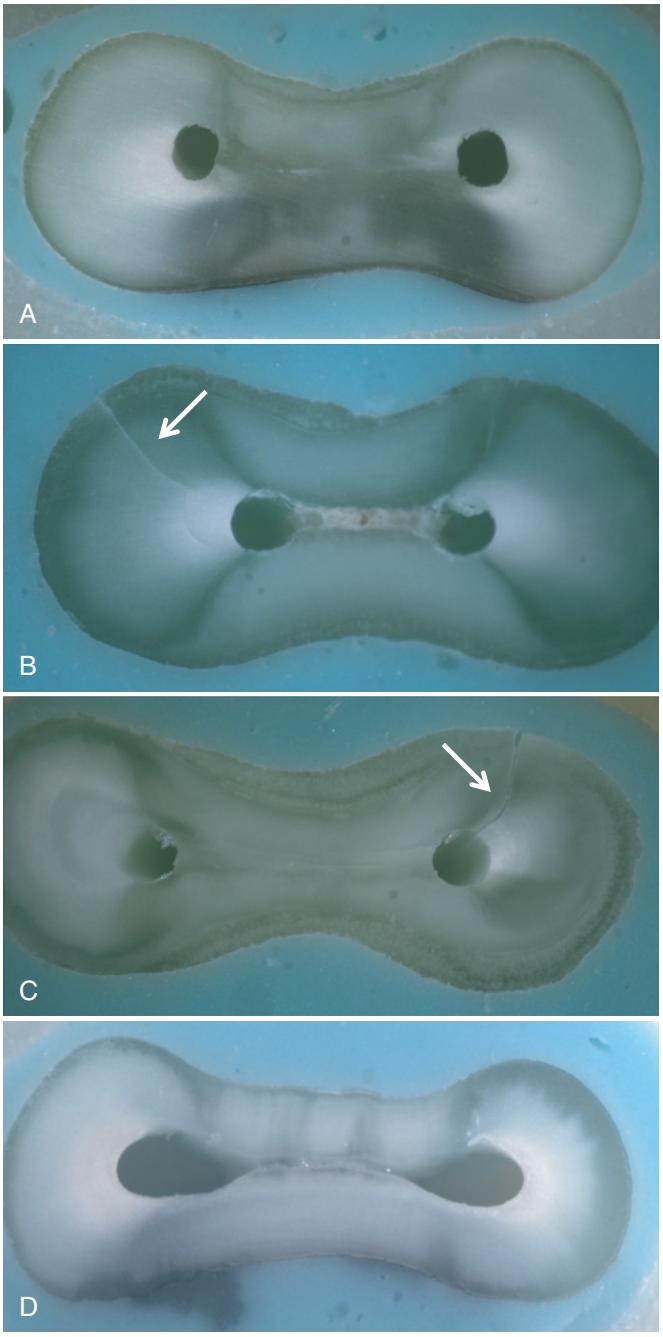

FIG. 21-29 **Microcracks generated in mesial roots of mandibular molars by rotary nickel-titanium files. A,** Control: hand instrumentation, generating no microcracks. Rotary instrumentation: microcracks generated in the remaining radicular dentin *(arrows).* **B,** A partial-thickness crack. **C,** A full-thickness fracture. **D,** Self-adjusting file instrumentation: no microcracks generated. (From Yoldas O, Yilmaz S, Atakan G, et al: Dentinal microcrack formation during root canal preparation by different NiTi rotary instruments and the self-adjusting file, *J Endod* 38:232, 2012.)

through-and-through fractures that were indistinguishable from VRFs.[10,76,77]

The relationship of these various findings to the subsequent clinical creation of a VRF has yet to be confirmed. In any event, considering the basic principles of fracture mechanics, the creation of microcracks should at least be considered as a potential predisposing factor.

Uneven Thickness of Remaining Dentin

The instrumentation of root canals often results in uneven thickness of the remaining dentin, particularly when curved canals are straightened by instrumentation.[67] Uneven dentin thickness can also occur upon excessive instrumentation of the mesial roots of the mandibular molars or first maxillary premolars, which may exhibit a distal or mesial concavity, respectively, that is not detectable in a common planar, periapical radiograph (Fig. 21-25, *C* and *D*). These areas, which have been referred to as "danger zones,"[30] may be characterized by a decrease in the remaining dentin thickness in which the application of internal strain may potentially lead to a fracture. The anatomic groove that is often found on the palatal side of the buccal root of maxillary bifurcated premolars is another example of such a hidden danger zone[48] (Fig. 21-25, *E* and *F*).

Lingual access, which is commonly used in incisors, may also result in a thinner buccal wall in the apical area as compared with the lingual wall. This phenomenon may be especially pronounced when thick, and thus rigid, instruments are used excessively. When lateral compaction was applied ex vivo in a similar case, the strain concentration was recorded on the thinner buccal side of the apical part of the tooth (Figs. 21-30 and 21-31).

The use of flexible nickel-titanium files and minimally invasive instrumentation with instruments such as the self-adjusting file may reduce such risks (see also Fig. 21-27 and Chapter 6).

Methods of Obturation

Certain obturation techniques, such as lateral compaction, involve the application of internal pressure with a spreader, which may cause strains[27,73] and subsequent propagation of microcracks into fractures across the full dentin thickness.[10,76] Other obturation methods may create less pressure, such as thermoplasticized gutta-percha, and may reduce the risk of VRFs (see Chapter 7).

Type of Spreader Used

The use of a more rigid and thick stainless-steel hand spreader may lead to increased strain in the radicular dentin and can result in an increased incidence of root fracture.[27,73] The introduction of more flexible finger spreaders, which have smaller diameter, may greatly reduce such risks.[27] Among the finger spreaders, devices composed of nickel-titanium allow insertion with less force than stainless-steel finger spreaders.[74] The nickel-titanium spreaders also allow a further reduction in the strain induced in the radicular dentin during obturation compared to traditional stainless-steel finger spreaders[68] (Fig. 21-32).

Post Design

Post selection, design, and seating have a significant effect on the strain distribution in the root. Excessively long or thick posts are considered a predisposing factor for VRFs.[23,25,61] The use of posts carries an inherent risk of root fracture, particularly if excessive sound dentin is removed during preparation. Posts should only be used when essential for core retention and should be avoided whenever a sufficient coronal tooth structure is available for the secure retention of the crown.[4,37]

Crown Design

When considering endodontically treated teeth, crowns with a ferrule margin (i.e., supported by a sound tooth structure all

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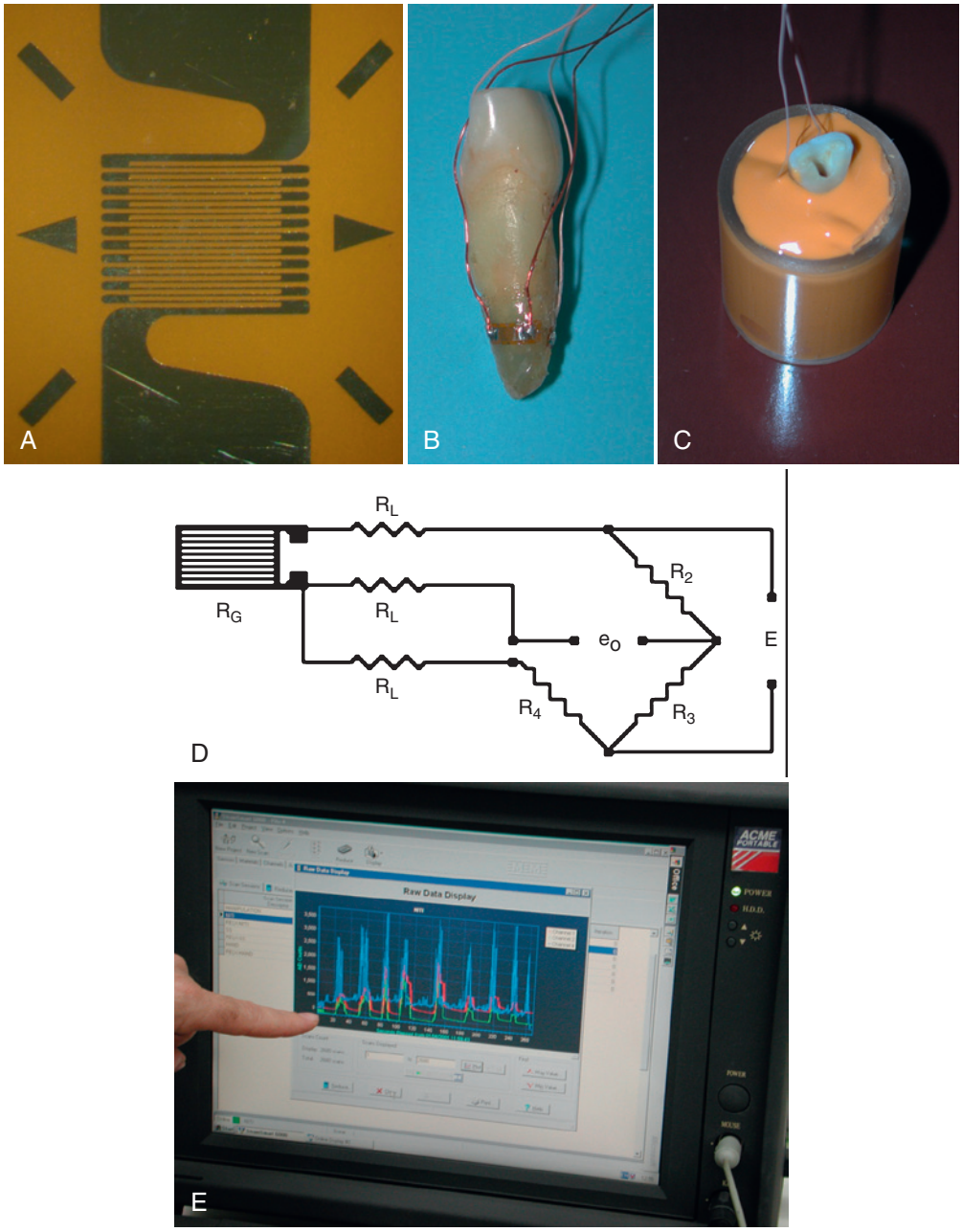

FIG. 21-30 Strain measurement setup. Microstrain gauges (**A**, greatly magnified) are attached to the surface of the root in the area(s) of interest (**B**). The tooth is embedded in a cylinder composed of flexible dental-impression material (**C**), and the strain gauge or gauges are wired with a 3/4 Wheatstone bridge circuit (**D**) and connected to a data collection system (**E**). The continuous registration of the force applied to the spreader and the strain that develops in a given area of the root allows for the analysis presented in Figs. 21-31 and 21-32. (From Pilo R: Development of strains and mechanical failure in dental roots undergoing root canal obturation and prosthetic rehabilitation. PhD thesis, Tel Aviv University, 2007, supervised by Zvi Metzger and T. Brosh.)

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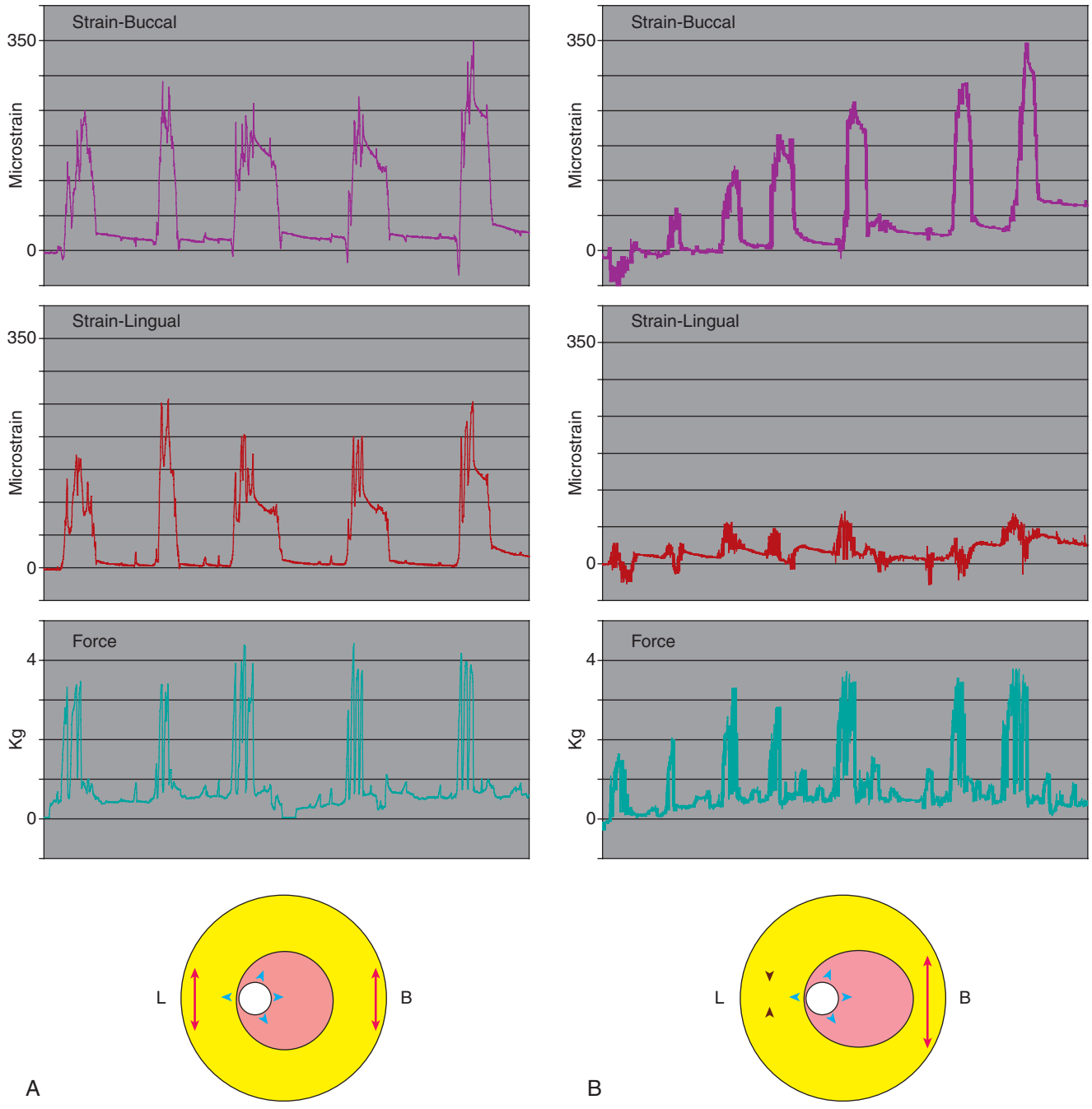

FIG. 21-31 Strain during lateral compaction: even versus uneven wall thickness. A pattern of the strain that develops in the buccal and lingual dentin of the apical area of a maxillary central incisor during lateral compaction. **A,** The thicknesses of the remaining buccal and lingual dentin were similar (even). Each manual insertion of the spreader was registered as a force peak. The tension strain was recorded for each insertion and was *similar* on the buccal and lingual sides. **B,** The thickness of the remaining dentin was lower on the buccal side than on the lingual side (uneven) due to the lingual access and rigidity of the instruments used. Each manual insertion of the spreader was registered as a force peak. The tension strain was recorded for each insertion on the buccal side, whereas compression was recorded on the lingual side. (From Pilo R: Development of strains and mechanical failure in dental roots undergoing root canal obturation and prosthetic rehabilitation. PhD thesis, Tel Aviv University, 2007, supervised by Zvi Metzger and T. Brosh.)

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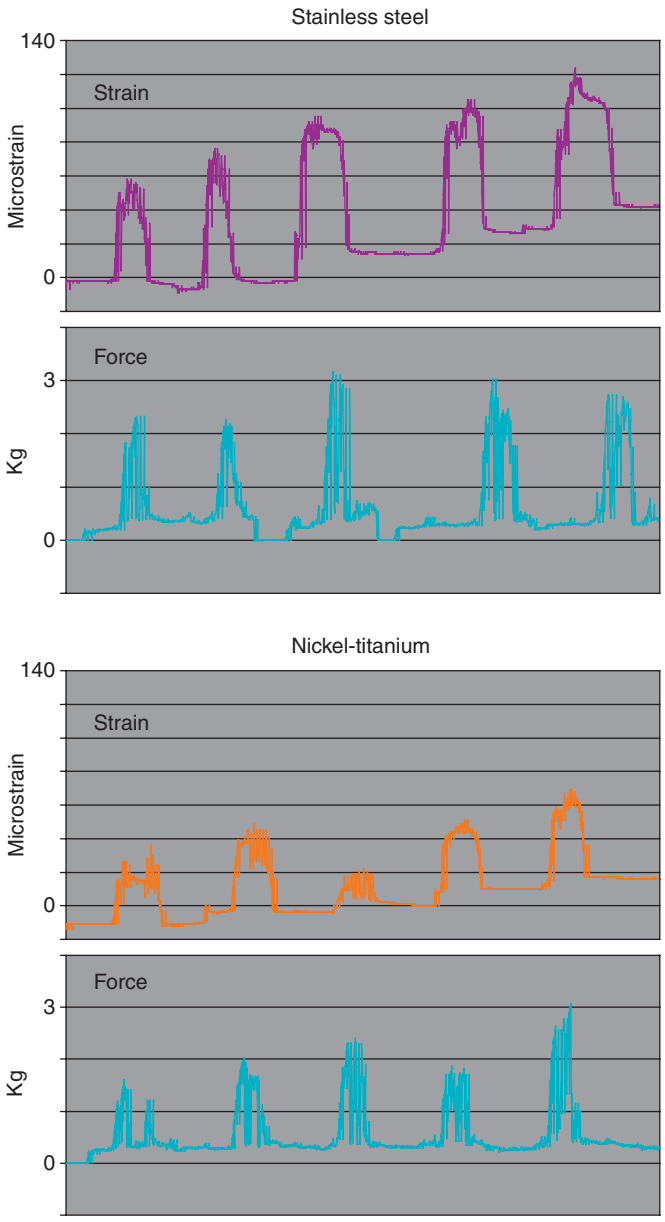

FIG. 21-32 Strain caused by stainless-steel versus nickel-titanium finger spreaders. The force applied to the finger spreader during lateral compaction caused strain on the buccal side of the apical part of the maxillary central incisors. Upper panels: the force and strain registered with a stainless-steel finger spreader. Lower panels: the root filling was removed, and another cycle of lateral compaction was completed for the same tooth using a nickel-titanium finger spreader. The spreader dimensions and depth of insertion were similar in both cycles. The nickel-titanium finger spreader required less force and generated lower strains than the stainless-steel finger spreader. (From Pilo R: Development of strains and mechanical failure in dental roots undergoing root canal obturation and prosthetic rehabilitation. PhD thesis, Tel Aviv University, 2007, supervised by Zvi Metzger and T. Brosh.)

around and beyond the gingival margins of the core) provide better strain distribution than similar restorations that are supported by the post and core alone.[4] This design may help to avoid yet another potential predisposing factor for VRFs.[26]

Conclusions

In conclusion, even though endodontic treatment may be necessary in many occasions, the impact of each of the iatrogenic predisposing factors should be minimized as much as possible, as they are likely to have a cumulative deleterious effect.

TREATMENT PLANNING

Prevention is the key to managing vertical root fractures. There are many predisposing factors and iatrogenic causes of these fractures, all of which should be minimized as much as clinically possible. VRFs may be present in teeth subjected to retreatment but are rarely present also in teeth that have never undergone endodontic treatment.[22] Therefore, comprehensive clinical, radiographic, and *periodontal examination* is imperative when evaluating any tooth that is planned for endodontic treatment or retreatment. A flexible periodontal probe is mandatory in such examinations.

When a VRF is determined to be present, extraction of the affected tooth or root is recommended as soon as possible. Any delay may increase the potential for additional periradicular bone loss and possibly compromise the placement of an endosseous implant. Thus the measures and means that may allow the dentist to make the diagnosis at early stages are important. Attempts to "repair" a fracture by filling the crevice with a variety of restorative materials have been reported; however, none of these repairs is considered a reliable long-term solution.[8,49,53,84]

SUMMARY

Because of the wide variety of different types of cracks and fractures in teeth, there may be a myriad of symptoms and presentations that may appear at different stages of the crack or fracture development, making their diagnosis often difficult. The awareness that many of these symptoms represent *stages in an evolving process* may make their interpretation easier for the operator.

The extensiveness of a crack may directly alter the prognosis assessment and treatment plan for a given tooth; the presence of a fracture may lead to the demise of a tooth and may compromise the periradicular bone. Therefore, developing a diagnosis, prognosis assessment, and treatment plan for teeth with suspected cracks and fractures is essential, with an emphasis on early detection. In addition, endodontic and restorative procedures should focus on minimizing any offending and predisposing factors that may perpetuate cracks and fractures.

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CHAPTER 22

Restoration of the Endodontically Treated Tooth

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CHAPTER OUTLINE

Special Features of Endodontically Treated Teeth
Compositional Changes in Nonvital Teeth and the Influence of Endodontic Therapy
Dentin Structure and Properties in Nonvital and Endodontically Treated Teeth
Fracture Resistance and Tooth Stiffness of Nonvital and Endodontically Treated Teeth
Aesthetic Changes in Nonvital and Endodontically Treated Teeth
Restorative Materials and Options
Direct Composite Restorations
Indirect Restorations: Composite or Ceramic Onlays and Overlays
Full Crowns
Luting Cements
Pretreatment Evaluation and Treatment Strategy
Pretreatment Evaluation
Treatment Strategy
Clinical Procedures
Tooth Preparation
Post Placement
Adhesive Procedures
Partial Restorations
Foundation Restoration Underneath Full Crowns
Crown Preparation and Temporary Restoration
Summary

SPECIAL FEATURES OF ENDODONTICALLY TREATED TEETH

Once endodontic therapy is completed, the tooth must be adequately restored. Indeed, given the large impact that poor or missing restorations have on the survival of endodontically treated teeth, one could make the argument that the restoration is actually the last step of endodontic therapy. However, it is important to realize that endodontically treated teeth are structurally different from nontreated vital teeth. Major changes following treatment include altered tissue physical characteristics, loss of tooth structure, and possibly discoloration. Research has analyzed these tissue modifications at different levels, including tooth composition, dentin microstructure, and tooth macrostructure. These studies indicate that it is critical to understand the implication of such features on tooth biomechanics, as they will largely influence the restorative approach and means (Table 22-1). Additional in vitro studies dealing with the complexity of the nonvital tooth substrate are reported in the literature; ultimately, clinical studies have documented the global effect of these changes on the long-term survival of endodontically treated teeth.

Compositional Changes in Nonvital Teeth and the Influence of Endodontic Therapy

The loss of pulpal vitality is accompanied by a slight change in tooth moisture content. This loss of moisture (9%) is attributed to a change in free water but not in water bonded to the organic and inorganic components.[65,69] This alteration was associated with a slight change in values for the Young modulus and proportional limit.[75] However, no decrease in compressive and tensile strength values was associated with this change in water content.[75] Only one study showed no difference in moisture content between vital and nonvital teeth.[123] No difference in collagen cross-linkage was found between vital and nonvital dentin.[140] Thus, nonvital teeth undergo rather minor changes in physical characteristics.

Sodium hypochlorite and chelators such as ethylenediamine tetra-acetic acid (EDTA), cyclohexane-1,2-diaminetetra-acetic acid (CDTA), ethylene glycol-bis-(β-amino-ethyl ether) N,N,N′,N′-tetra-acetic acid (EGTA), and calcium hydroxide ($Ca[OH]_2$) commonly used for canal irrigation and disinfection interact with root dentin, with either mineral content

TABLE 22-1
Specific Tissue Modifications and Possible Clinical Implications Following Loss of Vitality or Endodontic Treatment

Alteration Level	Specific Changes	Possible Clinical Implication
Composition	Collagen structure Tooth moisture Mineral composition and content	Increased tooth fragility Reduced adhesion to substrate
Dentin structure	Elasticity modulus and behavior Tensile and shear strength Microhardness	Increased tooth fragility
Tooth macrostructure	Resistance to deformation Resistance to fracture Resistance to fatigue	Increased tooth fragility Reduced retention/stability of the prosthesis

(chelators) or organic substrate (sodium hypochlorite).[78,116,119] Chelators mainly deplete calcium by complex formation and also affect noncollagenous proteins (NCPs), leading to dentin erosion and surface softening.[78,82,144] Depending on concentration, duration of exposure, and other factors (see Chapter 6), sodium hypochlorite can demonstrate a proteolytic action by hydrolysis of long peptide chains such as collagen.[68] These alterations are likely to impact dentin and root structure and alter bonding properties of this substrate.

Dentin Structure and Properties in Nonvital and Endodontically Treated Teeth

It is important to know that dentin displays a range of normal variations in its physical properties which must be distinguished from alterations related to loss of vitality or endodontic treatment. For instance, dentin microhardness and elasticity actually vary between peritubular and intertubular dentin and depend on tooth location. Peritubular dentin presents an elasticity modulus of 29.8 GPa, whereas intertubular dentin ranges from 17.7 GPa (close to pulp) to 21.1 GPa (close to the root surface).[70,85,101] Most if not all the decreases in hardness on approaching the pulp can be attributed to changes in hardness of the intertubular dentin.[84,85] Overall dentin elasticity modulus is considered to be in the range of 16.5 to 18.5 Gpa.[15,32,50,86,121,138]

The changes in mineral density due to the variation in the number and diameter of tubules within the tooth may also contribute to variations in the properties of dentin. Not surprisingly, dentin hardness values are inversely related to dentin tubule density.[124] Ultra microindentation measurements also demonstrated significantly higher values for hardness and elasticity modulus when forces were parallel to the tubules rather than perpendicular.[133] Differences in maximum strength and compressive strength were also found to vary according to tubule orientation.[121] The ultimate tensile strength (UTS) of human dentin is lowest when the tensile force is parallel to tubule orientation, showing the influence of dentin microstructure and anisotropy of the tissue.[93] No difference was found in the Young modulus of aged, transparent dentin (also called *sclerotic*) and normal dentin,[19,87,167] but the mineral concentration significantly increases and crystallite size is slightly smaller in transparent dentin, in relation with closure of the tubule lumens. Transparent dentin, unlike normal dentin, exhibits almost no yielding before failure. Its fracture toughness is also lowered by about 20%, whereas the fatigue lifetime is deleteriously affected.[87]

Interestingly, comparisons between vital and nonvital dentin of contralateral teeth demonstrate no or only minor differences in microhardness values after periods ranging up to 10 years after treatment.[94,154] Thus, the literature does not support a widely held belief that attributes particular weakness or brittleness to nonvital dentin. Others have suggested that nonvital teeth in older patients may have greater risk for fracture because the age-related generation of secondary or tertiary dentin would be lost. However, this is not the case because the only impact of age-related tissue changes is the aforementioned reduction in fracture toughness and fatigue lifetime attributed to dentin sclerosis.[87]

The chemicals used for canal irrigation and disinfection, as already mentioned, interact with mineral and organic contents and then reduce dentin elasticity and flexural strength to a significant extent,[64,157] as well as microhardness.[33,77,146] On the contrary, disinfectants like eugenol and formocresol increase dentin tensile strength by way of protein coagulation and chelation with hydroxyapatite (eugenol). Dentin hardness, however, did not prove to be influenced by the latter products.[112]

In conclusion, a possible decrease in tooth strength can be attributed to dentin aging and to a smaller extent to dentin alteration by endodontic irrigants.

Fracture Resistance and Tooth Stiffness of Nonvital and Endodontically Treated Teeth

In contrast to the aforementioned factors, the major changes in tooth biomechanics appear to be due to the loss of hard tissue following decay, fracture, or cavity preparation (including the access cavity prior to endodontic therapy).

The loss of hard tooth structure following a conservative access cavity preparation affects tooth stiffness by only 5%.[91,169] The influence of subsequent canal instrumentation and obturation lead only to a slight reduction in the resistance to fracture[91,169] and ultimately have little effect on tooth biomechanics.[91,137,169] From a clinical perspective, one can expect alteration of tooth biomechanics only in cases of nonconservative canal preparation or through the chemical or structural alteration triggered by endodontic irrigants, as previously mentioned.

In fact, the largest reduction in tooth stiffness results from excessive access preparation, especially the loss of marginal ridges. The literature reports a 20% to 63% and a 14% to 44% reduction in tooth stiffness following occlusal and MOD cavity preparations, respectively.[44,92,137] It was shown that an endodontic access cavity combined with an MOD preparation results in maximum tooth fragilization. The cavity depth, isthmus width, and configuration are therefore highly critical to the reduction in tooth stiffness and risk of fracture (Fig. 22-1).[74,83,96,122] This important point has profound clinical implications.

FIG. 22-1 The negative impact of poor initial biomechanical status on restoration success. **A, B,** Preoperative radiographic views following the removal of the old metallic foundation. **C,** A new amalgam core, using post and self-anchorage into mesial root structure, was performed. **D,** Prosthetic restorations on working model. **E,** Full arch view after 3 years. **F,** The tooth is symptomatic due to furcation involvement and periapical lesion. **G,** This untreatable tooth was finally extracted and replaced by an implant. **H,** Eight-year postoperative radiograph showing a stable situation. Other teeth with less extensive biomechanical damage overcame functional stresses.

The presence of residual hard tissue in the cervical area (which constitutes the *ferrule* for restorations) and a larger amount of residual mineralized tissue in general increase tooth resistance to fracture. It actually allows the axial walls of the crown to encircle the tooth, providing restoration retention and stabilization and reducing cervical tensile stresses.[6,23,161] Crown preparations with as little as 1 mm coronal extension of dentin above the margin double the fracture resistance of preparations, compared to those where the core terminates on a flat surface immediately above the margin[103,161]; therefore, a minimal 1-mm ferrule (and ideally 2 to 3 mm) is considered necessary to stabilize the restoration.[161] However, the widths of preparation shoulder and crown margin do not appear to influence fracture strength.[2] Taken together, the height of the ferrule is one of the most important elements in the long-term survival of restorations. These considerations have led to the conclusion that the most important part of the restored tooth is the tooth itself, and no contemporary restorative material or combination of materials will perfectly substitute for lost tooth structure.

Aesthetic Changes in Nonvital and Endodontically Treated Teeth

Several aesthetic changes may also occur in nonvital or endodontically treated teeth. For example, color change or darkening of nonvital teeth is a common clinical observation (Fig. 22-2). In addition, incomplete endodontic treatment can contribute to discoloration. For instance, inadequate cleaning and shaping can leave necrotic tissue in coronal pulp horns, resulting in tooth darkening. In addition, root canal filling materials (gutta-percha and root canal sealer cements, MTA-like materials) retained in the coronal aspect of anterior teeth can detract from the aesthetic appearance. Opaque substances also adversely affect the color and translucency of most uncrowned teeth. Biochemically altered dentin modifies tooth color and appearance. It is generally accepted that organic substances present in dentin (e.g., hemoglobin) might play an important role in this color change and also food and drink pigment penetration triggered by the absence of pulpal pressure. However, the respective contribution of these two phenomena and precise physicochemical mechanisms leading to discoloration are poorly understood or described in the literature.[34,67,131]

FIG. 22-2 Severe discoloration can significantly disturb aesthetics, even in the lateral area of the smile. When not treatable with bleaching agents or veneers, this condition might justify tooth preparation for a full crown.

Thin gingival tissue or, in general, thin biotype is considered a negative factor for aesthetic outcome of restorative and prosthetic treatment of discolored teeth.[110,111,118]

Endodontic treatment and subsequent restoration of teeth in the aesthetic zone require careful control of procedures and materials to retain a translucent, natural appearance. It is therefore strongly recommended that one avoid the use of potentially staining endodontic cements and clean all material residues left in the pulpal chamber and access cavity.

RESTORATIVE MATERIALS AND OPTIONS

As described previously, endodontic treatment, particularly excessive access preparations, can result in significant loss and weakening of tooth structure. Tooth structure lost during endodontic treatment increases the risk of crown fracture, with fatigue mechanisms mediating the fracture of roots over time. Restorations of endodontically treated teeth are designed to (1) protect the remaining tooth from fracture, (2) prevent reinfection of the root canal system, and (3) replace the missing tooth structure.

According to the amount of tissue to be replaced, restorations of endodontically treated teeth rely on different materials and clinical procedures. As a general rule, most structurally damaged teeth should be restored with an artificial crown.

Although the use of a crown built on post and core is a traditional approach, others have advocated the use of direct composite resins for restoring small defects in endodontically treated teeth. More recently, indirect restorations such as overlays or endocrowns made of composite resins or ceramics have also been used. The selection of appropriate restorative materials and techniques is dictated by the amount of remaining tooth structure. This is far more relevant to the long-term prognosis of endodontically treated teeth than any properties of post, core, or crown materials.

Direct Composite Restorations

When a minimal amount of coronal tooth structure has been lost after endodontic therapy, a direct resin composite restoration may be indicated. Composite resins are a mixture of a polymerized resin network reinforced by inorganic fillers. Contemporary composites have compressive strengths of about 280 MPa, and the Young modulus of composite resins is generally about 10 to 16 GPa, which is close to that of dentin.[134]

When properly cured, resin composites are highly aesthetic, exhibit high mechanical properties, and can reinforce the remaining tooth structure through bonding mechanisms. Typically, 500 to 800 mW/cm^2 of blue light for 30 to 40 seconds is necessary to polymerize an increment of composite that must be 1- to 3-mm thick. Unfortunately, the shrinkage that accompanies polymerization of contemporary composite resins remains a significant problem to the long-term success of these restorations. The use of an incremental filling technique, which helps to reduce shrinkage stresses during polymerization, is highly recommended. The amount of shrinkage will also depend on the shape of the cavity preparation and the ratio of bonded to unbonded (or free) surfaces.[36] This so-called C-factor is a clinically relevant predictor of the risk of debonding and leakage; restorations with high C-factors (> 3.0) are at greatest risk for debonding.[175] In other words, a direct composite

restoration may be indicated when only one proximal surface of the tooth has been lost; using an incremental filling technique is mandatory.

Classically, direct composite restorations have been placed in anterior teeth that have not lost tooth structure beyond the endodontic access preparation. In such cases, the placement of a direct composite restoration offers an immediate sealing of the tooth, which prevents coronal leakage and recontamination of the root canal system with bacteria. In vitro studies have demonstrated that the fracture resistance of small bonded restorations is nearly as great as that of intact teeth.[57,136]

Although direct composite resins may also be used for small restorations in posterior teeth, they are contraindicated when more than a third of coronal tissue has been lost. In one study,[136] it was reported that the resistance to fracture of endodontically treated teeth is reduced by 69% in cases where MOD cavities are present.[137] Under such conditions, a direct composite restoration may not be appropriate to prevent the tooth structure from fracture and reinfection. Furthermore, resin composite materials may require the use of reinforcing in vitro fibers to increase their mechanical resistance. Although most studies on the clinical performance of direct composite restorations were conducted on vital teeth, one clinical report indicates that direct in vitro fiber-reinforced composite restorations may represent a valuable alternative to conventional restorations of endodontically treated teeth.[37] On the contrary, inserting an in vitro fiber post in the root canal of an endodontically treated tooth before bonding a direct MOD restoration significantly reduces its fracture resistance compared to the same composite restoration without a post.[160]

Indirect Restorations: Composite or Ceramic Onlays and Overlays

Ceramic or resin composite onlays and endocrowns can also be used to restore endodontically treated teeth. Whereas overlays incorporate a cusp or cusps by covering the missing tissue, endocrowns combine the post in the canal, the core, and the crown in one component.[88,142] Both onlays and endocrowns allow for conservation of remaining tooth structure, whereas the alternative would be to completely eliminate cusps and perimeter walls for restoration with a full crown.[58] Onlays and overlays are generally constructed in the laboratory from either hybrid resin composite or ceramics.

Ceramics are a material of choice for long-term aesthetic indirect restorations because their translucency and light transmission mimic enamel. Whereas traditional feldspathic porcelains were sintered from a slurry, new ceramic materials may be cast, machined, pressed, or slip-cast, in addition to being sintered. New materials either are variations of feldspathic porcelains (e.g., In-Ceram, Cerec, IPS Empress) or may be fabricated from other ceramic systems, including alumina, zirconia, or silica.[3,38] Among these newer compositions is lithium disilicate, which offers high strength, high fracture toughness, and a high degree of translucency. Physical properties of these materials have improved to the point where they can survive high stress-bearing situations such as posterior restorations in endodontically treated teeth.[46,73] Researchers have examined 140 partial Cerec restorations (Vita MKII, feldspathic porcelain) adhesively cemented to endodontically treated teeth and found this treatment approach satisfactory after an observation period of 55 months.[9] Their results indicate that survival rates are higher for molars than for premolars.

Onlays, overlays, and endocrowns can also be fabricated from resin composites processed in the laboratory. Using various combinations of light, pressure, and vacuum, these fabrication techniques are claimed to increase the conversion rate of the polymer and consequently the mechanical properties of the restorative material. Other investigations have described the application of glass fiber–reinforced composite endocrowns on premolars and molars as single restorations or as abutment for fixed partial dentures.[58,59] One in vitro study by another research team indicates that composite inlays can partially restore the resistance to fracture of endodontically treated molars and prevent catastrophic fractures after loading.[28] Other investigators reported that composite resin MZ100 increased the fatigue resistance of overlay-type restorations in endodontically treated molars when compared to porcelain MKII.[97] Another study used three-dimensional finite element analysis to estimate bone resorption around endocrowns made up of high-(alumina) or low-elastic modulus materials (resin composites). They concluded that the higher resilience of resin composite restorations acts positively against the risk of periodontal bone resorption by reducing the amount of force transferred to root dentin.[4]

Full Crowns

When a significant amount of coronal tooth structure has been lost by caries, restorative procedures, and endodontics, a full crown may be the restoration of choice. In a few cases, the crown can be directly built on the remaining coronal structure, which has been prepared accordingly (see the Core Materials section). More frequently, the cementation of a post inside the root canal is necessary to retain the core material and the crown.[164] The core is anchored to the tooth by extension into the root canal through the post and replaces missing coronal structure. The crown covers the core and restores the aesthetics and function of the tooth.

An additional role of the post and core is to protect the crown margins from deformation under function and thereby to prevent coronal leakage. Because most endodontic sealers do not completely seal the root canal space, the coronal seal provided by the placement of a post and core will positively influence the outcome of the endodontic treatment.[148] The post's ability to anchor the core is also an important factor for successful reconstruction, because the core and the post are usually fabricated of different materials. Finally, the luting material used to cement the post, the core, and the crown to the tooth will also influence the longevity of the restoration. The post, the core, and their luting or bonding agents together form a *foundation restoration* to support the future crown.[106]

The Foundation Restoration: General Considerations

Although many materials and techniques can be used to fabricate a foundation restoration, no combination of materials can substitute for tooth structure. As a general rule, the more tooth structure that remains, the better the long-term prognosis of the restoration. The coronal tooth structure located above the gingival level will help to create a ferrule.[6,100,128] The *ferrule* is formed by the walls and margins of the crown, encasing at least 2 to 3 mm of sound tooth structure. A properly executed ferrule significantly reduces the incidence of fracture in

endodontically treated teeth by reinforcing the tooth at its external surface and dissipating forces that concentrate at the narrowest circumference of the tooth.[95,173] A longer ferrule increases fracture resistance significantly. The ferrule also resists lateral forces from posts and leverage from the crown in function and increases the retention and resistance of the restoration. To be successful, the crown and crown preparation together must meet five requirements:

1. The ferrule (dentin axial wall height) must be at least 2 to 3 mm.
2. The axial walls must be parallel.
3. The restoration must completely encircle the tooth.
4. The margin must be on solid tooth structure.
5. The crown and crown preparation must not invade the attachment apparatus.

Root anatomy can also have significant influence over post placement and selection. Root curvature, furcations, developmental depressions, and root concavities observed at the external surface of the root are all likely to be reproduced inside the root canal (see also Chapter 5). Within the same root, the shape of the canal will vary between the cervical level and the apical foramen.[62] As a result, severe alteration of the natural shape of the canal is often necessary to adapt a circular post inside the root. This increases the risk of root perforation, especially in mesial roots of maxillary and mandibular molars that exhibit deep concavities on the furcal surface of their mesial root.[16,89] The tooth is also weakened if root dentin is sacrificed to place a larger-diameter post. A study using three-dimensional electronic speckle-pattern interferometry (ESPI) evaluated the effects of root canal preparation and post placement on the rigidity of human roots.[93] ESPI has the major advantage of being able to assess tooth deformation in real time and can be used repeatedly on the same root because of the nondestructive nature of the test. Study results indicate that root deformability increases significantly after the preparation of a post space. Thus, preservation of root structure is also a guiding principle in the decision to use a post, the selection of the post, and the preparation of the post space. This is a reason why not every endodontically treated tooth needs a post and why more conservative approaches that do not rely on the use of a post are currently being developed. However, a post may be used in the root of a structurally damaged tooth in which additional retention is needed for the core and coronal restoration. Posts should provide as many of the following clinical features as possible:

- Maximal protection of the root from fracture
- Maximal retention within the root and retrievability
- Maximal retention of the core and crown
- Maximal protection of the crown margin seal from coronal leakage
- Pleasing aesthetics, when indicated
- High radiographic visibility
- Biocompatibility

From a mechanical point of view, an endodontic post should not break, should not break the root, and should not distort or allow movement of the core and crown. An ideal post would have an optimal combination of resilience, stiffness, flexibility, and strength. *Resilience* is the ability to deflect elastically under force without permanent damage. It is a valuable quality in endodontic posts, but too much flexibility in a narrow post compromises its ability to retain the core and crown under functional forces. *Stiffness* describes a material's ability to resist deformation when stressed. The stiffness of a material is an inherent physical property of that material, regardless of size. However, the actual flexibility of a post depends both on the diameter of a specific post and on the modulus of elasticity of the post's material. Posts with a lower modulus of elasticity are more flexible than posts of the same diameter with a higher modulus of elasticity. Posts made of nonstiff materials (low modulus of elasticity) are more resilient, absorb more impact force, and transmit less force to the root than stiff posts, but low-modulus posts fail at lower levels of force than do high-modulus posts.[99,120,143]

Excessive flexing of the post and micromovement of the core are particular risks in teeth with minimal remaining tooth structure, because these teeth lack their own cervical stiffness as a result of the missing dentin. Post flexion can also distort and open crown margins. Open margins can result in potentially devastating caries or endodontic leakage and apical reinfection. Extensive caries extending into the root can be as irreparable as root fracture. Because rigid posts flex and bend less than nonrigid posts, they can limit movement of the core and possible disruption of the crown margins and cement seal. However, the force must go somewhere. Force from a stiff post is transmitted to the root, next to the apex of the post. An attempt to strengthen a weak root by adding a stiff post can instead make the root weaker as a result of the force concentration of a stiff rod in a more flexible material. Stress concentration in the post/root complex can lead to the self-destructive process of cracking and fracturing. Root fracture is particularly a risk in teeth with minimal remaining tooth structure to support a ferrule.

Roots also flex under force, which is a function of both the modulus of elasticity of dentin and the diameter of the root. Dentin is relatively flexible, and posts can be flexible or stiff. Although no material can behave exactly like dentin, a post with functional behavior similar to that of dentin is beneficial when the post must be placed next to dentin. Posts have been developed with a modulus of elasticity closer to dentin than that offered by traditional metal posts. But posts are significantly narrower than roots, and the actual deflection of a post within dentin is a function of both the modulus of elasticity and the diameter. The modulus of elasticity of various posts, compared with that of dentin, represents only one aspect of flexion.

In summary, an ideal post would be resilient enough to cushion an impact by stretching elastically, thereby reducing the resulting stress to the root. It would then return to normal without permanent distortion. At the same time, this ideal post would be stiff enough not to distort, permanently bend, or structurally fail under mastication forces. Finally, the perfect post would combine the ideal level of flexibility and strength in a narrow-diameter structure, which is dictated by root canal morphology. Current post systems are designed to provide the best compromise between the desired properties and inherent limitations of available materials.

Why Roots Fracture

Structures subjected to low but repeated forces can appear to fracture suddenly for no apparent reason. This phenomenon, also known as *fatigue failure,* occurs when a material or a tissue is subjected to cyclic loading. Fatigue may be characterized as a progressive failure phenomenon that proceeds by the initiation and propagation of cracks; many failures of teeth or

materials observed in the mouth are fatigue-related. Because teeth are subjected to fluctuating cycles of loading and unloading during mastication, fatigue failure of dentin, posts, cores, crown margins, or adhesive components is likely to occur.[153] Mechanical loading will favor the propagation of microcracks that will progress from the coronal to the apical region of the tooth.

Initial failure of crown margins from fatigue loading is clinically undetectable. However, when measured in vitro, early failure resulted in significant leakage of crown margins, extending between the tooth, restoration, and post space. Particularly in teeth with minimal remaining tooth structure, fatigue can cause endodontic posts to bend permanently or break, or it can cause a fiber-matrix complex to disintegrate.

Fatigue failure of nonvital teeth restored with a post is more catastrophic because it may result in a complete fracture of the root. A post placed into a dentin root will function physically like any structural rod anchored in another material. This means that the forces applied on the post are transmitted to the root dentin with characteristic patterns depending on the modulus of elasticity of both the post and the dentin. If the post has a higher modulus than the dentin, the stress concentration is adjacent to the bottom of the post (Fig. 22-3). This is evident in clinical cases of root fracture originating at the apex of a rigid post.

When the stiffness of the endodontic post is similar to that of dentin, stresses are not concentrated in the dentin adjacent to the apex of the post but rather are dissipated by both the coronal and the root dentin (see Fig. 22-3). A resilient post can also prevent a sudden blow by stretching elastically, which reduces the transient forces against the tooth, but a post that is too elastic becomes too flexible for retaining a core and a crown when the tooth cannot do so on its own. A resilient post that is overloaded fails with less force than a stiffer post. This limits the amount of resilience that can be designed into a post.

Direct Foundation Restorations

In general, the evolution of foundation restorations has been to diminish invasiveness and eliminate some components in selected cases. When a sufficient amount of tissue is present at the periphery of the prepared tooth, a direct foundation restoration is indicated. In the direct technique, a prefabricated post is cemented inside the root canal, and the core is built directly on the prepared tooth. For other clinical situations, an indirect custom-cast post and core may be indicated.

Various materials can be used to fabricate a direct foundation restoration. Although there is growing interest in using resin-based materials such as resin composites or fiber-reinforced resin posts, more traditional materials such as amalgam are still used for that purpose.[27] For clarity, the components used for fabricating a direct foundation restoration (e.g., the endodontic post and core material) are described individually.

Posts

The large number of post designs and materials available on the market reflects the absence of consensus in that field. Based on what manufacturers or clinicians consider the most important properties, posts can be fabricated from metal (gold, titanium, stainless steel), ceramic, or fiber-reinforced resins. As a general rule, a post needs retention and resistance. Whereas *post retention* refers to the ability of a post to resist vertical

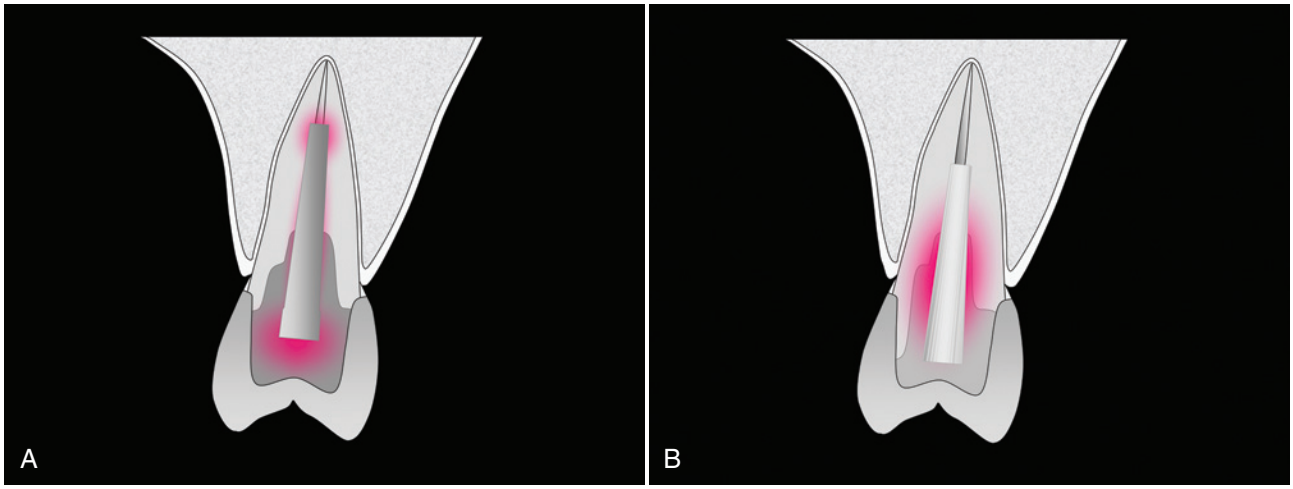

FIG. 22-3 **A,** Stress distribution within a metallic post and core foundation and residual tooth structure, according to photoelastic and FEM studies. The post is cemented and usually penetrates the apical portion of the root. Functional stresses accumulate inside the foundation, slightly around the post and further inside the canal, around the apex of the post; there is less stress buildup in the cervical area compared to that with a fiber post (as shown in Fig. 22-3, *B*). This configuration more ideally protects the coronocervical structures, but when failing, it results in severe untreatable root fractures. **B,** Stress distribution within a fiber post/composite foundation and residual tooth structure, according to photoelastic and FEM studies. The post is bonded to the canal walls and penetrates the canal less apically. Functional stresses mainly accumulate around the post in the cervical area. This configuration protects the cervical area less efficiently but tends to prevent untreatable root fractures. The presence of a ferrule is mandatory. (Adapted from Dietschi D, Duc O, Krejci I, Sadan A: Biomechanical considerations for the restoration of endodontically treated teeth: a systematic review of the literature—Part 1. Composition and micro- and macrostructure alterations, *Quintessence Int* 38:733, 2007.)

forces, *resistance* refers to the ability of the tooth/post combination to withstand lateral and rotational forces. Resistance is influenced by the presence of a ferrule, the post's length and rigidity, and the presence of antirotational features. A restoration lacking resistance form is not likely to be a long-term success, regardless of the retentiveness of the post.

Prefabricated Metallic Posts

Prefabricated metallic posts are frequently used for the fabrication of a direct foundation restoration. These posts are classified several ways, including by alloy composition, retention mode, and shape. Materials used to fabricate metallic posts include gold alloys, stainless steel, or titanium alloys. Metallic posts are very strong and, with the exception of the titanium alloys, very rigid.[90] One study indicates that the flexural strength of stainless steel posts is about 1430 MPa and that flexural modulus approximates 110 GPa.[130] On the other hand, titanium posts are less rigid (66 GPa) but exhibit a flexural strength (1280 MPa) similar to stainless steel.

The retention of prefabricated posts inside the root canal is also essential for successful restorations. Two basic concepts have been used to promote the retention of endodontic posts: active posts and passive posts. Active posts derive their primary retention directly from the root dentin by the use of threads. Most active posts are threaded and are intended to be screwed into the walls of the root canal. A major concern about threaded posts has been the potential for vertical root fracture during placement. As the post is screwed into place, it introduces great stresses within the root, causing a wedging effect.[163] Therefore, it is generally accepted that *the use of threaded posts should be avoided.* Furthermore, the improved retention once offered by threaded posts can now be achieved with adhesive luting cements (discussed later).[117] Passive posts are passively placed in close contact to the dentin walls, and their retention primarily relies on the luting cement used for cementation. The shape of a passive post may be either tapered or parallel.[139] A parallel post is more retentive than a tapered post but also requires removal of more root dentin during the preparation of the post space. Parallel posts are reported to be less likely to cause root fractures than tapered posts, although they are less conforming to the original shape of the root.[79,152,161] Unfortunately, modern techniques for root canal preparation use tapered nickel-titanium (NiTi) rotary shaping files, which result in a very wide tapered and unretentive canal exhibiting a significant divergence from apical to coronal.[145] Longer posts are often necessary to accommodate this problem and offer adequate retention; adequate length in the root canal is considered to be greater than 6 mm. When teeth are protected by crowns with an adequate ferrule, longer posts do not further increase fracture resistance.[80] Posts designed with mechanical locking features in the heads and roughened surface texture can show better retention of the core.[29]

Fiber Posts

A fiber post consists of reinforcing fibers embedded in a resin-polymerized matrix. Monomers used to form the resin matrix are typically bifunctional methacrylates (Bis-GMA, UDMA, TEGDMA), but epoxies have also been used. Common fibers in today's fiber posts are made of carbon, glass, silica, or quartz but the type, volume content, and uniformity of the fibers and the matrix are proprietary and vary among fiber post systems. These differences in the manufacturing process may reflect the large variations observed among different types of fiber posts subjected to a fatigue-resistance test.[63] Fibers are commonly 7 to 20 μm in diameter and are used in a number of configurations, including braided, woven, and longitudinal. The original fiber posts consisted of carbon fibers embedded in epoxy resin, but quartz-fiber posts are currently preferred for their favorable mechanical properties, aesthetic qualities, and ability to chemically bond to the polymer matrix.[49] One study indicates that the flexural strength of glass-, silica-, or quartz-fiber posts approximates 1000 MPa and that flexural modulus is about 23 GPa.[35] Current fiber posts are radiopaque and may also conduct the light for polymerization of resin-based luting cements. A light-transmitting post results in better polymerization of resin composites in the apical area of simulated root canals, as measured by hardness values.[141,174] To enhance bonding at the post/core/cement interfaces, several physicochemical pretreatments such as silanization or sand blasting of the post surface have been described. Research indicates that silanization, hydrofluoric etching, and sandblasting (with 30 to 50 μm Al_2O_3) do not modify the mechanical properties of different glass-, silica-, or quartz-fiber posts.[5]

It is generally accepted that bonding fiber posts to root canal dentin can improve the distribution of forces applied along the root, thereby decreasing the risk of root fracture and contributing to the reinforcement of the remaining tooth structure.[12,18] A well-adapted adhesively cemented fiber post is considered the most retentive with the least stress generated on the canal walls. In a retrospective study that evaluated three types of bonded fiber posts, investigators reported a 3.2% failure of 1306 fiber posts in recalls of 1 to 6 years.[54] More recently, another study reported survival rates of 98.6% and 96.8% for parallel-sided and tapered fiber posts, respectively, placed in anterior teeth covered with full-ceramic crowns after a mean observation period of 5.3 years.[156]

Zirconia posts are composed of zirconium dioxide (ZrO_2) partially stabilized with yttrium oxide and exhibit a high flexural strength. Zirconia posts are aesthetic, partially adhesive, rigid, but also brittle. Zirconia posts cannot be etched, and available literature suggests that bonding resins to these materials is less predictable and requires substantially different bonding methods than conventional ceramics.[11] When a composite core is built on a zirconia post, core retention may also be a problem. Controversies exist about the efficiency of airborne particle abrasion at establishing a durable resin bond to zirconia posts treated or not treated with a coupling agent.[1,127] Overall, there are concerns about the rigidity of zirconia posts, which tends to make those posts too brittle. Other reports indicate that the rigidity of zirconia posts negatively affects the quality of the interface between the resin core material and dentin when subjected to fatigue testing.[40,43]

Core Materials

The core replaces carious, fractured, or missing coronal structure and helps to retain the final restoration. Desirable physical characteristics of a core include (1) high compressive and flexural strength, (2) dimensional stability, (3) ease of manipulation, (4) short setting time, and (5) the ability to bond to both tooth and post. Core materials include composite resin, cast metal or ceramic, amalgam, and sometimes glass-ionomer materials. The core is anchored to the tooth by extension into the coronal aspect of the canal or through the endodontic post. The importance of retention between the

post, the core, and the tooth increases as remaining tooth structure decreases.

Composite Resin Core

Composite core materials take a number of strategies to enhance their strength and resistance; metal may be added, filler levels may be greater, or faster-setting ionomers may be used.[134] Composite core materials have been shown to exhibit slightly better mechanical values than conventional materials, but improvements are negligible.[176] However, they appear to be superior to silver-glass-ionomer cement and amalgam.[30] The advantages of composite core resins are adhesive bonding to tooth structure and many posts, ease of manipulation, rapid setting, and translucent or highly opaque formulations. Composite cores have been shown to protect the strength of all-ceramic crowns equally to amalgam cores. Bond strength of composite cores to dentin depends on a complete curing of the resin materials, so dentin bonding agents must be chemically compatible with composite core materials. Self-cure composite resins require self-cure adhesives and are mostly incompatible with light-cure adhesives.[26] However, no adhesive has been shown to completely eliminate microleakage at the margins of the restoration.[17] A degradation of the resin core or the marginal integrity of the crown can result in invasion of oral fluids. Therefore, as with all buildup materials for decimated teeth, more than 2 mm of sound tooth structure should remain at the margin for optimal composite resin core function.

Composite core materials can be used in association with metallic, fiber, or zirconia posts. This is frequently observed in the presence of structurally compromised teeth. They may provide some protection from root fracture in teeth restored with metal posts compared with amalgam or gold cores. Loosening of the post, core, and crown with composite core can occur, but composite cores have been shown to fail more favorably than amalgam or gold.[129] A retrospective study of the clinical performance of fiber posts indicates that fiber posts and cores have a failure rate ranging between 7% and 11% after a service period of 7 to 11 years and that post loosening may also occur.[53] Composite core materials are typically two-paste, self-cured composites, but light-curing materials are also available. The use of light-curing composite core materials generally eliminates the risk of chemical incompatibility between adhesives and self-curing resin core materials. Bonding light-cured resin composites to the irregular structure of the pulp chamber and canal orifices might eliminate the need for a post when sufficient tooth structure remains. Research indicates that bonding to the dentin walls of the pulp chamber is easier and superior to resin dentin bonds made on dentin canal walls.[7]

Amalgam Core

Dental amalgam is a traditional core buildup material with a long history of clinical success. Although there are many variations in the alloy's composition, more recent formulations have high compressive strength (400 MPa after 24 hours), high tensile strength, and a high modulus of elasticity. High-copper alloys tend to be stiffer (60 GPa) than low-copper alloys.

Amalgam can be used with or without a post. In the 1980s, investigators described the amalcore.[113] With the amalcore technique, amalgam is compacted into the pulp chamber and 2 to 3 mm coronally of each canal. The following criteria were considered for the application of this technique: the remaining pulp chamber should be of sufficient width and depth to provide adequate bulk and retention of the amalgam restoration, and an adequate dentin thickness around the pulp chamber was required for the tooth-restoration continuum rigidity and strength. The fracture resistance of the amalgam coronal-radicular restoration with four or more millimeters of chamber wall was shown to be adequate, although the extension into the root canal space had little influence.[81]

Amalgam can also be used in combination with a prefabricated metallic post when the retention offered by the remaining coronal tissue needs to be increased. Amalgam cores are highly retentive when used with a preformed metal post in posterior teeth; they require more force to dislodge than cast posts and cores.[102] Others have suggested the use of adhesive resins to bond amalgam to coronal tissue.[155]

Significant disadvantages of amalgam cores are the "nonadhesive nature" of the material, the potential for corrosion, and subsequent discoloration of the gingiva or dentin. Amalgam use is declining worldwide because of legislative, safety, and environmental issues.

Glass Ionomer Core and Modified Glass Ionomer Core

Glass ionomer and resin-modified glass ionomer cements are adhesive materials useful for small buildups or to fill undercuts in prepared teeth. The rationale for using glass ionomer materials is based on their cariostatic effect resulting from fluoride release. However, their low strength and fracture toughness result in brittleness, which contraindicates the use of glass-ionomer buildups in thin anterior teeth or to replace unsupported cusps. They may be indicated in posterior teeth in which (1) a bulk of core material is possible, (2) significant sound dentin remains, and (3) caries control is indicated.[172]

Resin-modified glass ionomer materials are a combination of glass ionomer and composite resin technologies and have properties of both materials. Resin-modified glass ionomers have moderate strength, greater than glass ionomers but less than composite resins. As a core material, they are adequate for moderate-sized buildups, but hygroscopic expansion can cause fracture of ceramic crowns and fragilized roots.[158] The bond to dentin is close to that of dentin-bonded composite resin and significantly higher than traditional glass ionomers. Today, resin composites have replaced glass ionomer materials for core fabrication.

Indirect Foundation Restorations: Cast Post and Core

For many years, use of the cast metal post and core has been the traditional method for fabricating the foundation restoration of a prosthetic crown. Classically, smooth-sided, tapered posts conforming to the taper of the root canal are fabricated from high noble alloys, although noble and base-metal classes of dental alloys have also been used. Noble alloys used for post and core fabrication have high stiffness (approximately 80 to 100 GPa), strength (1500 MPa), hardness, and excellent resistance to corrosion.[31]

One advantage of the cast post/core system is that the core is an integral extension of the post and that the core does not depend on mechanical means for retention on the post. This construction prevents dislodgment of the core from the post and root when minimal tooth structure remains. However, the cast post/core system also has several disadvantages. Valuable tooth structure must be removed to create a path of insertion

or withdrawal. Second, the procedure is expensive because two appointments are needed, and laboratory costs may be significant. The laboratory phase is technique sensitive. Metal casting of a pattern with a large core and a small-diameter post can result in porosity in the gold at the post/core interface. Fracture of the metal at this interface under function results in failure of the restoration. Most important, the cast post/core system has a higher clinical rate of root fracture than preformed posts.[47,159]

Studies on cast post retention have shown that the post must fit the prepared root canal as closely as possible to be perfectly retained. When a ferrule is present, custom cast posts and cores exhibit a higher fracture resistance compared to composite cores built on prefabricated metallic posts or carbon posts.[98] Cast posts are also known to exhibit the least amount of retention and are associated with a higher failure rate compared to prefabricated parallel-sided posts. In a classic retrospective study (1 to 20 years) of 1273 endodontically treated teeth in general practice, 245 (19.2%) were restored with tapered cast posts and cores. Among these, 12.7% were deemed failures. This failure rate was higher than that for the other passive post systems used. Of particular concern was the fact that 39% of the failures led to unrestorable teeth requiring extraction. Thirty-six percent of the failures were due to loss of retention, and 58% were due to the fracture of the root. It has been suggested that tapered smooth-sided posts have a "wedging" effect under functional loading, and it is this that leads to increased risk of root fracture.[162]

One 6-year retrospective study reported a success rate higher than 90% using a cast post and core as a foundation restoration.[8] The lower failure rate and fewer root fractures were attributed to the presence of an adequate ferrule and careful tooth preparation. Attention has also been drawn to the fact that the higher failure rate may be due to the fact that nearly half of the posts were shorter than recommended from the literature. A venting groove for the cement along the axis of the post results in less stress on residual tissues.

Luting Cements

A variety of cements have been used to cement endodontic posts and include traditional cements, glass ionomer cements, and resin-based luting cements.

Traditional Cements

Zinc phosphate cements or polycarboxylate cements are still used for cementation of posts and crowns. They are generally supplied as a powder and a liquid, and their physical properties are highly influenced by the mixing ratio of the components. Their compressive strength is about 100 MPa, and elastic moduli are lower than that of dentin (5 to 12 GPa). Zinc phosphate cement is mostly used for cementing metal restorations and posts; film thickness of the zinc phosphate cement is less than 25 μm. These cements provide retention through mechanical means and have no chemical bond to the post or to dentin but provide clinically sufficient retention for posts in teeth with adequate tooth structure.

Glass Ionomer Luting Cements

Glass ionomer cements are a mixture of glass particles and polyacids, but resin monomers may also be added. Depending on the resin content, glass ionomer cements can be classified as either conventional or resin-modified glass ionomer cements. Conventional glass ionomer cements have compressive strengths ranging between 100 and 200 MPa; the Young modulus is generally about 5 GPa. They are mechanically more resistant than zinc phosphate cements, and they can bond to dentin with values ranging between 3 and 5 MPa. Some authors still recommend the use of glass ionomer cements for the cementation of metallic posts. Major advantages of conventional glass ionomer cements are their ease of manipulation, chemical setting, and ability to bond to both tooth and post. On the contrary, resin-modified glass ionomer cements are not indicated for post cementation, because these cements exhibit hygroscopic expansion that can promote fracture of the root.

Resin-Based Luting Cements

Today there is a trend toward the use of adhesive cements for bonding endodontic posts during the restoration of nonvital teeth. The rationale for using adhesive cements is based on the premise that bonding posts to root canal dentin will reinforce the tooth and help retain the post and the restoration.[48] Contemporary resin-based luting cements have been shown to exhibit compressive strengths around 200 MPa and elastic moduli between 4 and 10 GPa.[24] These materials may be polymerized through a chemical reaction, a photopolymerization process, or a combination of both mechanisms. Photopolymerization of these resin-based materials is often necessary to maximize strength and rigidity.

Most luting cements require a pretreatment of the root canal dentin with either etch-and-rinse or self-etching adhesives. Both types of adhesives have been shown to form hybrid layers along the walls of the post spaces.[10] However, bonding to root canal dentin may be compromised by the use of endodontic irrigants such as sodium hypochlorite, hydrogen peroxide, or their combination.[115] Because these chemicals are strong oxidizing agents, they leave behind an oxygen-rich layer on the dentin surface that inhibits the polymerization of the resin.[151] Previous research has shown that the bond strength of C&B Metabond to root canal dentin was reduced by half when the dentin was previously treated with 5% sodium hypochlorite (NaOCl) or 15% EDTA/10% urea peroxide (RC Prep, Premier Dental, Plymouth Meeting, PA).[107] Other reports indicate that the contamination of the dentin walls by eugenol diffusing from endodontic sealers may also affect the retention of bonded posts.[66,168] Further, it is difficult to control the amount of moisture left in a root canal after acid etching, making impregnation of collagen fibers with etch-and-rinse adhesives problematic. The use of self-etching adhesives has been proposed as an alternative for the cementation of endodontic posts, because self-etching adhesives are generally used on dry dentin and do not require rinsing of the etchant. However, their efficiency at infiltrating thick smear layers like those produced during post space preparation remains controversial.[104,171] More recently, dual-curing adhesives have been developed to ensure a better polymerization of the resin deep inside the root canal. Dual-cured adhesives contain ternary catalysts to offset the acid-base reaction between the acidic monomers and the basic amines along the composite/adhesive interface.[105]

Although both self-curing and light-curing luting cements can be used for cementation of prefabricated endodontic posts, most resin cements have a dual-curing process that requires light exposure to initiate the polymerization reaction. Dual-curing cements are preferred because there are concerns as to whether light-curing materials are properly cured, especially

in areas of difficult light access such as the apical portion of the root canal. However, it has been reported that photocured composites generate more shrinkage stress and exhibit less flow than chemically cured composites.[52] Contraction stresses induced by polymerization also depend on the geometry of the post space and the thickness of the resin film. Previous research indicates that the restriction of flow of resin cements by the configuration of the root canal can significantly increase the contraction stress at the adhesive interface.[51,166]

In recent years, a number of techniques have been used to measure the adhesion of resin-based luting cements to root canal dentin. These methods include the pull-out tests, the microtensile bond strength tests, and the push-out tests.[45,61] Although laboratory tests confirmed that bond strengths ranging between 10 and 15 MPa can be obtained with modern resin-based luting cement, there is also evidence that frictional retention is a factor contributing to post retention.[14] It is generally accepted that bonding to dentin of the pulp chamber is more reliable than bonding to root canal dentin, especially at the apical level.[126] The lowered bond strength values recorded at the apical third of the root canal are likely to be related to the reduced number of dentinal tubules available for dentin hybridization. Shorter posts may be used when successful bonding occurs between fiber-reinforced posts and root dentin, because current adhesive luting cements can assist in the retention of posts in the root canal space.[132]

Another factor that may influence the performance of resin-based luting cements is the thickness of the cement layer. The cementation of endodontic fiber posts with thicker cement layers might be required when posts do not perfectly fit inside the root canal. Although a slight increase in cement thickness (up to 150 microns) does not significantly affect the performance of adhesive luting cements applied to root canal dentin, thicker layers may be detrimental to bond quality.[76,150]

One study indicates that bond strength to radicular dentin might be maximized by adopting procedures that compensate for polymerization stresses.[13] The bonding procedures are realized in two separate steps. The initial step allows optimal resin film formation and polymerization along the root canal walls, leading to more ideal resin-dentin hybridization without stresses imposed by the placement of the post. A second step bonds the post to the cured resin film. The polymerization shrinkage that occurs during the initial adhesive coating step reduces the effects of stress imposed when the resin-coated post polymerizes, thereby preserving the bond integrity.

Although the bonding performance of resin-based luting cements is well documented, other reports indicate that resin-dentin bonds degrade over time.[22,56] The loss of bond strength and seal are attributed to the degradation of the hybrid layer created at the dentin-adhesive interface. This is particularly true for etch-and-rinse adhesives, because the gelatinization of collagen fibers caused by phosphoric acids may restrict the diffusion of the resin within the interfibrillar spaces and may leave unprotected fibers available for degradation. Removing organic components from the demineralized dentin prior to bonding procedures has been suggested. The use of dilute NaOCl (0.5%) after acid etching or the conditioning of dentin smear layers with EDTA (0.1 M, pH 7.4) has been shown to produce more durable resin-dentin bonds made with single-step etch-and-rinse adhesives.[149]

Other research indicates that the degradation of denuded collagen fibrils exposed in incompletely infiltrated hybrid layers is driven by an endogenous proteolytic mechanism involving the activity of matrix metalloproteinases (MMPs).[21,125] The release of MMPs such as collagenases has been evidenced in both coronal and root dentin of fully developed teeth of young patients.[147] Researchers suggest that conditioning root canal dentin with a broad-spectrum protease inhibitor such as chlorhexidine (2 wt% chlorhexidine digluconate solution) might be useful for the preservation of dentin bond strength over time.[20]

Interestingly, these dentin-conditioning procedures, which may improve the resistance of the resin-dentin bond to chemical degradation, also act as antibacterial agents; this might be of interest in the endodontic context.

Self-Adhesive Cements

More recently, self-adhesive resin cements have been introduced as an alternative to conventional resin-based luting cements. Self-adhesive luting cements contain multifunctional phosphoric acid methacrylates that react with hydroxyapatite and simultaneously demineralize and infiltrate dental hard tissue.[108] They do not require any pretreatment of the tooth substrates, and their clinical application is accomplished in a single step. Therefore, the self-etching capability of these new cements reduces the risk for incomplete impregnation of the conditioned tissue by the resins and reduces technique sensitivity. The elastic moduli of chemically cured self-adhesive resin cements are relatively low (4 to 8 GPa) but generally increase when a dual-curing process is used. It is therefore recommended that all dual-cured resin cements receive maximal light to achieve superior material properties wherever clinically possible.[135] Adhesion performance to dentin was found comparable to multistep luting cements, but bonding to enamel without prior phosphoric acid etching is not recommended.[72] However, their long-term clinical performances need to be assessed before making a general recommendation for their use.

PRETREATMENT EVALUATION AND TREATMENT STRATEGY

Before any therapy is initiated, the tooth must be thoroughly evaluated to ensure treatment success. Each tooth must be examined individually and in the context of its contribution to the overall treatment plan and rehabilitation. This assessment includes endodontic, periodontal, biomechanical, and aesthetic evaluations. Planning of the restoration for endodontically treated teeth brings together all aforementioned biomechanical and clinical factors, as well as the various materials and procedures designed to address them.

Pretreatment Evaluation

Endodontic Evaluation

The prerestorative examination should include an inspection of the quality of existing endodontic treatment. New restorations, particularly complex restorations, should not be placed on abutment teeth with a questionable endodontic prognosis. Endodontic retreatment is indicated for teeth showing radiographic signs of apical periodontitis or clinical symptoms of inflammation. Restorations that require a post need a post space, which is prepared by removal of gutta-percha from the canal. Canals obturated with a silver cone or other inappropriate filling material should be endodontically retreated before

starting any restorative therapy. Because the probability for periapical tissue to heal after endodontic retreatment is reasonably high, the chances to retain a well-restored tooth in asymptomatic function over time are excellent.[119]

Periodontal Evaluation

Maintenance of periodontal health is also critical to the long-term success of endodontically treated teeth. The periodontal condition of the tooth must therefore be determined before the start of endodontic therapy and restorative phase. The following conditions are to be considered as critical for treatment success:

- Healthy gingival tissue
- Normal bone architecture and attachment levels to favor periodontal health
- Maintenance of biologic width and ferrule effect before and after endodontic and restorative phases

If one or more of the aforementioned conditions are not met owing to preexisting pathology or structural defects, treatment success or even feasibility can be compromised, sometimes suggesting extraction of weak teeth and replacement with dental implants rather than conventional therapy.

Biomechanical Evaluation

All previous events, from initial decay or trauma to final root canal therapy, influence the biomechanical status of the tooth and the selection of restorative materials and procedures. The biomechanical status can even justify the decision to extract extremely mutilated teeth that do not deserve extensive treatments that carry a limited probability of success. Important clinical factors include the following:

- The amount and quality of remaining tooth structure
- The anatomic position of the tooth
- The occlusal forces on the tooth
- The restorative requirements of the tooth

Teeth with minimal remaining tooth structure are at increased risk for the following clinical complications[114,165,170] (see Fig. 22-1 and Fig. 22-4):

- Root fracture
- Coronal-apical leakage
- Recurrent caries
- Dislodgment or loss of the core/prosthesis
- Periodontal injury from biologic width invasion

The amount and quality of remaining tooth substrate are far more important to the long-term prognosis of the restored tooth than any restorative material properties. One must consider that no restorative material can truly substitute for dentin or enamel and that a minimal amount of intact structure is mandatory to justify tooth maintenance and its strategic importance to the overall treatment plan. The presence of healthy adjacent teeth available as abutments or the option of dental implants are additional factors to be analyzed when justifying the restoration of endodontically treated teeth.

FIG. 22-4 Failure of prosthetic foundations can have dramatic consequences on both overlying restorations and surrounding tissues. A better understanding of compositional and structural changes that affect tooth resistance to repeated functional forces is mandatory to improve treatment success in endodontically treated teeth.

Tooth Position, Occlusal Forces, and Parafunctions

Teeth are subjected to cyclic axial and nonaxial forces. The teeth and associated restorations must resist these forces to limit potential damages such as wear or fracture. The degree and direction of forces depend on the location of the tooth in the arch, the occlusal scheme, and the patient's functional status.

In most occlusal schemes, anterior teeth protect posterior teeth from lateral forces through anterolateral guidance. In the context of very steep anterior guidance and deep vertical overbite, maxillary anterior teeth are sustaining higher protrusive and lateral forces from the mandibular anterior teeth. Restorations of damaged anterior teeth with heavy function should therefore be designed to resist flexion. Restorative components should be stronger than would be required for teeth with an edge-to-edge relationship and therefore vertical forces.

Posterior teeth normally carry more vertical forces, especially when maintaining canine and anterior guidance; they also sustain greater occlusal loads than anterior teeth, and restorations must be planned to protect posterior teeth against fracture. In the case of parafunctions, protection by anterior contacts is likely to be reduced or lost and posterior teeth then submitted to more lateral stresses, generating a higher demand for restorative materials.

The literature reports that average biting forces vary between 25 and 75 N in the anterior region and between 40 and 125 N for the posterior region of the mouth, depending on food type, dental status (dentate or edentulous), and patient anatomy and functional habits.[55,71] Those forces can easily reach 1000 N or above in case of parafunctions, showing how potentially destructive they can be for intact teeth and even more so for nonvital, fragilized teeth. Parafunctional habits (clenching and bruxism) are major causes of fatigue or traumatic injury to teeth, including wear, cracks, and fractures. Teeth that show extensive wear or sequelae from parafunctions, especially heavy lateral function, require components with the highest physical properties to protect restored teeth against fracture.

In general, modern strategy focuses on tissue preservation and also on the use of adhesion to stabilize the restoration for improved short- and long-term service. However, in certain conditions like reduced tooth support, conventional materials are not obsolete.

Aesthetic Evaluation and Requirements

Anterior teeth, premolars, and often the maxillary first molar, along with the surrounding gingiva, compose the aesthetic zone of the mouth. Changes in the color or translucency of the visible tooth structure, along with thin soft tissues or

TABLE 22-2

Clinical Protocols for Restoring Nonvital Teeth with Partial Restorations (Most Likely Procedures)

Treatment Approach	Indications	Tooth Preparation (Critical Guidelines)	Interface Treatment		Restoration Fabrication
			Tooth	Restoration	
Composite restoration	Minimal tissue loss	None	DBA	—	Direct multilayer
Veneer	Limited tissue loss	≥ 1 mm Buccal reduction, lingual enamel present, minimal to moderate discoloration only	DBA	1. Sandblasting or etching 2. Silane 3. Bonding resin	CP direct multilayer *or* *In laboratory:* Etchable CER: fired, pressed, or CAD-CAM
Overlay (composite/ ceramics)	Thin remaining walls	Minimum 2 mm occlusal reduction	DBA + composite lining	1. Sandblasting or etching 2. Silane 3. Bonding resin	*In laboratory:* CP: hand-shaped, light or heat cured, CAD-CAM Etchable CER: fired, pressed, or CAD-CAM
Endocrown (composite/ ceramics)	Loss of occlusal anatomy	Minimum 2 mm occlusal reduction, extension into pulpal chamber	DBA + composite lining	1. Sandblasting or etching 2. Silane 3. Bonding resin	*In laboratory:* CP: hand-shaped, light or heat cured, CAD-CAM Etchable CER: fired, pressed, or CAD-CAM

CAD-CAM, computer-aided design/computer-aided machined; *CER,* ceramic; *CP,* composite; *DBA,* dentin bonding agent.

biotype, diminish the chance for a successful aesthetic treatment outcome.

Potential aesthetic complications should be investigated before endodontic therapy is initiated. For instance, metal or dark carbon fiber posts or amalgam placed in the pulpal chamber can result in unacceptable aesthetic results, such as a grayish appearance of the overlying prosthetic restoration (especially with modern, more translucent full-ceramic crowns) or gingival discoloration from the underlying cervical area or root (see Fig. 22-1). All teeth located in the aesthetic zone also require critical control of endodontic filling materials in the coronal third of the canal and the pulp chamber to avoid or reduce the risk of discoloration. Careful selection of restorative materials, careful handling of tissues, and timely endodontic intervention are important for preserving the natural appearance of nonvital teeth and gingiva.

Treatment Strategy

General Principles and Guidelines

The post, the core, and their luting or bonding agents together form the *foundation restoration* to support the coronal restoration of endodontically treated teeth. The evolution of foundation restorations has been to diminish invasiveness, to use adhesion rather than macromechanical anchorage, and to eliminate intraradicular components in selected cases. These changing clinical concepts derive from both an improved understanding of tooth biomechanics and advances in restorative materials.

The foundation and its different constituents are then aimed at providing the best protection against leakage-related caries, fracture, or restoration dislodgment. Therefore, all aforementioned local and general parameters are to be systematically analyzed in order to select the best treatment approach and restorative materials. Prosthetic requirements are also to be taken into consideration to complete each case analysis. In general, abutments for fixed or removable partial dentures clearly dictate more extensive protective and retentive features than do single crowns, owing to greater transverse and torquing forces. This modern biomechanical treatment strategy is summarized in Figure 22-5.

Structurally Sound Anterior Teeth

Anterior teeth can lose vitality as a result of a trauma with no or minimal structural damage. They generally do not require a crown, core, or post; restorative treatment is limited to sealing the access cavity and direct composite fillings. Discoloration, whenever present, is addressed by nonvital bleaching, or, for untreatable or relapsing ones, with conservative restorative approaches such as direct or indirect veneers (Table 22-2).

Nonvital Posterior Teeth with Minimal/ Reduced Tissue Loss

The loss of vitality in posterior teeth resulting from trauma, decay, or a restorative procedure does not necessarily lead to extreme biomechanical involvement and therefore allows, in certain conditions, for conservative restorations.

Occlusal cavities or mesio/disto-occlusal cavities can be restored with either direct- or indirect-adhesive intracoronal restorations, providing residual walls are thick enough (proximal ridges and buccolingual walls more than 1.5-mm thickness). The three additional clinical factors that must be analyzed to ensure optimal treatment success are the configuration factor (C-factor), cavity volume, and dentin quality. For instance, a large class I cavity with contaminated and sclerotic dentin would clearly be a contraindication to the direct approach, despite the fact that it apparently falls within the indications of direct techniques. Conservative options must, however, always be analyzed under the light of functional and occlusal environment. They can only be considered in the absence of parafunctions and with anterior guidance, which limits overall functional loading and lateral or flexural forces.

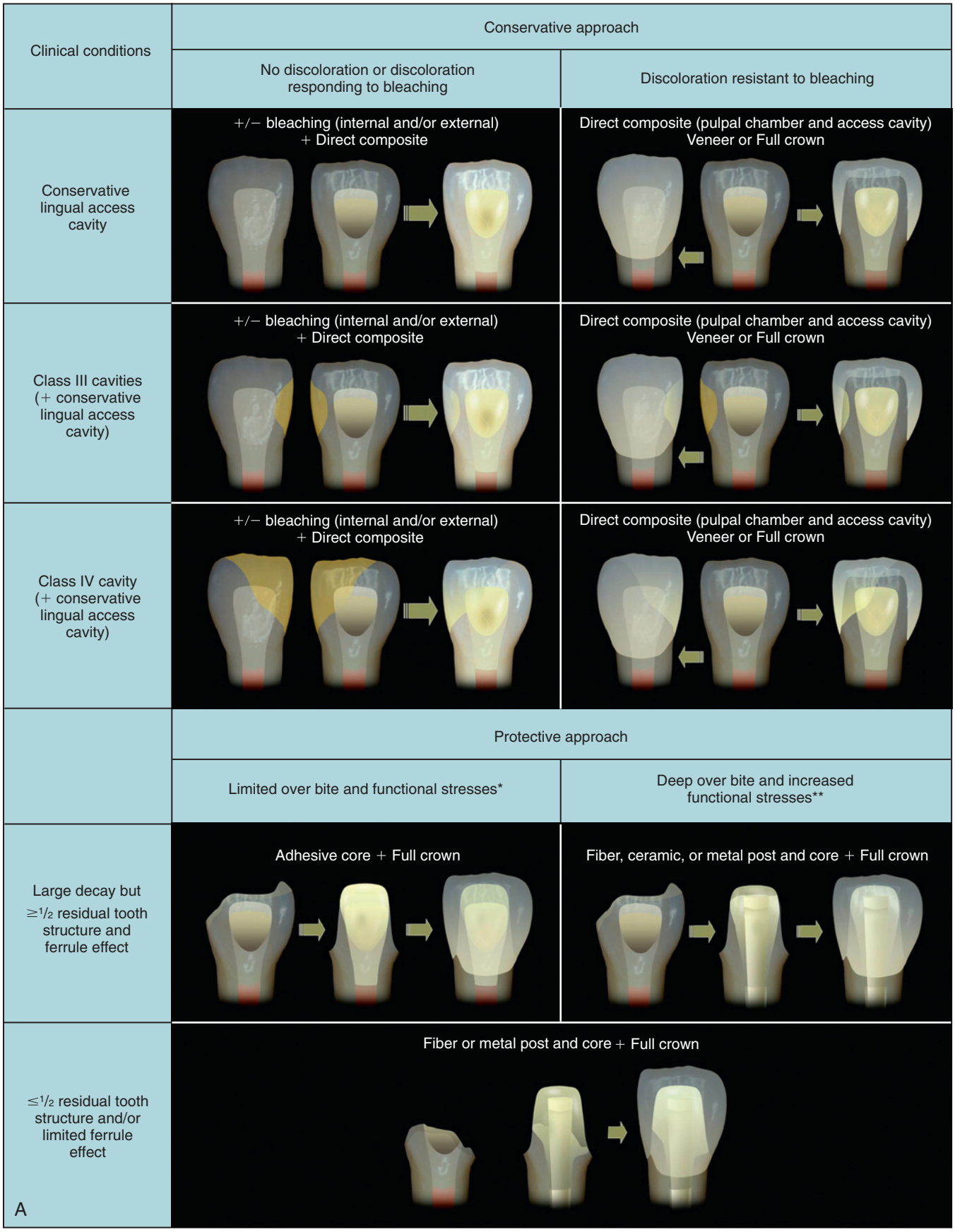

FIG. 22-5 A, Current recommendations for the treatment of nonvital anterior teeth. *Normal function and anterior guidance; **moderate to severe parafunctions and abnormal occlusion/anterior guidance.

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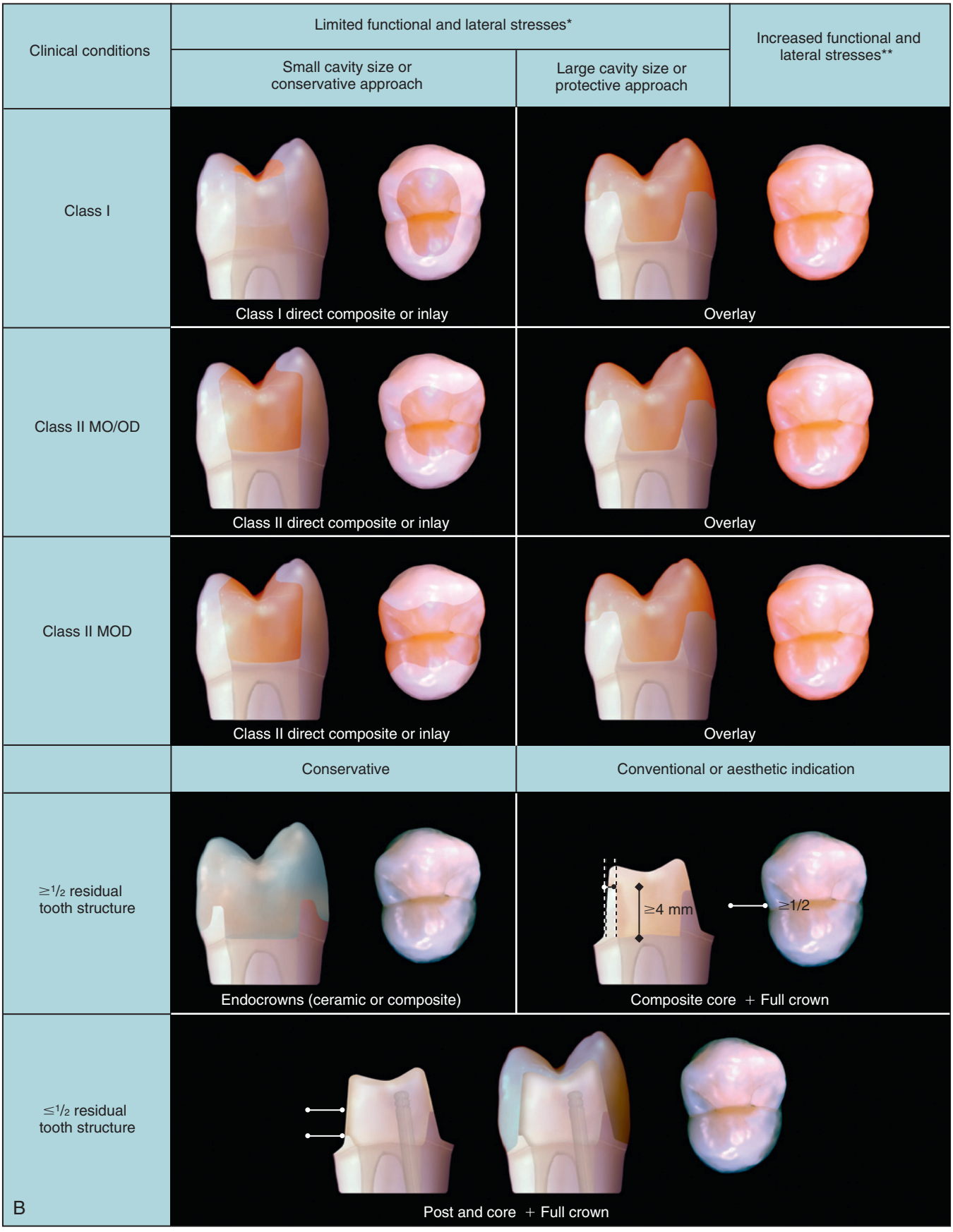

FIG. 22-5, cont'd B, Current recommendations for the treatment of nonvital posterior teeth. *Relatively flat anatomy and canine guidance, normal function; **group guidance, steep occlusal anatomy, parafunctions.

In less favorable biomechanical conditions (e.g.,) group guidance, steep occlusal anatomy, bruxism, clenching a protective approach with full occlusal coverage (onlay or overlay) is mandated to minimize the risk of fatigue failures (see Table 22-2).

Structurally Compromised Teeth

The decision for placing a post as well as the selection of a post system (rigid or nonrigid) depends once again on the amount and quality of remaining tooth structure and the anticipated forces sustained by this tooth (Table 22-3).

In general, rigid posts made of stiff materials (metal and ceramics) are indicated for teeth with minimal tooth structure that rely on the post to hold the core and crown. Because rigid posts flex and bend less than other types of posts, they are supposed to limit movement of the core and possible disruption of the crown margins and cement seal. But one must remember that stiff posts transmit more stresses to the root, next to the post apex, when conventionally cemented. An attempt to strengthen a weak root by adding a stiff post can instead make the root weaker as a result of the force concentration behavior of a stiff rod in a more flexible material. Adhesion therefore plays a crucial role, because a well-bonded post can help absorb stresses more evenly throughout the remaining tooth structure. Benefit and increased risk of fissure and

TABLE 22-3

Clinical Protocols for Restoring Nonvital Teeth with Full Prosthetic Restorations (Most Likely Procedures)

Treatment Approach	Indications	Tooth Preparation (Critical Guidelines)	Foundation		Restoration	
			Post	Core	Fabrication	Luting
Composite core	Reduced walls but > ½ crown height	Maintain all residual structures > 1 mm thickness (after core prep.)	—	DBA + composite Dual or LC, incremental	*In laboratory:* PFM or full ceramic restoration: slip-casting, pressed, or CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement
Composite core + ceramic post	More than ½ coronal structure lost, reduced wall height	Maintain all residual structures > 1 mm thickness (after core prep.)	Sandblasting or coating/silane + DBA + Dual cement or SA cement	DBA + composite Dual or LC, incremental	*In laboratory:* PFM or full ceramic restoration: slip-casting, pressed, or CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement
Composite core + in vitro fiber post	More than ½ coronal structure lost, reduced wall height	Maintain all residual structures > 1 mm thickness (after core prep.)	Sandblasting or coating/silane + DBA + Dual cement or SA cement	DBA + composite Dual or LC, incremental	*In laboratory:* PFM or full ceramic restoration: slip-casting, pressed, or CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement
Composite core + metal post	More than ⅔ coronal structure lost, reduced wall height	Maintain all residual structures > 1 mm thickness (after core prep.)	Sandblasting or coating/silane + DBA + Dual cement or SA cement	DBA + composite Dual or LC, incremental	*In laboratory:* PFM or full ceramic restoration: slip-casting, pressed, or CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement
Amalgam core (+/– metal post)	Alternative to composite core with metal post	Maintain all residual structures > 1 mm thickness (after core prep.)	No tt + nonadhesive cement or sandblasting/ coating/silane + DBA + Dual cement or SA cement	Amalgam placement in retentive cavity/ preparation	*In laboratory:* PFM restoration	Coating, sandblasting, or etching + silane and Dual or SA cement
Cast gold post and core (+/– porcelain)	More than ¾ coronal structure lost	Maintain all residual structures > 1 mm thickness (after core prep.) Internal walls are divergent	No tt/sandblasting + nonadhesive cement or sandblasting/ coating/silane + DBA + Dual cement or SA cement	No tt + nonadhesive cement or DBA + Dual cement or SA cement	*In laboratory:* PFM or full ceramic restoration: Zirconia/ CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement

CAD-CAM, computer-aided design/computer-aided machined; *CER,* ceramic; *DBA,* dentin bonding agent; *Dual,* dual curing; *LC,* light curing; *PFM,* porcelain fused to metal; *SA,* self-adhesive.

fracture must then be appropriately weighed against adhesion potential inside the root and post type, composition, and surface treatment.

In structurally sound teeth, nonrigid posts flex with the tooth under functional forces, reducing the transfer of force to the root and reducing the risk of root fracture. Flexion is of course related to post diameter. In structurally compromised teeth, which lack cervical stiffness from dentin and ferrule effect, excessive post flexion can be detrimental to the marginal seal and prosthesis longevity, so fiber posts are generally contraindicated.

White or translucent fiber posts are generally preferred underneath full-ceramic restorations, whereas stronger black carbon fiber posts, which can reflect through gingiva, tooth structure, or ceramic restorations, are usually used in teeth to be restored with gold or porcelain fused to metal crowns, as well as in zirconia-based restorations. The literature has largely overemphasized the impact of post color on restoration aesthetics. Metal or carbon post color can be masked with resin opaquer and gold post and core ceramized to enhance aesthetic integration. Such procedures can help to approach a more ideal restoration biomechanical behavior through the fabrication of rigid but more aesthetic foundations. Upper lateral and lower incisors, together with extremely thin biotype, are probably the only real aesthetic contraindication for metal or carbon fiber posts.

In cases of extreme tooth fragility secondary to caries, fracture, previous overenlargement of the root canal system, or immaturity, residual root structure can be unified and reinforced with adhesive bonding and composite before placing a normal-diameter post, forming an altogether cohesive unit, as previously described.

In conclusion, in a damaged tooth that is to be restored with a nonrigid post, 2 to 3 mm of cervical tooth structure must ideally remain to allow creation of a restoration as a whole that is resistant to flexion. Teeth with minimal tooth structure and limited ferrule effect need additional cervical stiffness from a more rigid post to resist distortion. In this situation, adhesive cementation is preferred to conventional cementation.

Structurally Compromised Anterior Teeth

Restoration of endodontically treated teeth becomes more complex as teeth or supporting structures become increasingly affected. A nonvital anterior tooth that has lost significant tooth structure requires restoration with a crown, supported and retained by a core and possibly a post as well.

When less than half the core height is present, or when remaining walls are extremely thin (less than 1 mm on more than three fourths of the tooth circumference), a post is needed to increase retention and stabilize and reinforce the foundation. Many post options are available nowadays, including titanium, fiber-reinforced resin, and ceramics. Adhesion is now the preferred mode of post cementation unless a long-term contamination of root dentin is obvious (e.g., with eugenol), making adhesion highly questionable.

In the latter situation or in the presence of flared canals (possibly as well when a limited ferrule effect is present), cast gold post and core are still considered a feasible option. Actually, in this extremely unfavorable biomechanical environment, this traditional treatment approach provides a higher rigidity in the cervical area, which is mandatory for restoration stability. Here, a fiber-reinforced composite foundation having higher flexibility might present less favorable biomechanical behavior, such as suggested by FEM studies[42] (see Fig. 22-3 and Table 22-2). Tooth extraction and implant placement or bonded bridge (particularly for lateral incisors) are also to be considered in this situation.

In the aesthetic zone, the post should not detract from the aesthetics of the coronal tooth structure, ceramic crown, or gingiva. Current restorative procedures allow fabrication of highly aesthetic ceramic coronal restorations that have no metal substructure. When such restorations with remarkably lifelike color and vitality are selected, it usually implies the use of nonmetal aesthetic posts, either ceramic or resin fiber-reinforced ones.

Structurally Compromised Posterior Teeth

Slightly decayed posterior teeth in the context of parafunctions or significantly fragilized premolars and molars require cuspal protection afforded by onlay restoration, endocrown, or a full crown. The need for a post and core depends on the amount of remaining tooth structure. When remaining walls (buccal and lingual) provide more than 3 to 4 mm height (from the pulpal chamber floor) and 1.5 to 2 mm thickness, core and restoration stability are granted through macromechanical retention or adhesion; then, posts are not needed (see Fig. 22-5). With current treatment strategy, *the post has become the exception rather than the rule for the restoration of nonvital posterior teeth*.

Additional Procedures

Periodontal crown lengthening surgery or orthodontic extrusion can expose additional root structure to allow restoration of a severely damaged tooth. In the smile frame, crown lengthening might, however, be limited by aesthetically adverse consequences (proximal attachment reduction); basically, buccal crown lengthening only can be considered as a potential indication for this procedure. In the posterior region, crown lengthening is limited by tooth and furcation anatomy or by loss of bone structure, which complicates future implant placement. As regards to orthodontic extrusion, root length and anatomy are the limiting factors of this procedure; short roots or conical anatomy are contraindications to orthodontic extrusion. Once again, when a long-lasting, functional restoration cannot be predictably created, it might be better to extract the tooth than to pursue heroic efforts to restore an extremely weak tooth, using complex, expensive, and unpredictable procedures.

CLINICAL PROCEDURES

The restoration of a nonvital tooth may include several restorative components such as the post, the core, and the overlying restoration. Within the same tooth, several interfaces such as post to radicular dentin, core to coronal dentin, core to post, and core to overlying restoration will be created. According to the biomechanical status, some or all restorative components and interfaces will be present and need to be addressed.

General clinical guidelines for restoring endodontically treated teeth with either partial or full crown restorations are presented in Table 22-2, with specific procedures for tooth preparation and treatment of the different interfaces involved. Clinical steps for all recommended treatment options are presented in Figures 22-6 through 22-14.

FIG. 22-6 Direct composite restorations on front teeth (following bleaching). Nonvital anterior tooth with sufficient amount of residual tissue and intact, healthy neighboring teeth, usually in rather young patients. **A,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha were removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. **B,** Bleaching procedures. Two sessions of internal bleaching were needed to adapt the color of both traumatized anterior teeth (tooth #11), using a mixture of 3% hydrogen peroxide and sodium perborate powder. Each bleaching session extended over a 10-day period. Between sessions and until the given delay to proceed with final restoration (1 to 2 weeks up to 6 to 8 weeks), access cavity is closed with either a temporary filling material or flowable resin composite. **C, D,** Restorative procedures. A layer of adhesive is applied over all cavity surfaces. An etch-and-rinse system is frequently preferred when the dentinal tissue is highly sclerotic or was contaminated (e.g., by eugenol). A hard cement such as glass ionomer (not a resin-modified glass ionomer because of water uptake and further expansion) was used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. Two or three layers are usually used to replace the missing tissue, preferably following the "natural layering concept," which aims to build up the restoration with two basic masses, namely dentin and enamel, with the help of a silicone index. **E, F,** Postoperative views. The two conservative restorations are shown after finishing and polishing, with the rubber dam still in place **(E)** and after several weeks **(F)**. Note that aesthetic integration cannot be evaluated before full rehydration has occurred (4 to 6 hours at least).

FIG. 22-7 Direct composite restoration on posterior tooth. Nonvital posterior tooth with sufficient amount of residual tissue and mostly intact, rather healthy, neighboring teeth. No significant discoloration of buccal tooth substrate and absence of severe parafunctions or group guidance or other unfavorable occluso-functional condition. **A**, Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for a later placement of a post. **B**, Adhesive procedure. An etch-and-rinse adhesive system was used here owing to the presence of sclerotic and contaminated dentin. **C-E**, Restoration base and layering technique. Resins or glass ionomer cements can be used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. A dentin composite mass is used to replace missing dentinal tissue, and enamel composite is used to build up proximal walls and the occlusal anatomy. **F**, The postoperative view demonstrates the advantage of this conservative approach in the context of teeth with appropriate biomechanical status.

FIG. 22-8 Veneer. Nonvital anterior tooth with sufficient amount of residual tissue and rather healthy neighboring teeth. Discoloration resisted bleaching or relapsed, despite several retreatments. Preferably thick biotype to limit the risk of gingival recession and loss of aesthetic integration. **A,** Closing the lingual cavity. A hard cement such as glass ionomer (not resin-modified glass ionomer because of water uptake and possible expansion) can be used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. **B,** Preparation. A sufficient amount of buccal tooth structure must be removed to allow for a good aesthetic result (preparation depth is dictated by the discoloration severity). Cervical margins closely follow gingival contours, preventing soft-tissue impingement, but they assume complete coverage of discolored tooth structure. **C, D,** Adhesive procedure. Hydrophobic bonding systems are preferred to wet-etched enamel when no dentin is exposed. An etch-and-rinse adhesive system is preferred when dentin is exposed (here, in the presence of significant areas of sclerotic dentin). Restoration. Ceramic is etched and silanated. Just before cementation, a last layer of bonding resin is applied but not light-cured until placement. **E,** Luting technique. A light-curing, highly filled, translucent and fluorescent restorative material is preferably used for cementation. This allows better control of restoration placement and minimal wear and fatigue of the cement layer. **F,** Postoperative view. The postoperative view demonstrates satisfactory aesthetics. This approach is sometimes suitable to meet a patient's high aesthetic demands.

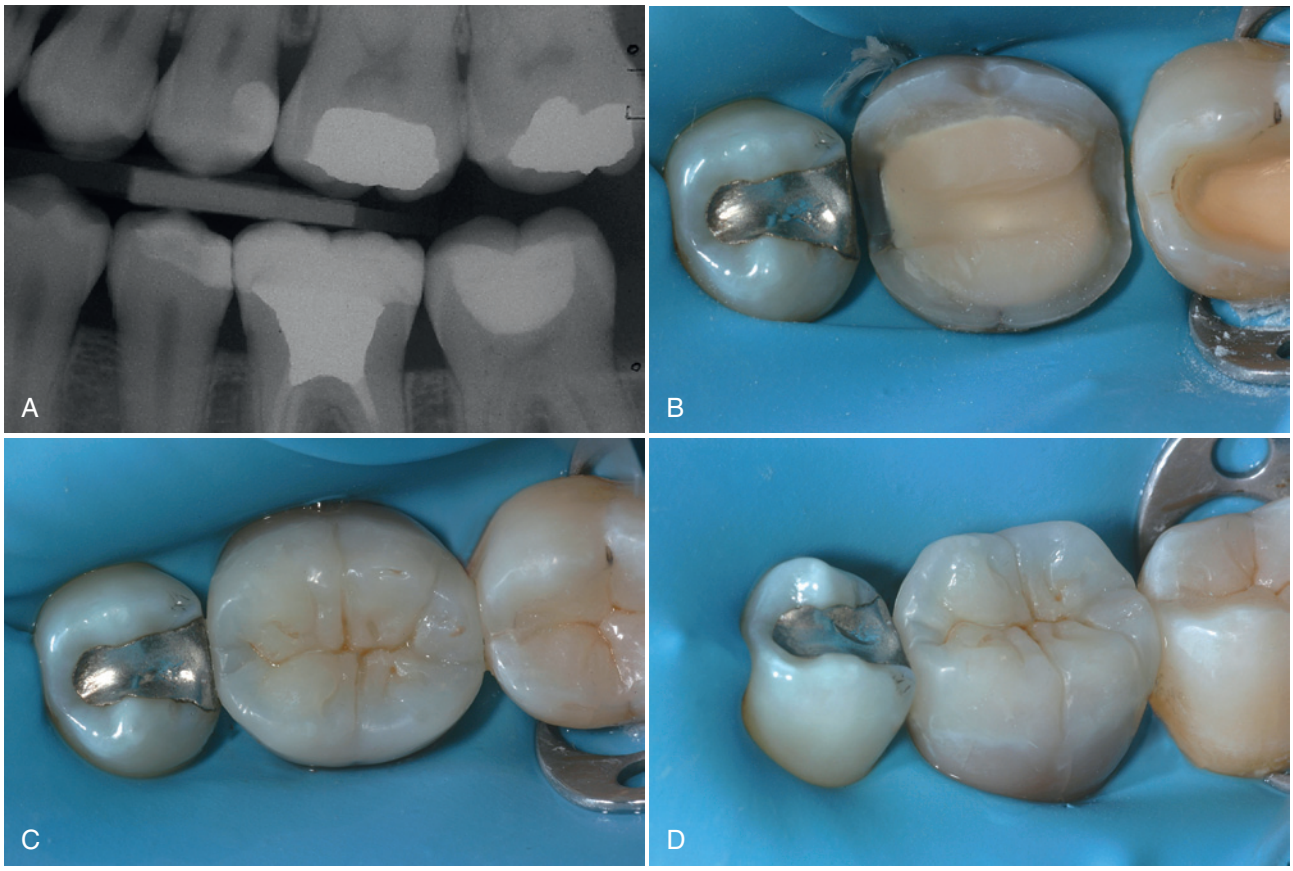

FIG. 22-9 Onlay/overlay. Nonvital posterior tooth with sufficient amount of residual tissue to justify partial restoration. Preparation involves both proximal surfaces or only two surfaces, but in a less favorable occluso-functional environment (thin walls). No significant discoloration of visible buccal tooth substrate. Option is favorable for short clinical crowns. **A, B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. Thin and weak occlusal walls are removed until reaching sufficient thickness for the restoration (1.5 to 2 mm in occlusal-free and occlusal areas, respectively). Adhesive procedure: an etch-and-rinse adhesive system is preferably used here owing to the presence of sclerotic and contaminated dentin (a self-etch adhesive system can also be used). A resin composite base is made with a restorative composite or preferably with flowable composite (for limited volume and thickness) to smooth internal angles, fill undercuts, and, when needed, relocate cervical margins occlusally. **C, D,** After impression and model fabrication, the restoration is generated in the laboratory to optimize restoration anatomy, function, and aesthetics. The restoration can be made of different tooth-colored materials: ceramics (fired, pressed, or CAD-CAM generated) or resin composite. This treatment option provides optimal tissue conservation, function, and aesthetics and is a good alternative to full crowns.

Tooth Preparation

The most important part of the restored tooth is the tooth itself. As described earlier, thickness and height of remaining dentin walls or cusps along with functional occlusal conditions are the determining factors in choosing the most appropriate restorative solution.

For partial intracoronal restorations, maximal tissue conservation is the only consideration for the clinician. In other situations, the selected restorative approach will usually necessitate some tooth preparation to comply with restoration design and thickness. Onlays, overlays, and endocrowns require about 1.5 to 2 mm occlusal space to guarantee restoration resistance to functional loads. For full crowns, a ferrule is needed to encircle the vertical walls of sound tooth structure above the restoration margin (1.5 to 3 mm), thus preventing a coincidence between core and restoration limits. Other preparation requirements presented in this chapter are to be respected to ensure treatment success. This means that a 4- to 5-mm height and 1-mm thickness of sound, suprabony tooth structure should be available to accommodate both the periodontal biologic width and the restorative ferrule.

Post Placement

The post is an extension of the foundation into the root of structurally damaged teeth, needed for core and coronal restoration stability and retention. The post is cemented or bonded into the root, according to tissue quality and post and core choice. The post performs both a mechanical and

FIG. 22-10 Endocrown. Nonvital posterior tooth with reduced amount of residual cervical tissue but sufficient tissue to provide supragingival restoration margins and good stabilization within former pulpal chamber. No or limited discoloration in visible buccal tooth substrate. Favorable option for short clinical crowns. **A, B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. Thin and weak walls are removed until reaching sufficient thickness (1.5 mm minimum); as much residual cervical structure as possible is then maintained to avoid restoration interference with periodontal tissues. **C,** Prosthetic restoration on working model. **D,** Restoration base. Adhesive procedures: An etch-and-rinse adhesive system is preferably used here owing to the presence of sclerotic and contaminated dentin (a self-etch adhesive system can also be used). Restoration base: A resin composite base is made with a restorative composite or preferably with flowable composite (for limited volume and thickness) to smooth internal angles, fill undercuts, and, when needed, relocate cervical margins occlusally. **E,** Impression and restoration fabrication. After impression and model fabrication, the restoration is generated in the laboratory to optimize restoration anatomy, function, and aesthetics. The restoration can be made of different tooth-colored materials: ceramics (fired, pressed, or CAD-CAM generated) or resin composite. **F,** Postoperative result. Despite their relatively rare application, endocrowns combine biomechanical advantages with facilitation of clinical procedures and minimal if any involvement of biologic width. It is an interesting alternative to full crowns. (From Rocca GT, Bouillaguet S: Alternative treatments for the restoration of nonvital teeth, *Rev Odont Stomat* 37:259, 2008.)

FIG. 22-11 Amalgam core. Nonvital posterior teeth with reduced tooth structure and wall height. Amalgam core stability is granted by extension of the restoration into the coronal portion of the canal (curved roots) and post (only if straight root anatomy) and remaining pulpal chamber undercuts. Today, this option is considered obsolete or historical because of the intrinsic staining of amalgam, the nonadhesive technique, and the less-than-ideal biomechanical behavior of amalgam. **A, B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Thin and weak walls are removed until reaching sufficient thickness (1.5 to 2 mm); existing retentions are used to stabilize the amalgam core, together with a post. The metal post (with passive and anatomic design, preferably made of titanium) extends into the root for about the same length as its coronal extension. **C-E,** Restoration fabrication. After placing a matrix or cupper ring to envelop the preparation, amalgam is compacted into the cavity, superior root canal portion, and around the post until the appropriate coronal volume is restored. Preparation of cores is made to manage optimal space for porcelain fused to metal (PFM) crowns. **F,** Posttreatment result. This treatment approach is feasible for teeth already restored with amalgam cores and when PFM restorations are mandated. Satisfactory aesthetics can be attained with intrasulcular restoration margins.

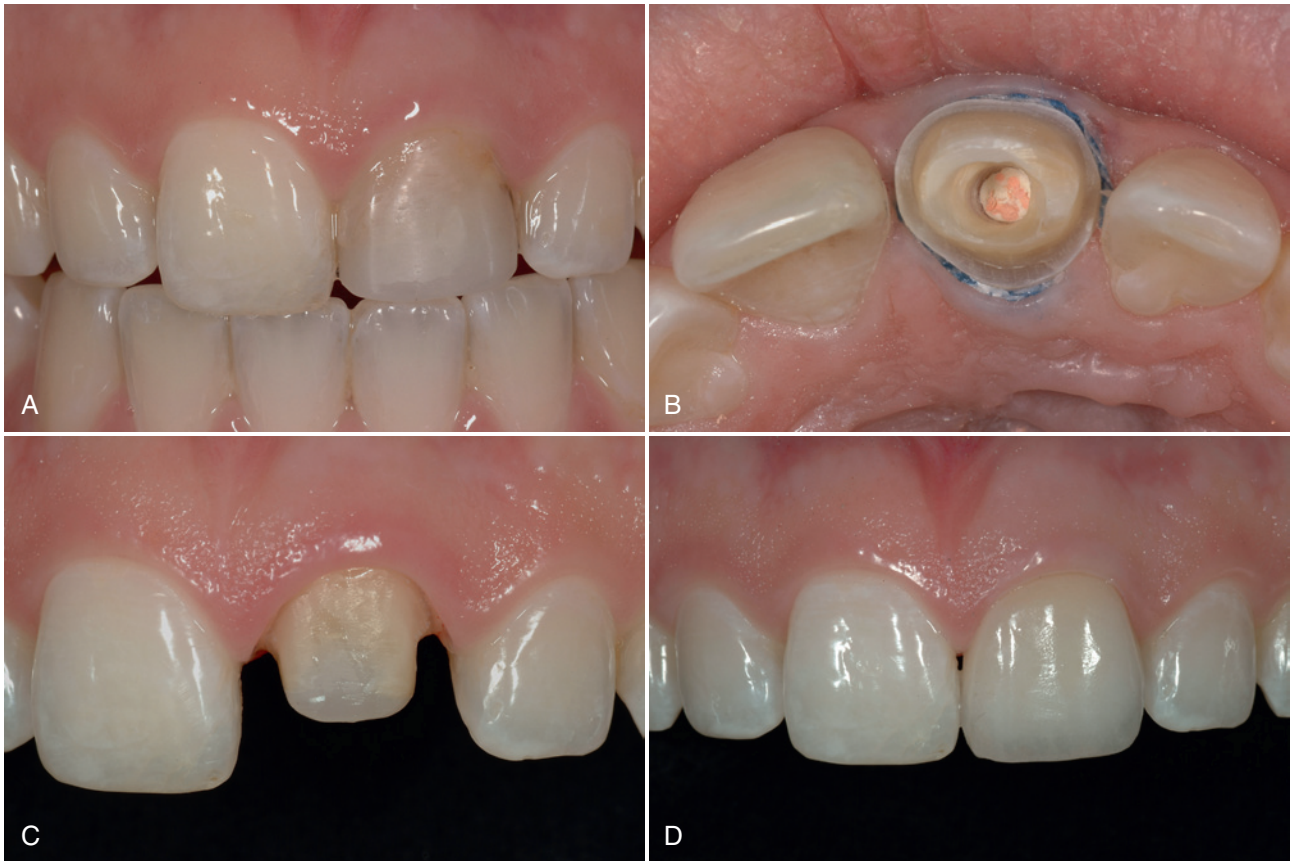

FIG. 22-12 Composite core (without post). Nonvital tooth with more than half the coronal structure left and sufficient wall thickness. Core stability is granted by adhesion, remaining pulpal chamber, and residual wall height. **A, B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. The tooth is prepared to evaluate precisely the height and thickness of residual tooth structure. Half of the abutment height and more than 1.5 mm wall thickness must be present to restore the tooth without a post. If aforementioned conditions are met, a thin layer of hard cement (calcium hydroxide, zinc phosphate, or glass ionomer; not resin-modified glass ionomer) is applied at the entrance of the root canal to enable possible retreatment in the future. **C,** Adhesive procedures and core fabrication. All walls are covered with the adhesive system (etch-and-rinse or self-etch) before applying in layers a direct light-curing restorative composite or a self-curing material, in case of limited restoration volume. **D,** Postoperative view. The advantage of this approach is the ease of application and excellent aesthetics. It is considered a good restorative solution for highly aesthetic ceramic crowns placed on teeth with limited tissue loss and discoloration.

biologic function by protecting the apical seal from bacterial contamination in case of coronal leakage. These functions should not be attained at the expense of tooth strength; post space preparation should be as conservative as possible to avoid an increased risk of root fracture. It is important to point out again that a post does not strengthen or reinforce a tooth. This elusive goal would only be achieved in a case of perfect cohesion between post and tooth substrate, nowadays only partially achievable. Therefore, the clinician must keep in mind that inherent strength of the tooth and its resistance to root fracture comes mostly from the remaining tooth structure and the surrounding alveolar bone. The tooth is weakened if dentin is sacrificed to place a larger-diameter post.

The post should be long enough to meet aforementioned biomechanical demands without jeopardizing root integrity. The standard parameters for post placement in a tooth with normal periodontal support are as follows:

1. In case of nonadhesive cementation (metal posts only)[60]:
   - Two thirds the length of the canal
   - A radicular extension at least equal to the coronal length of the core
   - One half the bone-supported length of the root
2. In case of adhesive cementation (fiber posts)[41,136]:
   - One third to one half the length of the canal, maximum
   - A radicular extension about the coronal length of the core

The first step for all types of post and core restorations is removal of gutta-percha and endodontic cement from the future post space. This procedure generally is best accomplished by the clinician providing the endodontic service, because that person has a clear knowledge of the size and form

FIG. 22-13 Composite core with white post (resin-reinforced fiber or zirconia post). Nonvital tooth with less than half the coronal structure left or insufficient wall thickness. Core stability is improved through adhesion and a tooth-colored post (resin-reinforced fiber or ceramic posts). **A,** Preoperative view. **B, C,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. The tooth is prepared to evaluate precisely the height and thickness of residual tooth structure. If less than half of the abutment height or 1.5-mm wall thickness remain, the abutment requires a post to increase core retention and foundation stability. **D, E,** Post-and-core fabrication. Post placement: The post space is prepared with ad hoc instruments until reaching a length equivalent to core height or slightly less depending on remaining wall height. Increased length is not needed, because retention is predominantly achieved through adhesion. Any tooth-colored post can be used, but fiber posts are usually preferred to limit stress buildup into the root structure, even though they provide less rigidity to the foundation. Post cementation: A self-curing adhesive system is applied on all cavity walls and into post preparation before luting the post with a dual-cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). Core fabrication: The remaining core volume is built up in layers, using a direct light-curing restorative composite or with a single increment of a self-curing material when only limited volume is to be completed. **F,** Postoperative view. The primary advantage of this approach is the excellent aesthetic outcome. It is considered a good restorative solution for highly aesthetic ceramic crowns placed on teeth with more tissue loss but moderate discoloration.

FIG. 22-14 Composite core with prefabricated metal post. Nonvital tooth with reduced coronal structure and ferrule effect or insufficient wall thickness. Core stability is improved through adhesion and a metal (opacified) post (titanium posts are usually preferred). **A,** Preoperative view. **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose the remaining tooth structure and a clean dentin substrate. The absence of coronal tissue and ferrule effect mandates the use of a more rigid post (ceramic posts are considered too rigid and contraindicated in this situation). **C-F,** Post and core fabrication. Post space preparation: The post space is prepared with ad hoc instruments until reaching a length equivalent to about core height. More length is not needed, because retention is predominantly achieved through adhesion. Post preparation: The metal post (titanium here) is sandblasted (silicoating can be used to increase adhesion; i.e., Rocatec or CoJet systems, 3M), followed by silane application. An opaque resin or flowable composite can be used to mask metal dark color and improve aesthetics. Post cementation: A self-curing adhesive system is applied on all cavity walls and into post preparation before luting the post with a dual-cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). Core fabrication: The remaining core volume is built up in layers, using a direct light-curing restorative composite or a single increment of a self-curing material when only limited volume is to be completed.

of the canal system. The initial post space preparation procedure is similar for most standardized post and core systems. The space, cleared of gutta-percha, has the form of the canal after cleaning and shaping. Proprietary systems are supplied with a series of drills to prepare the internal surface of the canal. The goal of post space formation is to remove little or no dentin from the root canal.

Adhesive Procedures

In general, both self-etch and etch-and-rinse adhesive systems can be used successfully on root dentin, both systems having a well-documented proof of efficacy. However, the use of etch-and-rinse adhesive systems may be advantageous in the presence of highly sclerotic or contaminated dentin, because phosphoric acids will etch dentin more deeply than self-etching primers. When treating the canal in the perspective of post cementation, a dual-curing adhesive system is preferably used to optimize polymerization. Self-adhesive cements have also demonstrated satisfactory performance for post cementation.

Partial Restorations

In the case of limited to moderate coronal substance loss, the restorative strategy for endodontically treated front teeth varies, from direct composite restoration using the same layering or application techniques as for vital teeth (possibly with the contribution of intracoronal or extracoronal bleaching) to full veneers, when discoloration is not fully treatable or tends to relapse. From a restorative standpoint, it is only necessary to mention that a 1- to 2-week delay is mandatory prior to adhesively restoring a bleached tooth. For more extensive restoration, it is even advised to wait up to 6 weeks or more (depending on the extent and duration of bleaching treatment) to allow for tooth final color stabilization and to proceed with shade selection.[39] Figures 22-6 and 22-7 describe the clinical steps involved in the restoration of moderately decayed front teeth using direct composite and bleaching or veneers.

In posterior teeth exhibiting limited to moderate tissue loss, direct to indirect partial or full occlusal restorations are proposed. Their benefit is to take advantage of residual tooth structure to stabilize the restoration and minimize the potential impact of a prosthetic rehabilitation on periodontal tissues. For instance, second molars definitely profit from a conservative approach using adhesively luted intracoronal and full occlusal coverage restorations. An endocrown is a modification of the aforementioned concept, with extension of the restoration into the pulpal chamber as an additional retentive and stabilization structure. Figures 22-8, 22-9, and 22-10 describe the clinical steps involved in the restoration of moderately decayed posterior teeth using onlays, overlays, and endocrowns.

Foundation Restoration Underneath Full Crowns

If significant coronal substance loss justifies a full-tooth coverage, the strategy for foundation fabrication varies as follows:

- Amalgam core with/without metal post
- Composite core without post
- Composite post with fiber or ceramic post
- Composite with prefabricated metal post
- Cast gold post and core

Amalgam Core

A coronal-radicular amalgam foundation may be realized with or without a post (Figs. 22-11 through 22-14). The amalgam coronal-radicular core without a post is indicated for posterior teeth that have a large pulp chamber and lateral walls at least 4 mm high. This restoration extends slightly into the coronal portion of the canals (1 to 1.5 mm). The core is then retained by a combination of the divergence of the canals and natural undercuts in the pulp chamber. A single, homogeneous material is used for the entire restoration, rather than the dual phases of a conventional preformed post and core.

When shorter lateral walls are present, a post is mandatory to improve core stability and retention. The preferred roots for post placement for molars are the palatal root in the upper jaw and the distal root in the lower jaw. Other roots, thinner and presenting more pronounced curvatures, are normally not indicated for post placement. Coronal-radicular amalgam foundations are not indicated for premolars owing to their smaller dimensions; adhesive foundations or cast gold post and core are preferred.

Cast Gold Post and Core

Cast gold post and core restorations can be fabricated with either direct or indirect techniques.

Direct Technique

In the direct technique, a castable post and core pattern is fabricated in the mouth on the prepared tooth. A preformed plastic post pattern is seated in the post space. To gain a path of withdrawal, undercuts are blocked out with resin composite rather than by removing healthy dentin structure. Acrylic resin is added to create a core directly attached to the post pattern. The finished pattern is removed from the tooth and cast in the laboratory.

Indirect Technique

With the indirect technique, a final impression of the prepared tooth and post space is made (Fig. 22-15). As with the direct technique, the path of withdrawal is made by undercut blockout, not by dentin removal. The castable final post and core pattern are fabricated on a die from this impression. The crown margins need not be accurately reproduced at this stage. Proprietary systems provide matched drills, impression posts, and laboratory casting patterns of various diameters. An impression post (preferably a repositionable one) is fitted to the post space, and a final impression is made that captures the form of the remaining coronal tooth structure and picks up the impression post. In the laboratory, a die reproduces the post space and the residual coronal tooth structure for fabrication of the post and core pattern.

With both techniques, the fabrication of a temporary crown with intraradicular retention is needed. This provisional restoration must stay in the mouth only for a limited time to prevent decementation or canal reinfection. This is why this approach is less popular today and replaced by direct techniques in most cases.

The cast post and core is cemented at the second appointment. The cementation process must be passive in nature and an evacuation groove made on post side to facilitate cement expulsion and limit seating pressure. Rapid seating, excessive cement, and heavy seating pressure (e.g., occlusal force) can

FIG. 22-15 Nonvital teeth with limited coronal structure or numerous abutments. Indirect post and core fabrication helps to achieve proper parallelism and core anatomy. Core retention is assumed by a conventional or adhesive cement. Today, this solution is considered mostly for extended metal-ceramic restorations or sometimes for multiple full-ceramic restorations. **A,** Preoperative view. **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose the remaining tooth structure and a clean dentin substrate. Despite the indirect approach, all healthy coronal structures must be maintained to improve foundation stability. Access cavity and coronal walls are prepared with minimal convergence (6 to 10 degrees) to allow for easy insertion. **C, D,** Cast gold post and core fabrication and luting. Post space preparation: The post space is prepared with ad hoc instruments until reaching a length equivalent to about core height or more. More length is needed here, as retention cannot always be achieved through adhesion (endodontic retreatment or highly sclerotic contaminated dentin). Then a conventional glass ionomer or zinc phosphate cement is used. Post and core preparation: The gold post is sandblasted (or eventually sandblasted and coated if adhesive procedures are possible) using Rocatec or CoJet systems, 3M + silane. Post cementation: The post and core is luted with the aforementioned conventional cements or adhesive technique. Next, a self-curing adhesive system is applied on all cavity walls and into post preparation (before the insertion of the post and core) and covered with a dual-cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). **E, F,** Restoration fabrication and placement. A second impression is needed for the final prosthetic work to optimize restoration precision and quality. Lab-made PFM or full ceramic crowns are made with optimal foundation design and alignment.

produce high hydraulic pressures inside the root that may be great enough to crack the root.

Crown Preparation and Temporary Restoration

When a coronal restoration is indicated, the amount of tooth structure remaining after final preparation is the most important determinant of the design of the post and core. In addition, the underlying sound tooth structure provides greater resistance to fracture than any post and core type, design, or material. Natural tooth structure should always be carefully preserved during all phases of post space and crown preparation. Otherwise, preparation of endodontically treated teeth is not different from that for vital teeth, with the exception of severely discolored teeth, which require a slightly deeper chamfer to hide discoloration with the prosthetic structure and intrasulcular margins to reduce the visibility of a dark cervical tooth structure.

Provisional crowns for endodontically treated teeth must be used only for short periods of time because the loss of cement seal—which is, of course, symptom free—can lead to leakage, canal reinfection, and even serious carious invasion, thereby jeopardizing the success of endodontic treatment and even resulting in tooth loss.

SUMMARY

Endodontically treated teeth represent a singular situation because of the attendant qualitative and quantitative alterations of the dental substrate. Current literature indicates that treatment success relies on an effective coronal seal that will prevent canal reinfection and an adequate restoration that will resist functional stresses applied to the remaining tooth structure. The purpose of this chapter is to help the clinician make treatment planning decisions concerning the restorative options available for endodontically treated teeth.

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CHAPTER 23

Vital Pulp Therapy

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CHAPTER OUTLINE

The Living Pulp
Pulpal Response to Caries
Reparative Bridge Formation
Procedures for Generating Hard Tissue Barriers
Direct Pulp Capping
Pulpotomy
Partial Pulpotomy
Indirect Pulp Capping
Indications for Vital Pulp Therapy
Materials for Vital Pulp Therapy
Calcium Hydroxide
Resin-Modified Glass Ionomer Cements and Hydrophilic Resins
Mineral Trioxide Aggregate (MTA)
Calcium Silicate–Based Cements (CSCs)
MTA Applications in Vital Pulp Therapy
Direct Pulp Capping with MTA
Pulpotomy with MTA
Vital Pulp Therapy Techniques
Diagnosis
Caries Removal
Hemostatic Agents
Treatment Considerations
Treatment Recommendations
Two-Visit MTA Pulp Capping
One-Step Pulp Capping
The Permanent Restoration
Postoperative Follow-Up and Recall

"There is nothing permanent except change."

—Heraclitus

Vital pulp therapy is designed to preserve and maintain pulpal health in teeth that have been exposed to trauma, caries, restorative procedures, and anatomic anomalies. The treatment can be completed for permanent teeth that show reversible pulpal injuries, and the outcomes depend on a variety of factors.[63,162,215] The prime objective in vital pulp therapy is to initiate the formation of tertiary reparative dentin or calcific bridge formation. This procedure is essential for the preservation of involved immature permanent teeth where root development may be incomplete and preservation of arch integrity is critical during maxillofacial development.[288]

Recent advances in pulp biology and dental materials have provided alternative treatment strategies for healthy and partially inflamed pulps. Vital pulps can be successfully treated if the clinician has an improved understanding of diagnosis and case selection, hemostasis, caries removal, magnification systems, bioactive capping materials, bonded composites, and other restorative materials. The treatment is particularly valuable in young permanent teeth that have not attained their complete adult length and exhibit thin-walled roots and wide-open apices.[52,219,350]

Immature teeth may require up to 5 years or more to gain apical closure after emergence into the oral cavity. They are characterized by large dentinal tubules that allow increased permeability for microbial penetration.[53] The vulnerability and perceived poor prognosis of vital pulp treatment for immature permanent teeth have prompted aggressive treatment recommendations that include extraction.[350] Alternately, with an accurate diagnosis and early intervention, new strategies for pulp preservation promote a domain for continued hard tissue formation that encourages apexogenesis. Maintaining pulp vitality in these teeth reduces the probability of fracture, through continued growth and natural strengthening of the tooth structures.[78,94]

The introduction of mineral trioxide aggregate (MTA) and other bioceramic or calcium silicate-based cements (CSCs), along with advanced treatment strategies, has markedly changed the long-held concept that pulp capping after carious pulp exposure should be avoided. According to Seltzer and Bender,[289,290] "Pulp capping is a questionable procedure even under ideal circumstances." They further stated that "pulp capping should be discouraged for carious pulp exposures, since microorganisms and inflammation are invariably associated." The perception that outcomes for direct pulp capping in a carious field are inconsistent and problematic is based on traditional protocols and materials that did not generate a favorable milieu for hard tissue formation.[139,197,235,334] This perspective has encouraged clinicians to deliver alternative treatments, such as pulpotomy or pulpectomy, particularly in immature permanent teeth.[53,350] This rigid approach is further complicated by the difficulty in

FIG. 23-1 **Radiographs of carious molars in patients aged 12 to 38 years. A,** Mandibular left first molar in a 23-year-old with minor symptoms. **B,** Asymptomatic maxillary right molar in a 16-year-old. **C,** Asymptomatic maxillary left first molar in a 38-year-old. **D,** Deep caries in the mandibular right first molar of a 12-year-old. All patients were referred to the endodontist for root canal treatment based on radiographic observation. They exhibited normal vitality with cold testing, and all were treated successfully with vital pulp therapy. (© Dr. George Bogen.)

establishing the appropriate diagnosis because clinical signs, symptoms, and radiographic evidence may not accurately reflect the histologic condition of the involved pulp tissue (Fig. 23-1).[165] However, based on a better understanding of pulp physiology, caries microbiology, and the inflammatory mechanisms responsible for irreversible changes in pulp tissue, teeth with the potential for repair and continued vitality can now be more readily identified and predictably treated.[335]

Advances in our knowledge of pulpal physiology and immunology, together with recently introduced dental materials, have markedly changed the treatment approaches for teeth with involved pulps. Bioactive CSCs, such as MTA, have changed the perception that treating direct carious pulp exposures is unpredictable and therefore contraindicated. From the introduction of indirect pulp capping by Pierre Fauchard in the eighteenth century, dentistry has recognized the innate reparability of the dental pulp when exposed to injury.[99] Subsequent advances have been complemented by newly developed dental materials that provide superior sealing properties and protect the pulp from microorganisms and their toxic by-products.

A goal in vital pulp therapy has been to identify bioactive pulp capping and pulpotomy agents and implement a consistent treatment regimen that favors pulp preservation. It is recognized that outcomes for vital pulp therapy can vary, depending on the age of the patient, extent of bacterial contamination, and degree of pulp inflammation. Perhaps of greater importance may be the choice of pulp capping material and the quality of the permanent restoration.[63] Appropriate case selection, through a detailed differential diagnosis using multiple tests paired with a careful radiographic interpretation, is paramount for establishing the best treatment for the problem tooth. In its guidelines, the American Academy of Pediatric Dentistry (AAPD) states, "Teeth exhibiting provoked pain of short duration relieved with over-the-counter analgesics, by brushing, or upon the removal of the stimulus without signs and symptoms of irreversible pulpitis, have a clinical diagnosis of reversible pulpitis and are candidates for vital pulp therapy."[13]

In young patients, assessment of pulpal status before treatment is often difficult, but the probability of favorable outcomes increases with a diagnosis of reversible pulpitis or normal pulp (AAPD). Furthermore, subjective and sometimes negative patient reports or pain associated with cold testing does not absolutely signify that the pulp capping or pulpotomy procedure will be unsuccessful. Because pulpal disease is microbial in nature,[179] this chapter describes the microbiology of caries and the associated physiologic reactivity of pulp tissue. Based on the understanding that pulp tissue has an innate potential for repair in the absence of bacterial contamination, the chapter reviews new treatment concepts in vital pulp therapy intended for the ultimate preservation of the pulpally involved permanent tooth.[72,172]

THE LIVING PULP

As reviewed in Chapter 12, the dental pulp is a highly vascular and innervated loose connective tissue that has the unusual distinction of being enclosed within a rigid envelope composed of enamel, dentin, and cementum.[321,362] These hard tissues impart mechanical support and offer protection from the oral microbiota.[200] When these tissues are examined together embryologically and histologically, they may be referred to as

the *dentin-pulp complex*.[68,339] The pulp tissue accomplishes several important functions, including immune cell defense and surveillance, nutrition, dentinogenesis, and proprioreceptor recognition.[309] Healthy pulp tissue can generate reparative hard tissue, secondary, and peritubular dentin in response to assorted biologic and pathologic stimuli.[309] The maintenance of dental pulp vitality, therefore, is essential to the long-term retention and normal functioning of the tooth.

The dental pulp encompasses four distinct structural zones: a cell-rich zone, and core, composed of major vessels and nerves; a cell-free zone; and the odontoblastic layer that lines the entire pulp periphery.[321] The cell-rich zone demonstrates a greater density of undifferentiated mesenchymal cells and fibroblasts than is present in the pulp proper. The central pulp core consists of nerve fibers, blood vessels, fibroblasts, undifferentiated mesenchymal stem cells, immunocompetent cells, ground substance, and collagen fibers. The cell-free zone of Weil is subjacent to the odontoblast layer and is linked by capillaries, fibroblastic processes, and an extensive network of unmyelinated nerve fibers. The odontoblastic zone lines the pulp circumference as an epithelioid layer and includes the large, columnar-shaped odontoblasts, nerve fibers, capillaries, and dendritic cells.[186]

Vital pulp tissue comprises various cell populations that include fibroblasts or pulpoblasts, undifferentiated mesenchymal cells, odontoblasts, macrophages, dendritic cells, and other immunocompetent cells. The cells of the subodontoblastic layer and odontoblasts form a thin border between the inside margin of the dentin and the periphery of the pulp; this border is known as the *Höehl cell layer*.[130] The odontoblasts are tall, columnar-shaped cells separated from the mineralized dentin by predentin and characterized by processes that extend into the dentin and possibly to the dentinoenamel junction.[137,298,321,354] Odontoblasts are credited with formation of the mineralized predentin-dentin matrix, which is composed of an assortment of molecules, including phosphoproteins, glycoproteins, proteoglycans, and sialoproteins.[186] Repair mechanisms in the dental pulp are similar to those observed in normal connective tissue injured by trauma. When the enamel and dentin are challenged and the pulp exposed to advancing microorganisms, inflammatory changes can induce pulp necrosis, which precedes progressive pathologic changes that can include infection and its complications.[35,44]

Pressoreceptors and proprioreceptors protect the dentin-pulp complex against excessive occlusal loading while circulating immune competent cells confront bacterial challenges. Pulpless teeth with minimal remaining tooth structure that undergo root-filling procedures and are restored with post and core systems combined with cuspal coverage restorations are more vulnerable to irreparable fracture because of the loss of any protective proprioceptive mechanisms.[225,273,307] Investigations have shown that moisture depletion from dentin and the relative reduction of tooth stiffness are minimal after root canal treatment.[154,175,206] Although root canal treatment can prolong tooth survival, the cumulative loss of tooth structure from it and restorative care may precipitate tooth loss.[54,89,346] Root-filled teeth also demonstrate an increased susceptibility to recurrent caries, either because of poor marginal integrity of the permanent restoration or as a result of the modification of the biologic environment in these teeth.[226]

The pulp undergoes physiologic, pathologic, and defensive changes during its life.[240,333] These age-related transitions include continued dentin apposition, causing gradual narrowing of pulp volume and circumference.[96,241] The atrophy results in fibrosis, dystrophic calcification, degeneration of odontoblasts, and increased cellular apoptosis.[33,96] The aging of human dental pulp cells is primarily characterized by the formation of reactive oxygen species and senescence-related beta-galactosidase activity.[199] In addition, sensitivity to dental pain is reduced due to a decrease in fast-conducting A-delta fibers and diminished pulp repair, partly attributed to decreases in the levels of substances such as alkaline phosphatase.[217,295] An array of extracellular matrix macromolecules regulated by pulp cell activity contributes to tissue differentiation and growth, defense mechanisms, reactions to inflammatory stimuli, and the formation of calcified tissues.[333]

A comparison analysis of gene expression levels that reflect the activity of biologic cell function, proliferation, differentiation, and development found them markedly higher in young pulps compared to older dental pulps.[333] Analyses of young dental pulps indicated a greater expression level in cell and tissue differentiation, proliferation, and development of the lymphatic, hematologic, and immune systems compared to older dental pulps where the apoptosis pathway is highly expressed.[333] Although A-beta fiber function remains constant with aging, a decrease in A-delta fibers with age may reduce the perception of dental pain transmitted by these faster-conducting fibers.[217] Pulp volume and root canal lumen dimensions also diminish with advancing age as a result of continual deposition of dentin.[96,241] These age-related changes in tissue differentiation and organization, growth regulation, defense mechanisms, responses to inflammatory stimuli, and the deposition of calcified tissue are regulated by pulp cell activity and an assortment of extracellular matrix molecules.[333]

PULPAL RESPONSE TO CARIES*

The advance of invading microorganisms in carious lesions is the essential cause of pulpal inflammation and potential tissue necrosis. Acidogenic gram-positive bacteria, predominantly oral streptococci and lactobacilli, produce metabolic by-products during active caries that demineralize enamel and dentin.[147] Immune responses and pulpal inflammation occur when the caries front advances to within 1.5 mm of the pulp and bacterial antigens and metabolites diffuse through the dentinal tubules.[32,174,255] The main by-product in active carious lesions is lactic acid, which contributes to the demineralization of tooth structure. If the bacterial challenge continues, immune cell responses lead to increased inflammation and edema, initially characterized clinically by pulpal pain. Inflammation is a complex protective biologic response designed to remove injurious stimuli produced by pathogens and reestablish pulpal equilibrium. Prolonged inflammation in the low-compliance environment of the pulp space eventually leads to pulp disintegration and apical pathosis.[147,328]

The caries invasion is initially blocked by protective innate immune responses; it progresses to an adaptive immune response when bacteria directly approach the pulp.[146] The pioneer microbes during caries progression first encounter a positive outward flow of dentinal fluid, characterized by the deposition of immunoglobulins and serum proteins that slow

*Adapted from Hahn and Liewehr.[146-148]

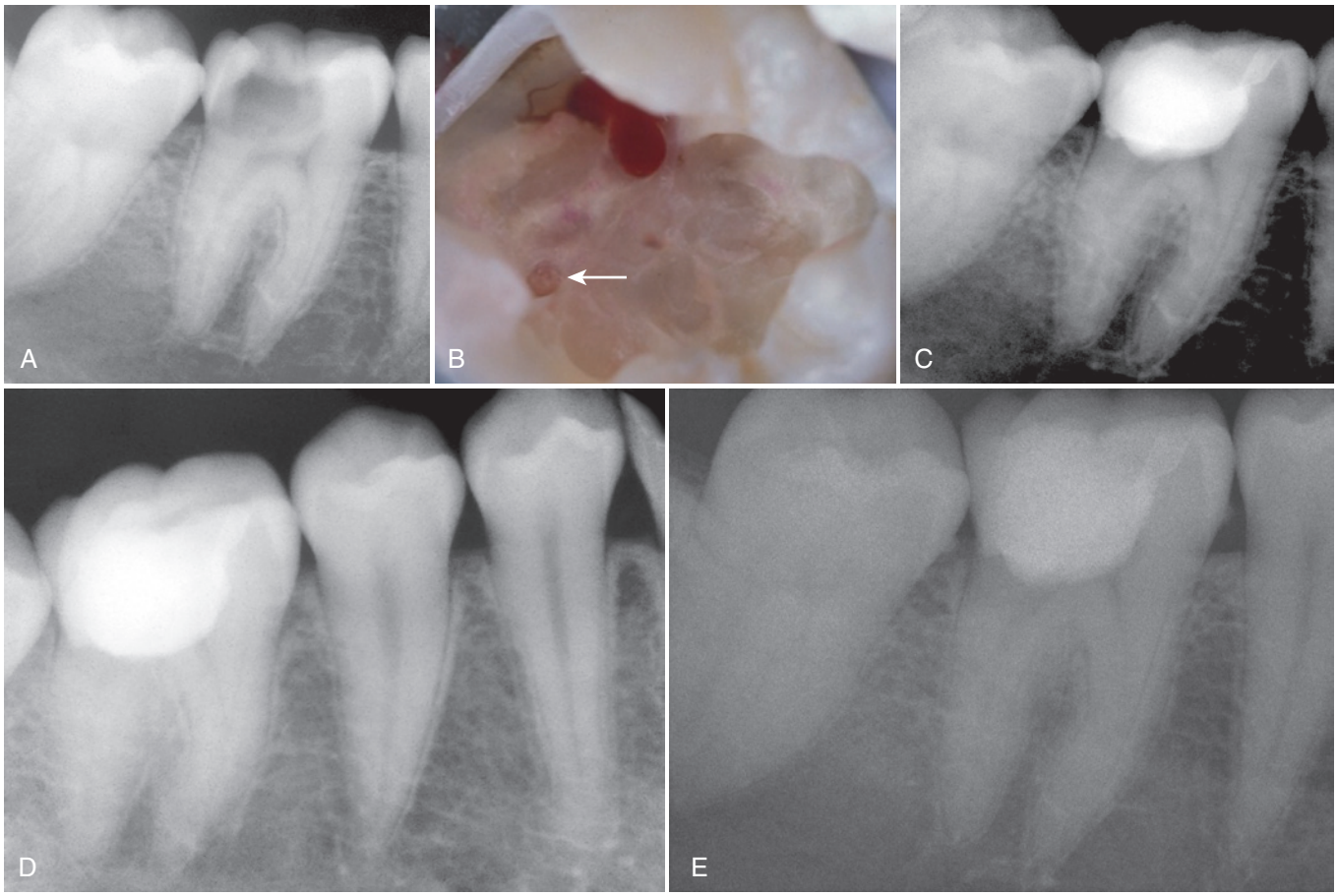

FIG. 23-2 Eighteen-year-old patient who presented with a carious mandibular right first molar that was sensitive to chewing. Cold testing elicited a short, nonlingering but painful response. **A,** Preoperative radiograph showing deep caries near the pulpal roof. **B,** Photograph after caries excavation using caries detector dye and 5-minute sodium hypochlorite hemostasis. Note the distobuccal pulp horn exposure and reactionary (reparative) dentin over the distolingual horn *(arrow).* **C,** Radiograph after bonded composite placement at the second visit, after confirmation of mineral trioxide aggregate (MTA) curing and continued pulp vitality. **D,** Three-year radiographic recall. Patient was asymptomatic and had normal responses on cold testing. **E,** Thirteen-year, 6 month radiographic review. Patient was asymptomatic and responded to vitality testing. The bonded composite restoration was intact and had no marginal degradation. (© Dr. George Bogen.)

the diffusion of bacterial antigens.[147] Potent microbial metabolites, such as lipoteichoic acid and lipopolysaccharide, also activate the innate immune system. The bacterial by-products stimulate signaling by Toll-like receptors in odontoblasts when the odontoblasts first encounter the carious front. They also stimulate proinflammatory cytokines, including interleukin-1, interleukin-8, interleukin-12, tumor necrosis factor alpha, vascular endothelial growth factor, and transforming growth factor beta (TGF-β).[108,156,361] Vascular endothelial growth factor promotes vascular permeability and angiogenesis. Dentin mineralization and matrix metalloproteinase secretion are also induced by the increased expression of TGF-β. Caries bacteria also activate complement pathways and induce the proinflammatory cytokine interferon gamma, which is responsible for killing phagocytosed bacteria by activated macrophages.[160] Odontoblasts also participate in the adaptive response to microbial invasion of dentin by the synthesis of a modified mineralized matrix known as *reactionary dentin,* a structurally altered hard tissue characterized by diminished tubularity.[62] As microorganisms advance toward the pulp, the modified helical structure in reparative dentin effectively constricts tubular lumen diameter and forms an active barrier against advancing pathogens (Fig. 23-2).[61]

As pulpitis progresses, vasoactive neuropeptides contribute to increased vascular permeability and intrapulpal blood flow. Increases in neuropeptide concentration and nerve sprouting characterize neurogenic inflammation, which can cause a transient increase in interstitial tissue pressure and contribute to painful pulpitis.[47,277] Immune cells attempt to control neurogenic inflammation with the secretion of peptides such as somatostatin and β-endorphin.[243] The primary effector cells in innate responses include natural killer cells, neutrophils, monocytes, and macrophages. Immature dendritic cells and T cells contribute to immunosurveillance during the progression of caries. Macrophages participate in the innate and adaptive immune responses by eliminating both pathogens and senescent cells while contributing to tissue homeostasis by repairing and remodeling tissue after inflammation.[146]

Cytokines are small, cell-signaling proteins secreted by innate immune cells that induce phagocyte extravasation during inflammation. Chemokines secreted by odontoblasts, fibroblasts, immature dendritic cells, and macrophages stimulate leukocyte recruitment by directing monocyte and neutrophil migration extravascularly to sites of infection.[146] Persistent infection engages the adaptive immune system, which can lead to edema and increased intrapulpal pressure, causing tissue destruction, acute phase protein production, and cell death, leading to tissue necrosis.[148] As the caries front progresses to the pulp, prompt clinical intervention, through removal of decay and bacterial antigens before irreversible pulpitis commences, can resolve pulpal inflammation and promote recovery.

REPARATIVE BRIDGE FORMATION

The foremost objective in vital pulp therapy is to encourage protective hard tissue barrier formation after injury. The process is initiated when regenerated odontoblast-like cells recruited from the cell-rich zone and subodontoblastic layer advance the repair of pulpodental defects after migration of highly vascularized tissue to the site. The repair process after pulp capping is characterized by four steps: (1) moderate inflammation, (2) recruitment and advance of dedicated adult reserve stem (progenitor) cells, (3) proliferation of the progenitor cells, and (4) terminal differentiation.[132] Strong evidence supports the role of inflammation as a prerequisite for tissue repair to proceed.[104]

The osteoblast/odontoblast-like progenitors responsible for reparative calcific bridge formation are either fibroblasts, inflammatory cells that undergo phenotypic conversion, or potentially resident stem cells activated by cytokines released during the inflammatory process.[130] Progenitor cell differentiation may also be modulated during inflammation by activation of antigen-presenting dendritic cells or triggered by specific odontoblast and fibroblast membrane receptors.[130] Currently, the origin of the differentiated odontoblast-like cells remains controversial. Fibroblasts, perivascular cells, bone marrow stem cells, and undifferentiated mesenchymal stem cells have all been proposed as potential progenitors.[338] A recent histologic study, however, suggests that the amorphous, atubular calcified repair tissue formed subjacent to the calcium hydroxide (CH) placed on the pulp wound in the absence of odontoblasts is produced by pulpal fibroblasts.[276a] Therefore, such mineralized hard tissue is not genuine dentin, but repair tissue that has been called "reparative dentin," for lack of a better term.

Odontoblast replacement in nonhuman primates directly after pulp exposures capped with CH has been examined during the cell migration and replication stages.[111] Newly differentiating odontoblast-type cells showing initial matrix formation were demonstrated as early as day 8 at the CH-pulp interface. The continual influx of labeled differentiating cells indicated that the original derivation was from the deeper, central pulp tissue that required two DNA replications before terminal differentiation.[111] Investigations have also indicated that reparative bridge mineralization may be more dependent on the extracellular matrix than on the capping material selected.[171,189,259]

During healing, initial calcification immediately after pulp amputation is characterized by a proliferation of extracellular matrix vesicles situated between the forming cells and the injured pulp surface.[152,264] The formation of needle-like crystals and osmophilic material within vesicles proceeds with an accumulation of crystals and aggregate at the calcified fronts, along with the disappearance of the vesicular membrane. The crystals produced during the calcification process are associated with phosphate and calcium ions, similar to fundamental calcification processes shown by other normal and pathologic calcified tissues.[152] Defects in pulpal healing and dentinal bridge formation are associated with different pulp capping materials and include pulpal inflammation, bacterial microleakage, operative debris, and tunnel defects.[188]

PROCEDURES FOR GENERATING HARD TISSUE BARRIERS

Direct Pulp Capping

The treatment options for permanent teeth that encourage pulp preservation include direct and indirect pulp capping and partial or complete pulpotomy. *Direct pulp capping* is defined as "placing a dental material directly on a mechanical or traumatic vital pulp exposure" and "sealing the pulpal wound to facilitate the formation of reparative dentin and maintenance of the vital pulp."[12] The procedure is indicated for pulp exposures incurred as a result of caries removal, trauma, or tooth preparation. When mechanical exposures occur during tooth preparation, the exposed tissue is generally not inflamed. However, in cases of trauma or carious exposure, the degree of inflammation is the key predetermining prognostic factor. According to the American Association of Endodontists, "In a carious pulp exposure, underlying pulp is inflamed to a varying or unknown extent."[12] The major challenge in direct pulp capping is the proper identification and removal of the acutely inflamed or necrotic tissue compromised by longstanding exposure to oral microorganisms.[210]

Pulpotomy

Pulpotomy, or *pulp amputation,* is a more intrusive procedure defined as "the removal of the coronal portion of the vital pulp as a means of preserving the vitality of the remaining radicular portion: may be performed as emergency procedure for temporary relief of symptoms or therapeutic measure, as in the instance of a Cvek pulpotomy."[12] After complete amputation of the coronal pulp, a capping material is placed over the pulp floor and the remaining exposed tissue in the canal orifices. Dressing materials of varying toxicity have been used for this purpose, including ferric sulfate, creosote, phenol, zinc oxide eugenol, polycarboxylate cement, glutaraldehyde, CH, and formaldehyde; some of these dressing materials embalm any remaining tissue.[265]

The procedure is recommended for primary teeth for which short-term outcomes are generally favorable. Formocresol has been the accepted "standard" universal pulpotomy agent in primary teeth and is recommended for young adult teeth; however, it has considerable drawbacks that put into question its continued use in humans.[195,196,281,318] It has been identified as carcinogenic and genotoxic, and experiments have shown a high incidence of internal resorption in nonhuman primate models.[120,203,207] Changes in the root canal system apical to formocresol placement can also create challenges when orthograde root canal treatment is attempted.[278] Comparative studies have demonstrated MTA to be an appropriate replacement for formocresol for primary molar pulpotomy.[48,164,306] Recent

investigations also support the use of MTA and other CSCs for application in pulpotomy procedures in permanent teeth.[17,300]

Partial Pulpotomy

Partial pulpotomy (shallow pulpotomy, or Cvek pulpotomy) is defined as the removal of a small portion of the vital coronal pulp as a means of preserving the remaining coronal and radicular pulp tissues.[12] After the pulp has been exposed and is visualized after hemostasis, inflamed or necrotic tissue is removed to uncover deeper, healthy pulp tissue in the pulp chamber.[77,257] Partial pulpotomy and direct pulp capping can be viewed as similar procedures, but they differ in the amount of vital pulp tissue remaining after treatment. Partial pulpotomy is the preferred option in elective treatment procedures for teeth diagnosed with anatomic anomalies, such as dens invaginatus.

Indirect Pulp Capping

Indirect pulp capping is defined by the AAPD as "a procedure performed in a tooth with a deep carious lesion approximating the pulp but without signs or symptoms of pulp degeneration. Indirect pulp treatment is indicated in a permanent tooth diagnosed with a normal pulp with no signs or symptoms of pulpitis or with a diagnosis of reversible pulpitis."[13] The treatment can be completed as a one-step or two-step procedure (stepwise technique) with the objective of arresting the active carious lesion.[37] Indirect pulp capping has been shown to be an effective technique for caries and patient management in the primary dentition, but it remains controversial in permanent teeth.[46,107,116,210,212] The technique is similar for both primary and permanent teeth, except that permanent teeth require reentry for the removal of residual carious tissue; reentry also is considered necessary to confirm reactionary dentin formation.[53,178] However, data from a recent investigation have questioned the need to reopen cavities to remove residual infected dentin in a subsequent visit.[209]

The treatment requires prudent case selection, including identification of asymptomatic patients with no suspicion of irreversible pulpitis. Pulp exposures are avoided during caries excavation by removal of the superficial demineralized necrotic dentin and then the removal of the peripheral dentin.[119] After excavation, the remaining carious dentin is lined with CH and sealed with a provisional material, such as intermediate restorative material (IRM) or a resin-modified glass ionomer (RMGI). The patient returns in 8 to 12 weeks for placement of a permanent coronal restoration. Advocates of indirect pulp capping argue that pulp healing can be compromised if the carious dentin barrier is removed during excavation and that the prognosis of vital pulp therapy (direct exposure) is unfavorable.[36] A challenging aspect of indirect pulp capping is determining the exact boundary point where caries excavation is terminated. Therefore, the technique is based primarily on subjective criteria and the operator's skill.[201] Further complicating the process is the presence of potential voids under the provisional restoration; during the mineralization process, these can permit dentin to lose volume during desiccation. Another drawback is the rapid reactivation of dormant lesions after restoration failure.[36] However, in younger patients with management issues, indirect pulp capping has shown promising results for immature permanent teeth in which the apical foramina are large, the canal walls are thin, and pulp vascularization is pronounced.[88,138,201,323]

INDICATIONS FOR VITAL PULP THERAPY

Vital pulp therapy is recommended for all teeth diagnosed with reversible pulpitis or partially inflamed pulps in which the remaining healthy tissue can be conserved to generate a hard tissue barrier that seals and protects the pulp from future microbial insult. The introduction of new bioactive materials, along with modified protocols, make more teeth with deep caries, traumatic injuries, and mechanical exposures viable candidates for innovative pulp therapies designed to potentiate and maintain pulpal survival. Treatment outcomes for direct pulp capping and pulpotomy procedures depend on multiple factors, beginning with a differential diagnosis that takes into account pulp testing, radiographic evaluation, clinical evaluation, and the patient history to determine a rational prognosis. Outcomes also depend on case selection, hemostatic agents, choice of pulp capping material, and the integrity of the sealed permanent restoration. The underlying purpose in vital pulp therapy is to avoid or delay root canal therapy and advanced restorative care because these, together, may reduce long-term tooth survival compared to teeth with vital pulps.*

MATERIALS FOR VITAL PULP THERAPY

A variety of pulp dressing materials have been investigated and used over the past century to encourage bridge formation and pulp preservation. A short list of compounds includes CH products, calcium phosphate, zinc oxide, calcium-tetracycline chelate, zinc phosphate and polycarboxylate cements, Bioglass, Emdogain, antibiotic and growth factor combinations, Ledermix, calcium phosphate ceramics, cyanoacrylate, hydrophilic resins, RMGI cements, hydroxyapatite compounds and, recently, MTA and other CSCs.† Other strategies designed to arrest invasive caries and promote repair of underlying tissues include the use of lasers, ozone technology, silver diamine fluoride, and bioactive agents that stimulate pulpal defense mechanisms.‡ Retrospective investigations have shown varying success rates of 30% to 85% for direct pulp capping in humans, depending on the method, hemostatic agent, and dressing material used.§ The search to identify and produce the ideal pulp capping material continues, and remarkable progress has been made in pulp preservation research in the past decade.

Calcium Hydroxide

Calcium hydroxide has long been considered the universal standard for vital pulp therapy materials. The introduction of CH into dentistry is credited to Hermann in the 1920s.[158] Although the material demonstrates many advantageous properties, long-term study outcomes in vital pulp therapy have been inconsistent.[21,28,30,165] Desirable characteristics of CH include an initial high alkaline pH, which is responsible for stimulating fibroblasts and enzyme systems. It neutralizes the low pH of acids, shows antibacterial properties, and promotes pulp tissue defense mechanisms and repair. The drawbacks of CH include weak marginal adaptation to dentin, degradation and dissolution over time, and primary tooth resorption.

*References 54, 55, 89, 234, 251, 282, 314, 331, and 346.
†References 31, 34, 113, 155, 161, 182, 262, 265, 308, 311, 360, and 368.
‡References 45, 67, 79, 132, 239, 261, 279, 367, and 369.
§References 21, 26, 30, 81, 151, 222, and 349.

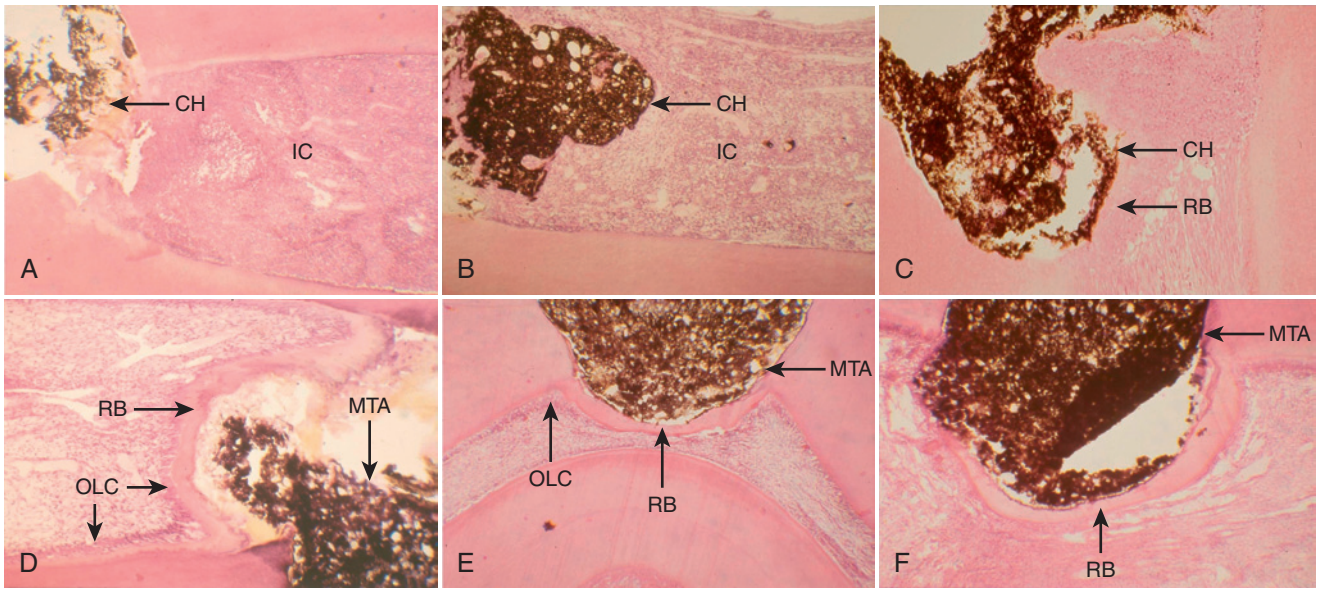

FIG. 23-3 Reparative bridge formation compared in dog pulps using mineral trioxide aggregate *(MTA)* and calcium hydroxide *(CH)*. **A,** Two-week pulp response to CH showing inflammatory cells *(IC)*. **B,** Tissue specimen showing lack of bridge formation and disorganized tissue proximal to CH. **C,** An 8-week specimen with partial reparative bridge (RB) formation subjacent to CH. **D,** Two-week pulpal response to MTA showing notable barrier formation and layer of organized odontoblast-like cells *(OLC)*. **E,** Pulp tissue section demonstrating complete calcificbridge formation proximal to MTA at 4 weeks. **F,** Sample section pulp capped with MTA at the 8-week period showing organized hard tissue formation with no inflammatory cell infiltrate. (Loma Linda University, Loma Linda, California.)

Reparative bridge formation subjacent to CH can also be characterized by tunnel defects.* Histologically, CH demonstrates cytotoxicity in cell cultures and has been shown to induce pulp cell apoptosis.[14,131,287]

Tunnel defects have been demonstrated in reparative hard tissue bridges associated with both CH and CSCs.[10] However, the primary difference between the two pulp therapy agents is that CH products are absorbable over time and dimensionally unstable. The slow disintegration of the CH after hard tissue barrier formation can allow microleakage, thus permitting a slow ingress of microorganisms through calcific bridge defects. This can induce subsequent pulpal degeneration, further leading to potential dystrophic calcification and pulpal necrosis. Over extended periods, this problematic outcome of CH pulp capping can complicate nonsurgical root canal treatment if required.[237]

Clinical retrospective investigations have shown variable success rates over 2- to 10-year recall periods for direct CH pulp capping in humans.[21,28,30,165] Two current studies have examined the efficacy of CH as a direct pulp capping agent. One study examined the survival rate of 248 pulp-capped teeth that were diagnosed either as having normal pulps or as exhibiting spontaneous pain; the researchers found an overall survival rate of 76.3% with an average recall period of 6.1 years.[81] Treatment outcomes were less favorable for teeth showing spontaneous pain, in older compared to younger patients, and in teeth restored with glass ionomer cements. The probability of pulps becoming nonvital after CH pulp capping was greater within the first 5 years of treatment.

*References 14, 25, 73, 74, 131, 134, 238, and 286.

The second study observed 1,075 teeth directly pulp capped with a CH-based agent; these teeth had either healthy pulps or showed signs of reversible pulpitis.[349] Inclusion criteria limited pulp chamber roof exposures to no larger than 2 mm in diameter. Successful outcomes were 80.1% after 1 year and 68% after 5 years; this diminished to 58.7% after a 9-year observation period.

The results of the two studies indicate increasing failure rates over time, attributable to absorption of the material under permanent restorations proximal to mineralized bridges with tunnel defects. Another investigation has confirmed decreasing success rates with CH pulp capping with extended recall periods.[222] Calcium hydroxide clearly has many favorable characteristics as a vital pulp therapy agent. However, the material also demonstrates inherent physical weaknesses and can no longer be considered the preferred universal agent in vital pulp therapy (Fig. 23-3).[105,162a,222,224a,247]

Resin-Modified Glass Ionomer Cements and Hydrophilic Resins

Adhesive systems were introduced in the early 1980s as potential agents for direct pulp capping of cariously and mechanically exposed pulps.[170,216,355] These materials include RMGI cements, composite resins, and hydrophilic resins. Hydrophilic resins and RMGI cements initially showed favorable outcomes in preliminary pulp capping investigations with nonhuman primates based on standards set by the International Organization for Standardization (ISO).[71,315-317] However, the transitional use of these materials in human subjects did not demonstrate the corresponding biocompatibility or consistent reparative bridge formation.[2,90,131,153,166,235] Investigations that

have examined responses of resin-based materials in human teeth have demonstrated unfavorable histologic reactions when the material is placed directly or in close proximity to pulp tissue.[2,95,143,236,274] Histologic sections from these studies typically demonstrate the presence of inflammatory cell infiltrates consistent with pulp cell cytotoxicity, subclinical adhesive failures at the pulp interface, and a profound absence of biocompatibility.*

Research data have shown that increasing concentrations of triethylene glycol dimethacrylate (TEGDMA), a common dentin bonding compound, differentially increase the levels of apoptotic and necrotic cell populations after direct exposure.[305] Moreover, even low levels of TEGDMA diminish alkaline phosphatase activity and calcium deposition, thus inhibiting pulp cell mineralization and potential reparative bridge formation.[127] Alternatively, adhesive resins have shown some promise when combined with additives or growth factors, such as hydroxyapatite powder, dental matrix protein-derived synthetic peptides, calcium chloride ($CaCl_2$), calcium phosphate, and antibacterial agents, including 12-methacryloyloxydodecylpyridinium bromide (MDPB).[76,183,312] It is evident that bonding agents used for direct pulp capping do not predictably generate a favorable environment for pulp healing and hard tissue formation.[299] However, hydrophilic resins and RMGI cements provide excellent seals when they are combined with light-cured composites in permanent restorations and then placed directly over pulp capping materials such as MTA.[20,101,249]

Mineral Trioxide Aggregate (MTA)

Mineral trioxide aggregate was introduced as a pulp capping material by Torabinejad and associates in the mid-1990s.[113] Most preliminary experimental and current clinical data in vital pulp therapy are based on the proprietary material ProRoot MTA (Tulsa/Dentsply, Tulsa, Oklahoma). The cement consists of hydraulic calcium silicate powder containing various oxide compounds, including calcium oxide, ferric oxide, silicon oxide, sodium and potassium oxides, magnesium oxide, and aluminum oxide.[51] The material exhibits favorable physiochemical characteristics that stimulate reparative dentinogenesis by recruitment and activation of hard tissue–forming cells, contributing to matrix formation and mineralization.[260] Soluble cytokines and growth factors that mediate wound repair of the dentin-pulp complex are nested in the extracellular matrix, and MTA stimulates reparative hard tissue formation by sequestering these growth factors and cytokines embedded in the surrounding dentin matrix.[190,191,327,340] Calcium hydroxide and calcium silicate hydrate, the principal by-products formed during hydration of mixed MTA, contribute to a sustained alkaline pH.[50,117] The setting properties of the hydroscopic silicate cements are not affected by the presence of tissue fluids or blood.[329]

During the setting process, the gradual release of calcium ions encourages reparative barrier formation by promoting signaling molecules, such as vascular endothelial growth factor (VEGF), macrophage colony-stimulating factor (MCSF), TGF-β, and interleukins IL-1β and IL-1α.[214,267] MTA demonstrates superior marginal adaptation to dentin compared to CH-based agents; MTA forms an adherent interfacial layer during mineral nucleation at the dentin surface that appears similar in composition to hydroxyapatite when examined with X-ray diffraction, energy-dispersive X-ray analysis, and scanning electron microscopy (SEM).[260,283,330]

If the pulp is injured, wound healing and the repair process can advance only after the initiation of the inflammatory reaction.[130,205] Similar to CH, MTA induces an inflammatory cascade that results from calcium ion release and the creation of an alkaline environment, producing tissue necrosis. Both MTA and CH have been shown to stimulate and increase the Höehl cell mitosis index in rodent models.[82,83] MTA activates the migration of progenitor cells from the central pulp to the injury site and promotes their proliferation and differentiation into odontoblast-like cells without inducing pulp cell apoptosis.[260] MTA also stimulates in vitro the production of messenger ribonucleic acid (mRNA) and increases protein expression of the mineralized matrix genes and cellular markers crucial for mineralization after matrix formation.[260]

Gray MTA has been shown to enhance cell proliferation and survival of cultured human dental pulp stromal cells.[267] The biocompatibility of set MTA up-regulates the expression of transcription factors, angiogenic factors, and gene products, such as dentin sialoprotein, osteocalcin, and alkaline phosphatase.[266] Odontoblast signaling proteins are essential in the differentiation of progenitor cells into the odontoblast-like cells responsible for repair and hard tissue deposition.[266,267] After MTA pulp capping, both sialoprotein and osteopontin have been observed in the fibrodentin matrix at the exposure site during the process of reparative hard tissue formation.[194]

Dental pulp cells differentiate into the odontoblastic cell line in the presence of the signaling molecules, such as TGF-β, heme oxygenase-1 enzyme, and bone morphogenetic proteins BMP-2, BMP-4, and BMP-7.[141] MTA most likely up-regulates fibroblast secretion of BMP-2 and TGF-β1 and therefore stimulates and promotes mineralization and hard tissue regeneration.* MTA induces a time-dependent environment that is proinflammatory and promotes wound regeneration through up-regulation of cytokines.[275] Immunohistochemical analyses show that cytokines, including myeloperoxidase, inducible nitric oxide synthase, VEGF, nuclear factor-kappa B (NF-κB), activating protein-1, and cyclooxygenase-2, show increased expression in the presence of MTA. Cytokine up-regulation is responsible for inducing biomineralization by producing apatite-like clusters on collagen fibrils at the MTA-dentin interface. MTA does not affect the generation of reactive oxygen species, thereby positively influencing cell survival. MTA also has been shown to improve the secretion of IL-1β , Il-6, and IL-8.[49,57,66] However, an inhibitory effect on dental pulp cells has been demonstrated in the presence of MTA, which may be attributed to the release of aluminum ions.[231] The data indicate that MTA promotes a biocompatible, noncytotoxic, antibacterial environment and surface morphology that are favorable for reparative calcific bridge formation. MTA stimulates the release of the dentin matrix components necessary for hard tissue repair and regeneration in mechanically exposed healthy and partially inflamed pulps (Figs. 23-4 to 23-6).†

Calcium Silicate–Based Cements (CSCs)

A variety of new bioactive CSCs or bioceramic materials have been developed since the introduction of MTA.[85,128,198]

*References 2, 90, 236, 258, 274, and 299.

*References 84, 141, 142, 149, 291, 313, and 357.
†References 6, 113, 142, 230, 247, 276, and 326.

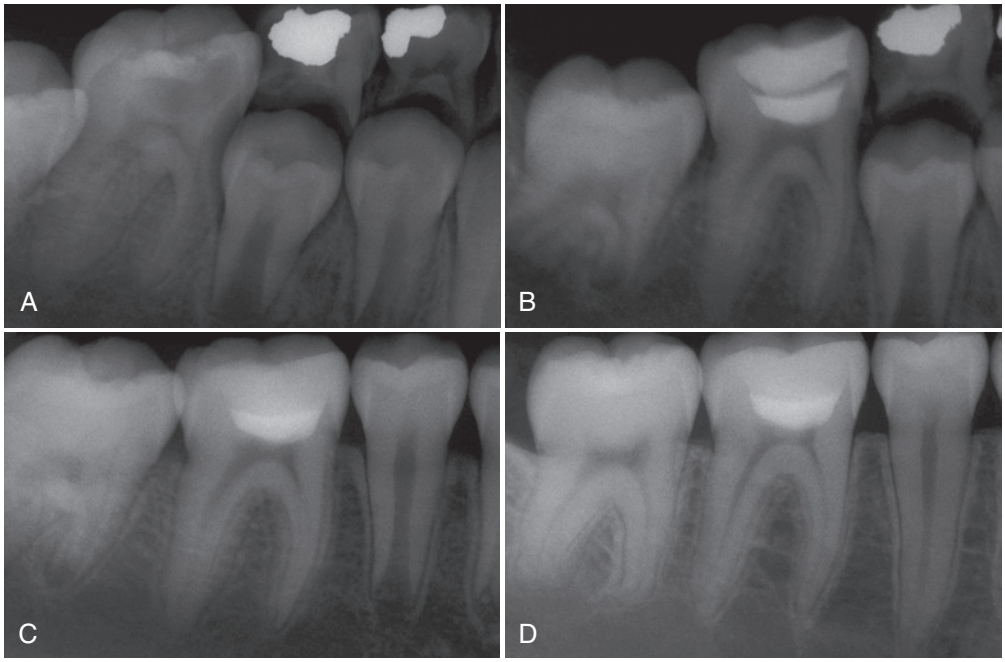

FIG. 23-4 Symptomatic mandibular right first molar in a 9-year-old patient. **A,** Preoperative film; cold testing evoked a short period of discomfort. **B,** Postoperative radiograph after sodium hypochlorite hemostasis, direct mineral trioxide aggregate (MTA) pulp caps on 0.5- and 1-mm exposures, and wet cotton pellet with Photocore® provisionalization. **C,** One-year radiographic review; tooth responds normally to cold vitality test. **D,** Control radiograph at 8 years. Patient was asymptomatic and exhibited normal pulp testing response. (© Dr. George Bogen.)

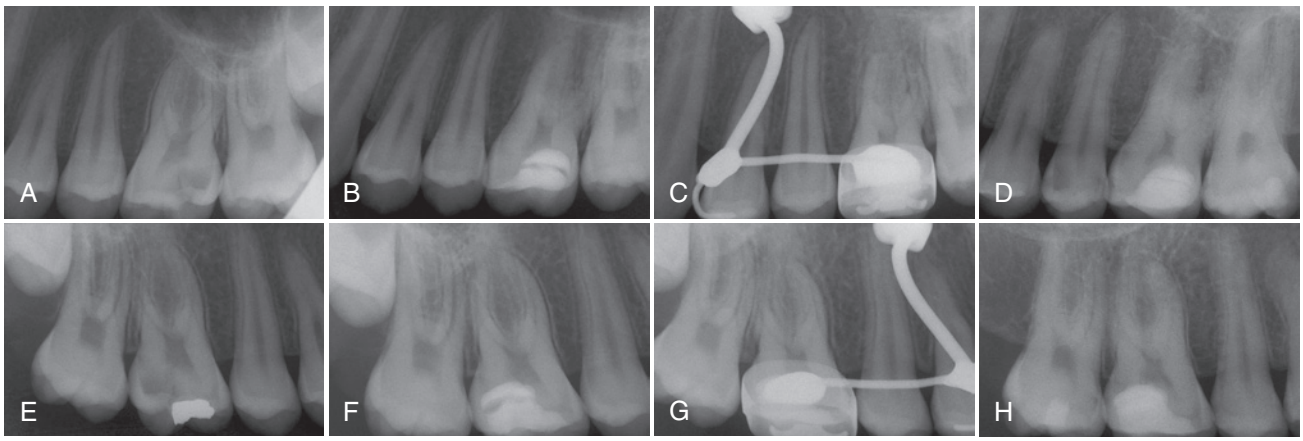

FIG. 23-5 **A** and **E,** Radiographs of maxillary right and left first molars with deep distal caries in a 12-year-old patient. **B** and **F,** Posttreatment radiographs after direct mineral trioxide aggregate (MTA) pulp caps, wet cotton pellets, and Photocore provisionalization. **C** and **G,** One-year radiographic recall; the patient was in active orthodontic treatment. **D** and **H,** Radiographic controls taken 7.5 years after MTA direct pulp caps. Note caries in maxillary left second premolar in **D** (patient was advised). The patient was asymptomatic and tested normal on cold tests at the 1- and 7.5-year recall periods for both molars. (© Dr. George Bogen.)

Preliminary investigations with CSCs have demonstrated physiochemical and bioinductive properties comparable to those of MTA, indicating the potential future application of these materials in vital pulp therapy.[42,98] Some of these tricalcium-based materials include BioAggregate (Innovative Bioceramix, Vancouver, British Columbia), Biodentine (Septodont, Cambridge, Ontario, Canada), MTA-Angelus, MTA Bio, and MTA Branco (MTA-Angelus, Londrina PR, Brazil).[133,202] Other formulations include EndocemMTA (Maruchi, Wonju-si, Gangwon-do, South Korea) and Endosequence root repair material (Brasseler USA, Savannah, Georgia). Additional compounds are currently undergoing clinical investigations to establish their safety and efficacy.[16,242]

The main components of MTA and the new CSCs are tricalcium silicate and dicalcium silicate, major components of Portland cement. Hydraulic tricalcium silicates promote reparative barrier formation by up-regulation of transcription factors after gaining immediate strength on hydration. However, research data are limited on direct pulp capping and pulpotomy treatments in humans using these new bioceramic products.

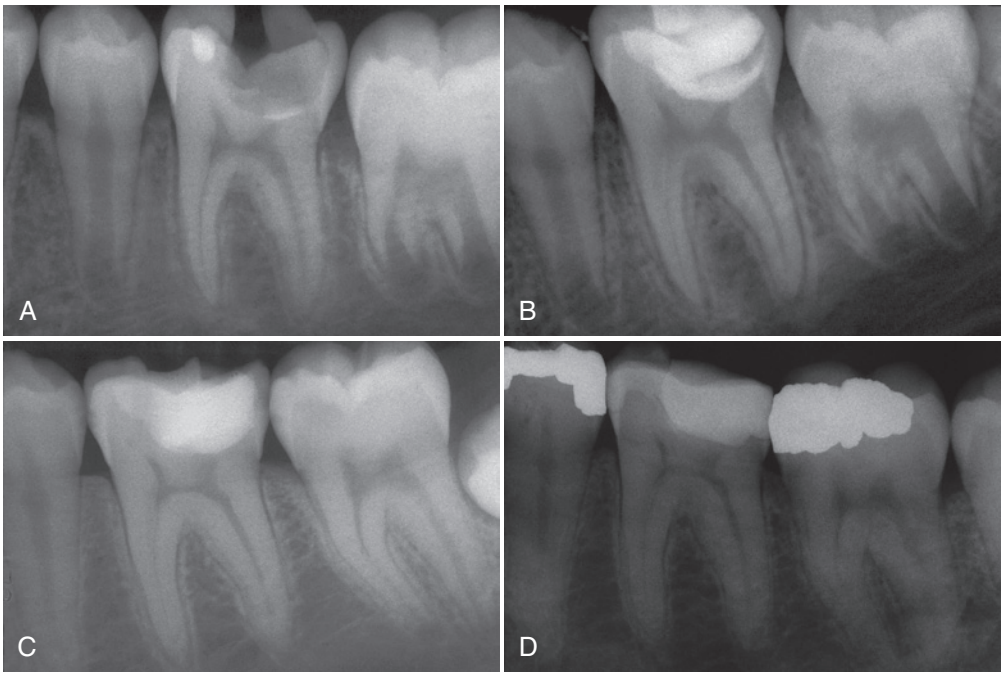

FIG. 23-6 Direct pulp capping of a mandibular left molar in an 11-year-old patient. **A,** Pretreatment radiograph showing large carious lesion after loss of a temporary restoration. **B,** Radiograph after mineral trioxide aggregate (MTA) placement with moist cotton pellet, and an unbonded Photocore® interim restoration was placed over a 2-mm pulp exposure. **C,** Four-year radiographic recall. The molar responded normally to carbon dioxide (CO_2) ice testing. **D,** Thirteen-year recall radiograph showing typical periapical structures and completed root formation. Cold testing responses were normal, and the patient was asymptomatic. (© Dr. George Bogen.)

BioAggregate is a bioinductive tricalcium cement that can induce mineralization in osteoblast cells by increasing levels of osteocalcin, collagen type 1, and osteopontin gene expression.[363] Hydration of the cement results in the formation of calcium silicate hydrate and CH, showing high concentrations of silica and calcium phosphate.[136] This characteristic is consistent with materials used in vital pulp therapy to promote hard tissue formation. Investigations using X-ray diffraction show the material to have a composition similar to that of MTA, but BioAggregate contains tantalum rather than bismuth oxide to provide radiopacity.[268] BioAggregate demonstrates remarkable biocompatibility compared to MTA, inducing cell differentiation in both human periodontal ligament and gingival fibroblasts.[356] Both fresh and set mixtures of MTA and BioAggregate have shown antimicrobial properties effective against *Enterococcus faecalis* in vitro when combined with equal amounts of human dentin powder.[366] The material also shows a greater resistance to dislodgement in an acidic environment compared to MTA, in addition to higher fracture resistance when used as a filling material.[150,336]

Biodentine is a tricalcium silicate–based cement that also demonstrates exceptional bioactive properties with potential for both direct and indirect pulp capping procedures. The cement has a short setting time of 10 minutes and does not induce genotoxic or cytotoxic effects when measured with the Ames mutagenicity test. It is considered a biocompatible dentin replacement material for use under various restorative materials as a base or liner, and it does not alter human pulp fibroblast cytodifferentiation.[198] SEM analysis demonstrates the sealing ability of Biodentine to be similar to that of MTA; Biodentine forms needle-like crystals resembling apatite at the dentin interface.[91] The material induces odontoblast-like cell differentiation, stimulates biomineralization, and promotes hard tissue formation when used as a pulp capping material.[293,365]

Another promising material for vital pulp therapy is MTA-Angelus, which has a basic formulation of 25% bismuth oxide and 75% Portland cement. The composition eliminates calcium sulfate, providing a short setting time of 10 minutes, which is preferable for one-visit pulp capping or pulpotomy procedures (Fig. 23-7).[42] Variations in bismuth oxide and the presence of iron characterize the chemical composition of MTA-Angelus, and the crystalline structures formed on hydration are similar to gray and white ProRoot MTA.[302] MTA-Angelus and ProRoot MTA have been compared experimentally as pulp capping agents in intact, caries-free human teeth.[5] Histomorphologic examination of extracted teeth revealed that the two materials produced similar responses with regard to inflammation and hard tissue formation.[5,193] MTA-Angelus also demonstrates antifungal properties and a lower compressive strength than ProRoot MTA.[29,180]

Endosequence root repair material shows low cytotoxicity, antibacterial activity against *E. faecalis,* and strong potential as a pulp capping material.[80,370] Another material, calcium-enriched mixture (CEM) cement, has also demonstrated excellent physical and biologic properties in vital pulp therapy investigations.[17-19,256]

This new generation of CSCs appears promising when used as vital pulp therapeutic agents and current investigations appear to support these materials' future potential.

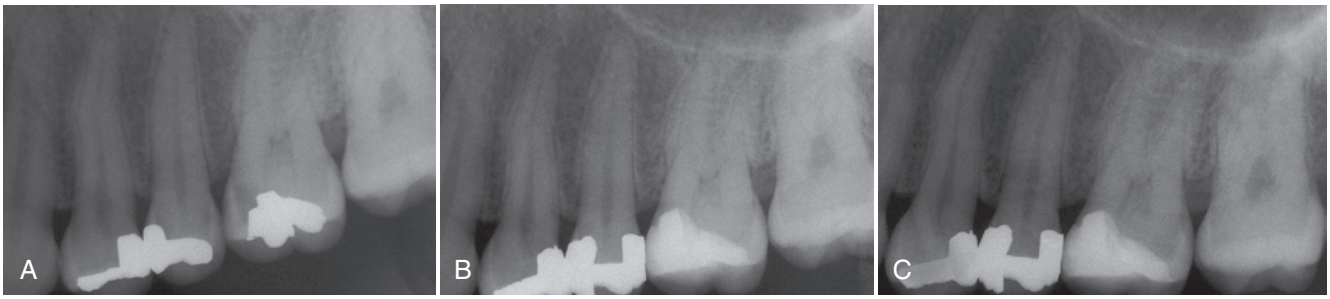

FIG. 23-7 Thirty-four-year old patient who presented with sensitivity to hot, cold, and sweet foods. **A,** Preoperative radiograph of maxillary left first molar showing mesial caries and an occlusal amalgam restoration. **B,** Posttreatment radiograph after 1.5-mm wide pulp exposure, direct "fast set" mineral trioxide aggregate (MTA-Angelus) pulp cap, and bonded composite restoration. The tooth was symptomatic for 1 hour after the local anesthetic had dissipated. **C,** Two-year radiographic review; the tooth has a normal response to cold testing. (© Dr. George Bogen.)

MTA APPLICATIONS IN VITAL PULP THERAPY

Direct Pulp Capping with MTA

Controlled prospective investigations on direct pulp capping in humans using MTA against cariously exposed pulps are limited. Collectively, most studies are inconsistent with regard to case selection, treatment strategies, and clinical protocols.[232] The subsequent spectrum of outcomes is reflected by the absence of standardized guidelines for caries removal, hemostatic agents, single- versus two-visit delivery sequences, and the choice and placement of a capping material.

Unsatisfactory outcomes for direct capping due to variations in treatment delivery and protocols were clearly demonstrated in a cohort investigation completed by predoctoral dental students.[229] Fifty-one direct MTA pulp caps completed in a carious field were radiographically and clinically evaluated for a 12- to 27-month period. The overall success rates using Kaplan-Meier analysis were 67.7% at 1 year and 56.2% at 2 years. The poor outcome can be attributed to an absence of strict control protocols with regard to caries removal, selection of hemostatic agents, and appropriate magnification, illumination, and thickness combined with area coverage of MTA. As a result, the investigation concluded that the amount of hemorrhaging after pulp exposure was not a determining factor in the clinical outcome, contrary to other findings.[63,215]

More impressive results for pulp maintenance and continued vitality after direct pulp capping procedures have been demonstrated in several other contemporary studies.[4,6,204,222] An investigation examined 30 immature permanent teeth exhibiting wide-open apices directly pulp capped with MTA and restored temporarily with IRM.[109] Definitive permanent composite restorations were placed 2 weeks later, after confirmation of pulp vitality. In these cariously exposed immature permanent teeth, the success rate at a 2-year review was 93%.

Another observational investigation examined direct pulp capping after carious exposures of mature and immature permanent teeth completed using MTA in a two-visit sequence.[40] Forty-nine teeth were examined in patients aged 7 to 45 years over a 1- to 9-year period, with an average 3.94-year observation time. The study incorporated a strict protocol that included detector dye–aided caries excavation using the dental operating microscope, 5.25% to 6% sodium hypochlorite (NaOCl) hemostasis, thick MTA placement on pulp exposures and surrounding dentin, coupled with adhesion-based permanent restorations placed at a subsequent visit to compensate for the delayed setting properties of ProRoot MTA. Based on subjective symptomatology, cold testing, and radiographic evaluation, 97.96% of teeth showed a favorable outcome. All 15 patients with immature apices showed continued root formation, with apical closure over a 6- to 10-year period; five patients with large or multiple exposures exhibited pulpal calcification (Fig. 23-8).

The improved survival outcomes seen in this MTA pulp capping study can be credited to changes in established treatment protocols and the incorporation of improved pulp capping materials. Progress in the caries removal process, magnification systems, NaOCl hemostasis, MTA selection, and adhesion technology take advantage of advances in vital pulp therapy to surpass the outcomes seen with accepted traditional methods (Figs. 23-9 and 23-10).

The unique physiochemical properties of MTA also promote a superior environment for pulpal repair and bridge formation, compared to CH products.[162a,204,222,224a,247] MTA is a hygroscopic cement that sets in the presence of blood and serum, produces a gap-free interface with dentin, and generates a sustained alkaline pH; in addition, the surface morphology of the hardened cement allows for predictable bonding with current adhesion systems. Growth factors necessary for hard tissue formation are activated by MTA through the gradual release of calcium ions during cement curing.[190] The small particle size and alkaline pH contribute to the entombment of remaining cariogenic bacteria at the dentin-MTA interface, impeding bacterial ingression and caries progression and discouraging continued pulpal injury.[250,359a]

Pulpotomy with MTA

The decision to remove a small or large portion of the coronal pulp is based on visual inspection of the pulp tissue and the ability to achieve hemostasis after pulp exposure during either caries excavation or exposure as a result of trauma (partial or shallow pulpotomy). The coronal pulp tissue can also be removed completely to the pulp floor or cervical area (pulpotomy) in the case of molars and some premolars.[118,122] The AAPD guidelines state, "A pulpotomy is performed in a tooth with extensive caries but without evidence of radicular pathology when caries removal results in a carious or mechanical

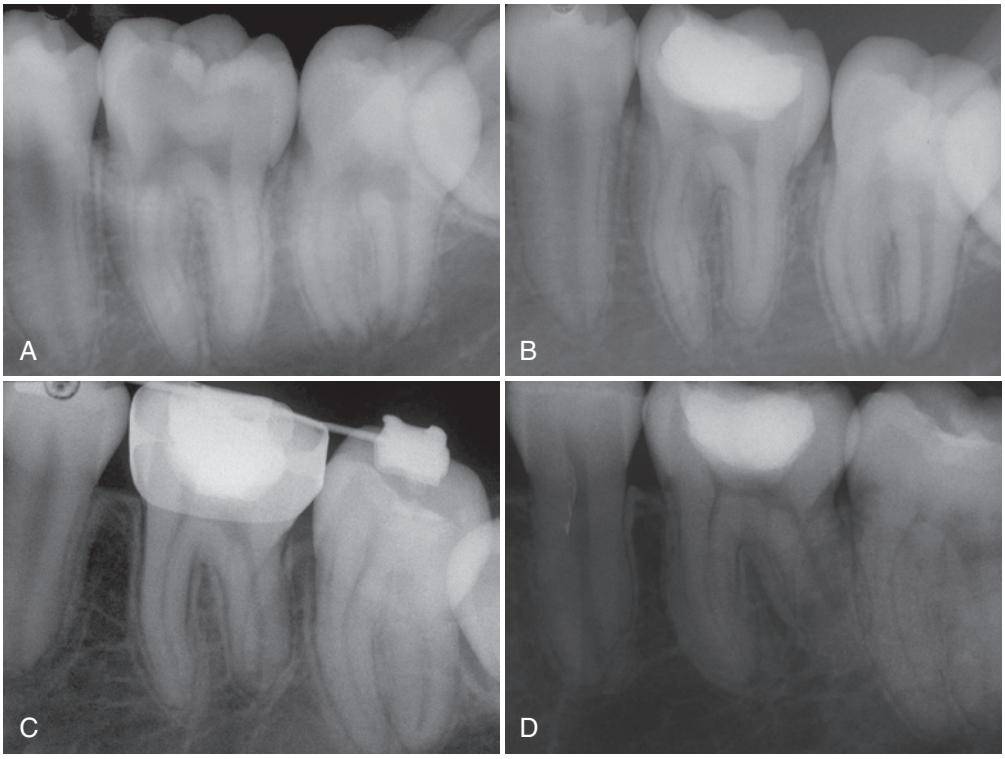

FIG. 23-8 An 11-year-old who presented with deep occlusal caries in a mandibular left first molar. Responses to cold testing were normal, although the patient complained of a sleepless night. **A,** Preoperative radiograph. **B,** Radiograph after direct mineral trioxide aggregate (MTA) pulp caps were applied, followed by composite restoration placement on the second visit. The tooth had two large exposures, 1.5 and 2 mm in diameter. **C,** Three-year radiographic review with molar in full banding during orthodontic treatment. **D,** Radiographic review at 9.5 years showing no evidence of periapical pathosis or notable pulp calcification. Carbon dioxide cold testing indicated normal vitality. (© Dr. George Bogen.)

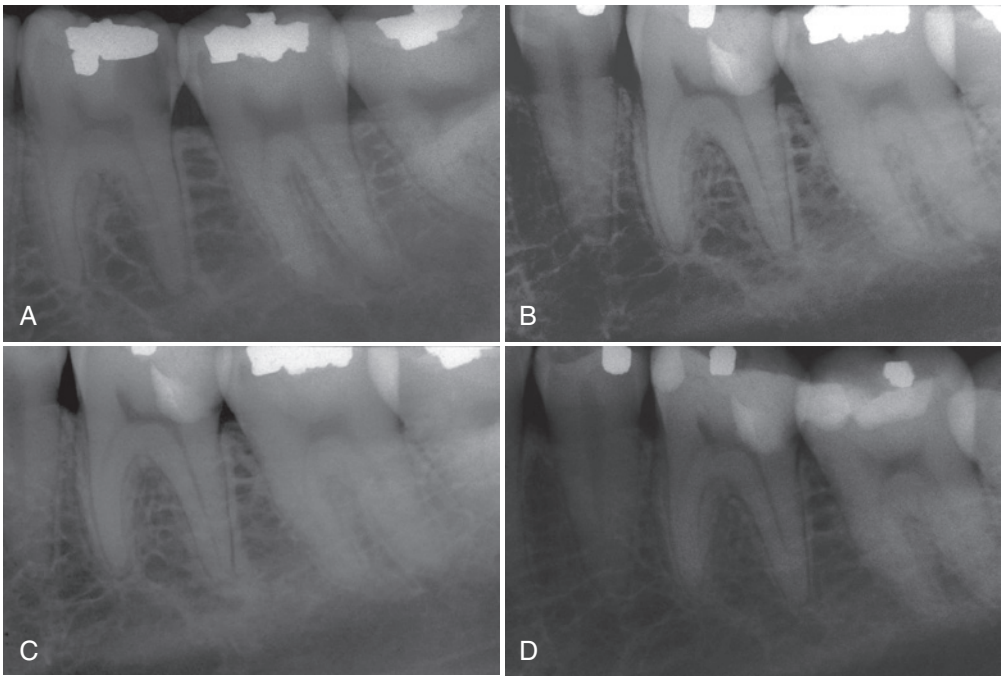

FIG. 23-9 **A,** Deep caries in a partially symptomatic mandibular left first molar in a 29-year-old patient. **B,** Direct pulp cap with mineral trioxide aggregate (MTA); the final restoration was placed during the second visit after MTA curing. **C,** Radiographic recall at 1 year. **D,** Seven-year recall radiograph showing regular periapical appearance. The molar responded normally to cold testing at both follow-up visits. (© Dr. George Bogen.)

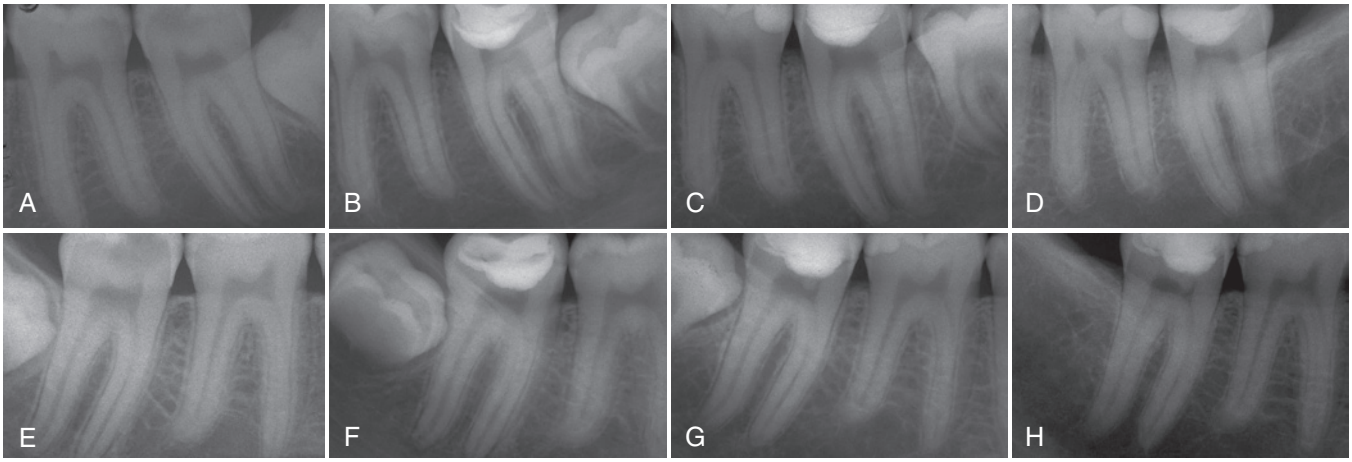

FIG. 23-10 A and E, Radiographs of mandibular left and right second molars with deep distal caries in a 22-year-old patient. B and F, Posttreatment radiographs after direct gray mineral trioxide aggregate (MTA) pulp caps, wet cotton pellets, and Photocore® provisionalization. C and G, Two-year radiographic recalls. D and H, Radiographic controls taken 10 years after pulp capping. Both teeth responded positive to cold tests at the 2- and 10-year recall periods. The third molars had been extracted. (© Dr. George Bogen.)

pulp exposure."[13] If bleeding cannot be controlled after 10 minutes of direct exposure to NaOCl after removal of unhealthy tissue, complete removal of the coronal pulp to the pulp floor is the preferred option.

Sodium hypochlorite serves as an excellent diagnostic tool to differentiate irreversible from reversible pulpitis and to help determine whether to proceed with partial pulpotomy, complete pulpotomy, or pulpectomy. This decision can be of paramount importance in young permanent teeth with open apices, in which removal of tissue contaminated by microorganisms can reverse symptoms and stabilize inflamed tissue.[64,100] Investigations have shown that the proliferative response of pulp tissue after exposure progresses several millimeters into the pulp from the injury site.[77] The removal of 1 to 3 mm of peripheral tissue to access the deeper, healthy tissue, in cases of trauma or when carious exposures reveal inflamed tissue, ensures pulp survival (Fig. 23-11).

Partial pulpotomy for treating direct pulp exposures in immature permanent teeth using CH products has been shown to be a reliable treatment option with proper case selection.* However, improved success rates ranging from 93% to 100% have been demonstrated using MTA for pulpotomies in permanent teeth.[28,272,352] Moreover, pulpotomies completed with MTA in primary molar teeth do not show pathologic complications such as internal resorption, which typically are seen with CH, formocresol, and ferric sulfate.[97]

It has been recommended that pulpectomy be avoided in immature permanent teeth with vital canal tissue so as to protect the remaining radicular pulp tissue and thus encourage continued root development and apexogenesis.[112,287,303] However, with the introduction of MTA, predictable root-end closure and maturogenesis can be achieved with new approaches using regenerative, revascularization, and apexogenesis procedures (Fig. 23-12).† Although controversy remains regarding the type and quality of tissue produced in regenerative procedures for nonvital teeth, current treatment options for immature teeth strengthen roots by increasing wall thickness and root length.[177,211]

Complete pulpotomy for mature irreversibly inflamed permanent molars represents a novel approach in treating symptomatic teeth while preserving pulp canal tissue.[17] A current randomized clinical trial compared full pulpotomies completed with MTA and CEM; the study examined postoperative pain, along with radiographic and clinical outcomes in patients diagnosed with irreversible pulpitis.[18] At a 1-year follow-up for 413 pulpotomized teeth, the clinical success rates were 98% for MTA and 97% for CEM. Similarly, the radiographic success rates were 95% for MTA and 92% for CEM. Most patients experienced a significant reduction in pain intensity postoperatively over a 7-day period. This conservative strategy for irreversibly inflamed teeth may be beneficial for patients in underserved areas globally.

VITAL PULP THERAPY TECHNIQUES

Diagnosis

A differential diagnosis based on symptoms and clinical findings is the goal in the assessment of pulp vitality. However, an accurate determination of the pulpal condition before treatment initiation can be more challenging in younger patients.[304] Establishing a diagnosis of reversible versus irreversible pulpitis in immature teeth can be complicated by subjective symptoms and testing responses that may not accurately reflect the histopathologic condition of the involved pulp.[52] However, efforts should be directed toward the ultimate goal of pulpal preservation and continued apexogenesis in immature permanent teeth.[38,39] A diagnosis of irreversible pulpitis, based on signs and symptoms, along with clinical testing procedures, does not preclude vital pulp therapy options. Regardless of the treatment choice of pulp capping or partial or complete pulpotomy, preservation of the radicular pulp and apical papilla allows for root maturation in cases of trauma or deep caries.[121,122,140,292]

Acceptable diagnostic quality intraoral radiographs of the involved tooth must be taken to evaluate accurately the extent

*References 27, 121, 213, 220, 256, 272, and 332.
†References 39, 41, 110, 129, 169, 244, 322, and 353.

FIG. 23-11 Eight-year-old patient who presented with a crown fracture of the maxillary left central incisor 2 hours after trauma. **A,** Periapical radiograph of the traumatized tooth revealed immature root formation with a horizontal crown fracture. **B** and **C,** Dental dam isolation showing a complicated crown fracture with three pulp exposures. **D,** Incisal view after partial pulpotomy. **E,** Incisal view after hemostasis and mineral trioxide aggregate (MTA) placement. **F,** Control radiograph after 5 years showing apexogenesis with absence of apical pathosis. **G,** Clinical photograph of reattached tooth fragment after adhesive bonding. **H,** Radiographic recall at 7 years with recently placed composite after loss of coronal fragment. Pulp testing showed normal vitality with no evidence of pathosis. **I,** Clinical photograph showing slight discoloration of the composite 1 year after placement. (Courtesy Dr. Katharina Bücher and Dr. Jan Kühnisch, Munich, Germany.)

of root formation and periradicular or furcation changes associated with the periodontal ligament and supporting bone.[219] In young permanent teeth, the stage of root development directly influences the diagnosis and treatment options.[53] Because the faciolingual dimension of most immature roots is greater than the mesiodistal dimension, apical closure may be difficult to determine radiographically.[53,187] Teeth that demonstrate radiographic evidence of deep caries should not be planned for aggressive procedures, such as pulpectomy, without the benefit of thermal (cold) testing (see Fig. 23-1).

Before arriving at treatment decisions, the clinician should carefully assess all available information; the medical history, patient report, radiographic evidence, clinical evaluation, and vitality (cold) testing are recommended. Periodontal probing, mobility assessment, and the presence of any localized swelling or sinus tracts should be recorded during the evaluation. Radiographs, including bite wings and periapical views, should be evaluated for periapical and furcation pathosis, resorptive defects, and pulpal calcification resulting from trauma or previous restorations.

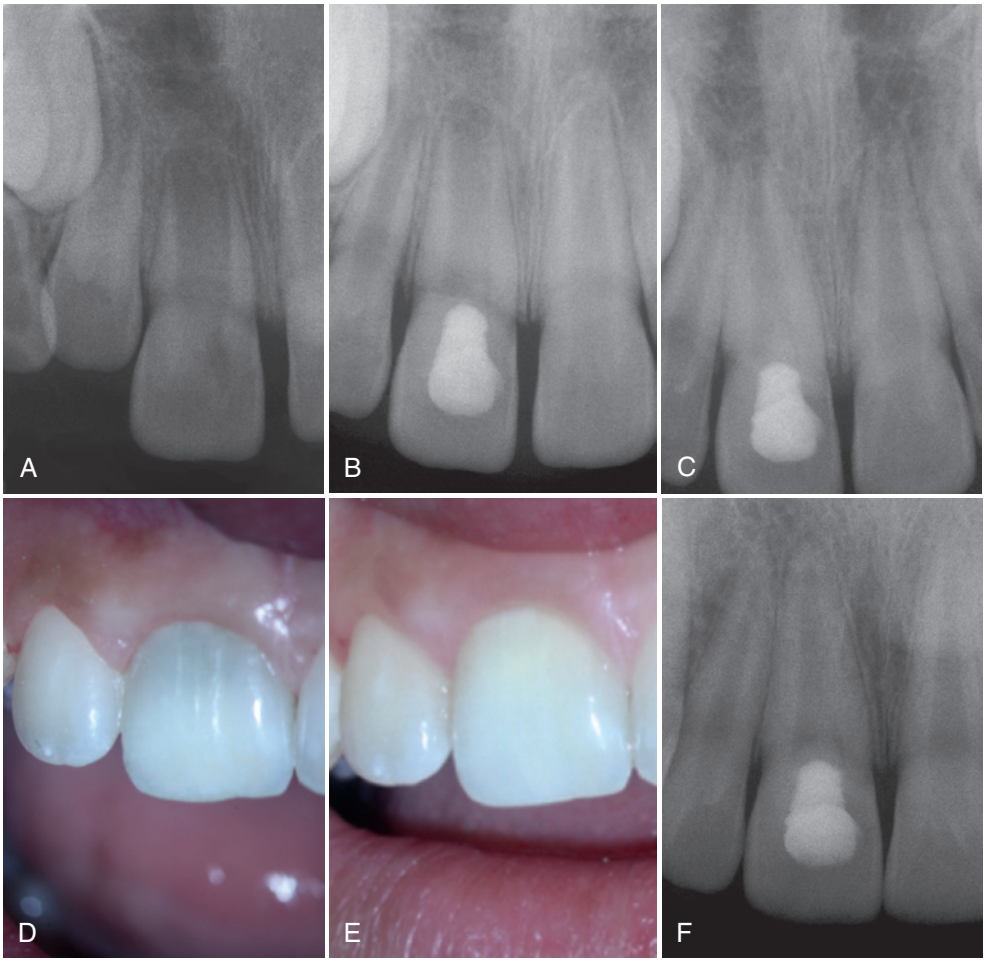

FIG. 23-12 Eight-year-old patient who presented with pulp exposure after traumatic injury. **A,** Periapical radiograph of maxillary right central incisor showing wide-open, immature apex. **B,** Radiograph 6 months after pulp amputation with a high speed diamond bur, saline irrigation, white mineral trioxide aggregate (MTA) partial pulpotomy, and placement of adhesive composite restoration. **C,** Two-year radiographic review; note apical maturation. **D,** Photograph showing coronal staining at 4-year recall. **E,** Photograph after MTA removal, reparative bridge confirmation, and internal bleaching with sodium perborate for 6 days. The hard tissue bridge was covered with a thin layer of phosphate cement before bleaching. **F,** Four-year radiographic review after MTA removal and bonded composite placement. Note presence of thick reparative calcific bridge and complete apical closure. The incisor remained asymptomatic and responded normally to cold testing at all time periods. (Courtesy Dr. Michael Hülsmann, Göttingen, Germany.)

Subjective symptomatology can be reviewed after clinical and radiographic assessments preclude the presence of unconditional irreversible pulpal disease. Patients with deep carious lesions often experience sensitivity to cold, heat, or sweet or acidic foods, and cold tests may evoke a short lingering response of 1 to 2 seconds. This may not be a definitive indicator that the pulp is irreversibly damaged. Determination of the pulpal condition with the aid of contemporary testing methods can be challenging, even for veteran clinicians, because of possible excessive responses to pulp percussion and palpation testing in children.[123,181,350] Clinical evidence indicates that cold testing with carbon dioxide ice is a more reliable prognosticator of pulp status in immature permanent teeth than electronic testing devices.[53] However, a diagnosis of irreversible pulpitis or pulp necrosis should be considered for teeth that generate pain on percussion.

Recent clinical investigations have demonstrated that a diagnosis of symptomatic irreversible pulpitis and acute apical periodontitis may not proscribe pulp capping and pulpotomy procedures when MTA or other CSCs that have been shown to reverse the inflammatory process are used.[64,100] Clinically, the difference between reversible and irreversible pulpitis is often determined on the basis of the duration and intensity of pain.[122] Unprovoked spontaneous pain of long duration or unrelenting symptoms forcing sleep deprivation are consistent with irreversible pulp inflammation or an acute periapical abscess.[350]

Another important consideration in the differential diagnosis is a patient with displacement trauma, which can display a transient apical breakdown radiographically that mimics periapical radiolucencies.[15] Teeth that experience luxation-type injuries can discolor and may not respond to cold testing for up to 4 months before they recover normal color and vitality. Also, biologically or pharmacokinetically immunosuppressed patients may not respond to conventional treatments because of abnormal function of related repair mechanisms.[208,343] Most clinical investigations clearly indicate that successful outcomes

for vital pulp therapy decrease as the patient's age increases. Although aging of the pulp diminishes pulpal volume, vascularity, and host immune responses, functional repair mechanisms can still provide favorable treatment outcomes in older patients (Fig. 23-13).[1,234]

The initial pulpal diagnosis can be confirmed after visualization of the exposed pulp and during hemostasis assessment. If no hemorrhaging is seen, this area of the tissue is most likely necrotic, and the tissue must be removed with a high-speed round diamond bur until bleeding is evident (Fig. 23-14). After hemostasis with NaOCl, a large bulk of MTA can be placed directly against the remaining tissue. Alternatively, if hemorrhage control cannot be achieved after 10 minutes of direct contact with 3% to 6% NaOCl, the pulp is likely to be irreversibly involved, and a full pulpotomy or pulpectomy is recommended.

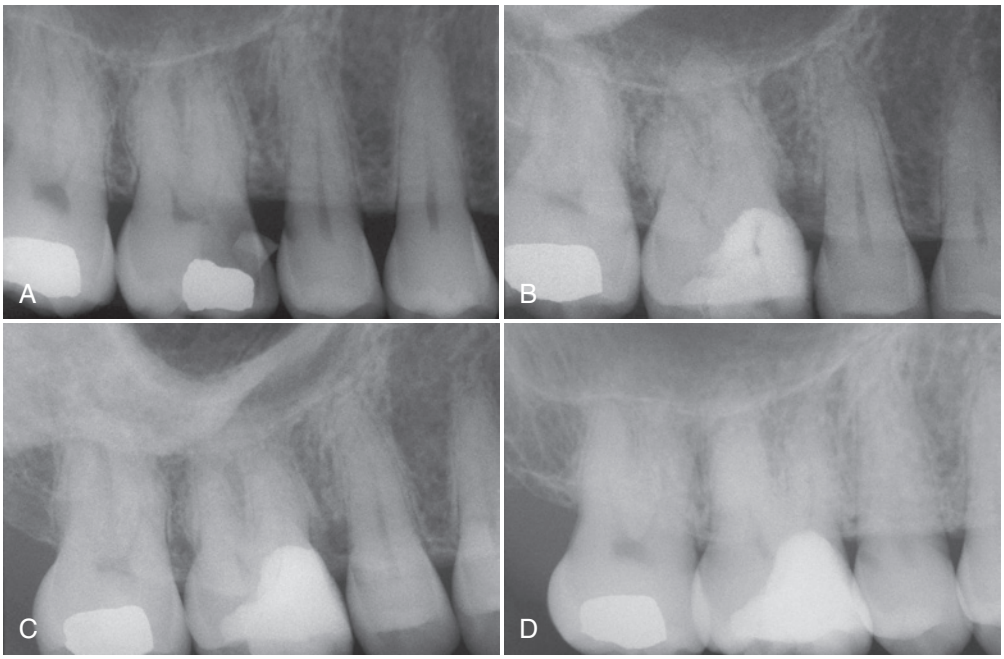

FIG. 23-13 Fifty-one-year-old patient who presented with a deeply carious but asymptomatic maxillary right first molar. **A,** Preoperative radiograph reveals extensive mesial caries and an occlusal amalgam. **B,** Postoperative radiograph after 1.5-mm pulpal exposure, sodium hypochlorite hemostasis, mineral trioxide aggregate (MTA) direct pulp cap, wet cotton pellet placement, and Photocore® provisionalization. **C,** Radiograph 1 week after pulp capping and placement of a permanent bonded composite restoration. The patient was asymptomatic and showed a positive response to cold testing. **D,** One-year radiographic recall; cold testing revealed normal vitality. (© Dr. George Bogen.)

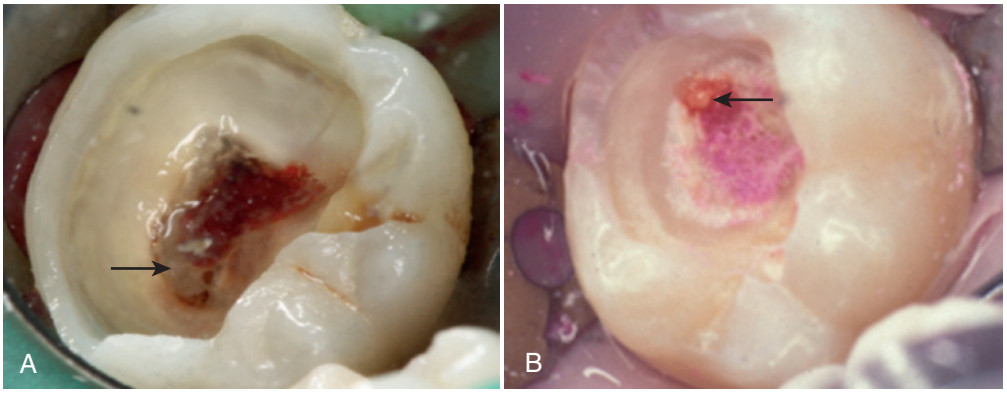

FIG. 23-14 Clinical examples of diseased pulp tissues after sodium hypochlorite hemostasis. **A,** Photograph of exposed pulp tissue of a mandibular right first molar in a 13-year-old patient. Note necrotic pulp tissue *(arrow)* that was subsequently removed with the remaining coronal pulp during a complete pulpotomy procedure. **B,** Clinical presentation of mandibular right molar in a 7-year-old patient after pulp exposure during caries excavation using a caries detector dye. Note the extruded seminecrotic, nonhemorrhagic tissue *(arrow).* The tooth later underwent mineral trioxide aggregate (MTA) partial pulpotomy and permanent restoration. (© Dr. George Bogen.)

Although the size of the pulp exposure has no significant bearing on the final outcome, some clinicians falsely assume that larger exposures have an unfavorable prognosis.[215] Pulp sizes are underestimated on radiographs.[58-60] The size of the pulp exposures may also be overestimated, which could affect the decision-making process, leading clinicians to abandon more conservative vital pulp options.[135] Pulp dimensions may also vary with various racial groups and between genders.[59,296,345]

Teeth that have a history of trauma or previous restorations or that display pulpal calcification have a poorer prognosis than teeth showing only initial caries. In the selection of a specific vital pulp treatment, it is important to consider the remaining tooth structure and future restorative plan. In patients with uncontrolled caries or extensive loss of coronal structure, in which full coverage is indicated, pulpotomy rather than pulp capping is recommended.[43,52,324]

Caries Removal

The main objective in caries removal is the identification and complete removal of the infected tissue while preserving sound tooth structure; this contributes to pulpal protection and continued vitality. Caries removal is enhanced with the aid of a caries detector dye and optical magnification; however, some studies have indicated that dyes can cause excessive and unnecessary removal of healthy tooth structure.[24,218,359]

Caries removal has traditionally been completed somewhat subjectively using hand instruments and slow-speed burs. The procedure is performed using an explorer and tactile sense to differentiate soft from hard dentin to determine infected from noninfected dental tissue. However, this method can have shortcomings because clinicians may leave decay at the dentinoenamel junction and unnecessarily remove dentin that still has the potential to remineralize under a sealed restoration.[124] Furthermore, it has been found that the ability to remove caries varies among operators and during different time periods for the same operator.[124,125]

Investigators in the early 1970s used SEM to identify two different layers of carious dentin.[125] Teeth show two distinct layers of carious dentin as the result of gram-positive bacteria releasing lactic acid as their main by-product. The outside carious layer subjacent to the dentinoenamel junction exhibited demineralized hydroxyapatite crystals that were dissolved by acidic bacterial byproducts; this layer also featured unbound and altered collagen denatured by microbial proteolytic enzymes.[271] A fuchsin dye suspended in propylene glycol was used to reveal that this necrotic and infected layer could be selectively stained, identified, and removed objectively, thus preserving the inner carious layer that remained capable of remineralization. A highly significant difference in the total colony-forming units in stained and unstained dentin has been demonstrated.[364]

The second demineralized carious layer proximal to the pulp featured degraded hydroxyapatite crystals but contained collagen with intact intermolecular cross-links unaffected by cariogenic acids and not stainable with caries detector dyes.[125,126,285] If the second inner layer can be identified and preserved during caries excavation, the remaining pulp tissue and odontoblasts subjacent to the carious zone will be subjected to less trauma, which contributes to pulpal protection and survival.[245,280] The second layer proximal to the pulp has a stronger capacity to remineralize when paired with bonded composite restorations to prevent bacterial microleakage.[233,245]

The two carious layers have been further classified into four zones (pink, light pink, transparent, and apparently normal) when analyzed by atomic force microscopy and transverse digital microradiography.[271] Consistent with previous investigations, the four zones reinforce the concept that increasing levels of demineralization decrease the peritubular dentin rating and mechanical properties of dentin.[271]

Caries detector dyes can be considered a valuable tool in caries excavation when attempts are made to preserve remineralizable dentin and to minimize trauma to the pulp.[87,168,173] Investigations in human, dog, and nonhuman primate models have demonstrated this regenerative characteristic in caries-affected dentin.[178,184,233,319,320] Several studies have questioned the efficacy of caries removal using a caries detector dye. Not all stainable dentin can be classified as infected, and the absence of staining does not eliminate the potential for residual cariogenic bacteria.[185,271] However, dyes allow the operator to visually inspect, under magnification, infected dentin that may have been overlooked, particularly at the dentinoenamel junction, a situation that may compromise the outcome for vital pulp therapy.[185,358] Although a compromise, it may be preferable clinically to inadvertently remove a small excess amount of dentin than to leave infected tissue with active caries.

Hemostatic Agents

A wide range of hemostatic solutions and methods have been recommended to control a bleeding pulp exposure. These include various concentrations of NaOCl; 2% chlorhexidine; MTAD (DENTSPLY Tulsa Dental Specialties, Tulsa, Oklahoma); 30% hydrogen peroxide (Superoxol); ferric sulfate; disinfectants, such as Tubulicid (Global Dental Products, North Bellmore, New York); epinephrine; direct pressure with cotton pellets soaked in sterile water or saline; and the use of lasers.[8] Sodium hypochlorite in concentrations of 1.5% to 6% is currently regarded as the most effective, safe, and inexpensive hemostatic solution for pulp capping and partial and complete pulpotomy procedures.[145,351] First used as a wound antiseptic during World War I and referred to as Dakin's solution, NaOCl became a valuable hemostatic agent in dentistry for direct pulp exposures in the late 1950s.[144,163,310] The antimicrobial solution provides hemostasis and disinfection of the dentin-pulp interface, chemical amputation of the blood clot and fibrin, biofilm removal, clearance of dentinal chips, and removal of damaged cells at the mechanical exposure site.[27,97,144,212] Concentrations of 1.5% to 6% in direct contact with pulp tissue do not appear to adversely alter pulp cell recruitment, cytodifferentiation, and hard tissue deposition.[92] Sodium hypochlorite also shows excellent efficacy as a hemostatic agent at lower dilutions (0.5%).[337]

When direct pulp exposures occur in a carious field, the ability to attain hemostasis remains the most crucial factor in the success of vital pulp therapy. This was demonstrated in an innovative study that examined outcomes of teeth directly pulp capped with a hard-setting CH after pulp exposures were generated during caries excavation.[215] Caries removal during the investigation was aided by a caries detector dye, and 10% NaOCl was used for hemostasis. The 2-year success rate was 81.8%. A statistical analysis of key factors revealed that preoperative thermal responses, percussion sensitivity, the diameter of the exposure, the age of the patient, and the tooth type and location had no significant influence on the outcome. The degree of bleeding and its control at the time of exposure constituted the most critical predictor for outcome assessment.

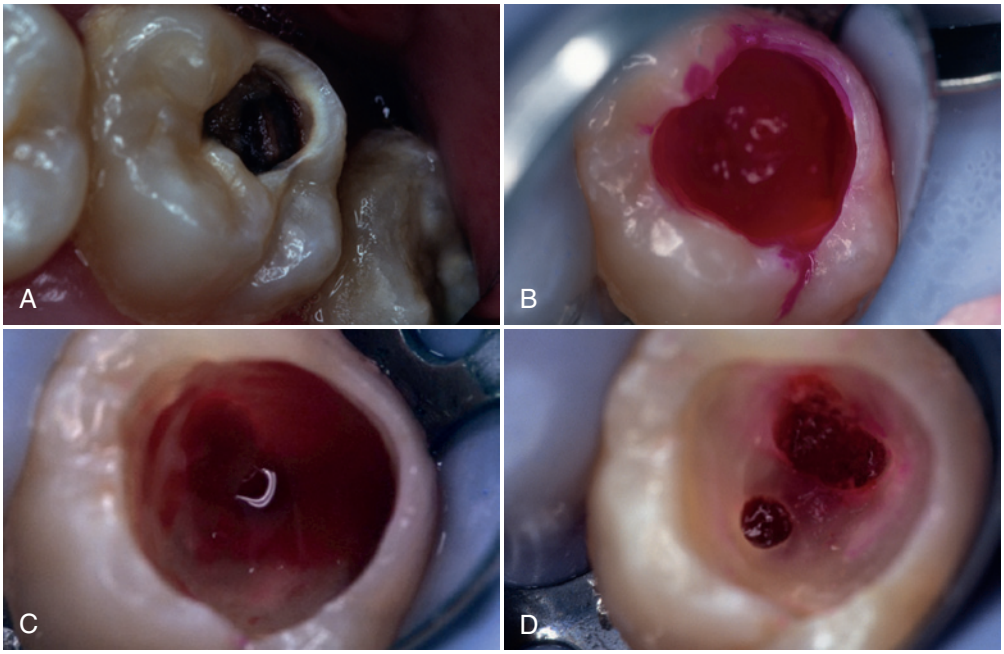

FIG. 23-15 Clinical sequence showing a cariously involved maxillary right first molar in an asymptomatic 20-year-old patient. **A,** Large occlusal-buccal carious lesion. **B,** Caries detector dye staining after initial removal of debris and infected dentin. **C,** Occlusal view of pulp roof exposure with 6% sodium hypochlorite hemostatic solution. **D,** Two large pulp exposures (2 mm and 2 · 4 mm) after caries removal and sodium hypochlorite hemostasis. Note that bleeding is absent and there is no evidence of necrotic tissue. (© Dr. George Bogen.)

When hemostasis can be attained, pulpal repair and reparative dentin formation can proceed normally, in the absence of microbial challenges, when MTA is used as the pulp dressing.[63,179]

Sodium hypochlorite is not only an effective hemostatic agent, it can also be considered an invaluable diagnostic tool for assessing the difference between irreversibly and reversibly inflamed pulps (Fig. 23-15). During the inflammatory process, as cariogenic bacteria approach the pulp, higher levels of immunoglobulins (e.g., IgA, IgG, IgM) and inflammatory markers have been detected, including elastase and prostaglandin E_2.[248] The presence of these mediators can contribute to increased intrapulpal pressure and may play a critical role in the pathogenesis of the irreversibly inflamed pulp.[248,325]

The coronal pulp must be considered irreversibly inflamed when hemorrhage cannot be arrested after 5 to 10 minutes of direct exposure to NaOCl. If bleeding continues, partial or complete removal of the coronal pulp is indicated, with the goal of gaining hemostasis before MTA placement (Figs. 23-16 and 23-17). Sodium hypochlorite has demonstrated effective hemostatic and antiseptic properties in humans without adversely affecting pulp repair, healing, and tertiary dentinogenesis.[92] An investigation into pulp-capped human third molars treated with either CH or a self-etching adhesive system histologically examined pulps at 30 and 60 days after the use of 2.5% NaOCl for hemostasis.[102] Histologic evidence demonstrated no impairment of the repair process after the use of NaOCl, although CH appeared to outperform the resin-based pulp capping agent. Current data clearly support the use of 1.25% to 6% NaOCl solutions in humans as a safe and appropriate hemostatic agent for direct pulp capping and pulpotomies.*

*References 3, 22, 92, 102, 144, 162, 215, 337, 342, and 351.

Treatment Considerations

An important and often unrecognized aspect of direct pulp capping in a carious field is the potential survival of undetected cariogenic bacteria, in dentinal tubules of the dentin adjacent to the exposure site, after caries removal. These microorganisms can compromise treatment even after meticulous caries excavation and NaOCl disinfection. Unfortunately, traditional pulp capping protocols have directed clinicians to place pulp dressings only at the exposure site, without considering the adjacent dentin. Therefore, it is recommended that MTA or CSC be placed over the exposure sites and most of the surrounding dentin with the goal of entombing residual microorganisms.[359a] This notable change in pulp capping strategy can improve treatment outcomes in symptomatic and asymptomatic teeth featuring extensive caries and multiple exposures after caries excavation.[39] Together with an extensive application of MTA or CSC, a cement thickness of 1.5 mm or greater increases the likelihood of bacterial neutralization and minimizes further microbial challenges.

This concept in pulp capping with MTA becomes more critical in one-step pulp capping protocols that use slow-set CSCs covered with RMGI cements. These cements are water-based bonding agents and are not affected by small amounts of water on the dentin or MTA/CSC surface.[86] Unset RMGI cement has an initial pH of about 1.5 and demonstrates no polymerization shrinkage stress, and the material acts as a self-etching primer. Although the bond strength reaches only 25% of comparable resin bonding systems (approximately 10 MPa), the bond is reliable and resistant to disintegration.[86] Although the active chemical constituents are inflammatory and toxic when used for pulp capping and placed directly on exposed pulp tissue, RMGI cements produce only mild inflammatory

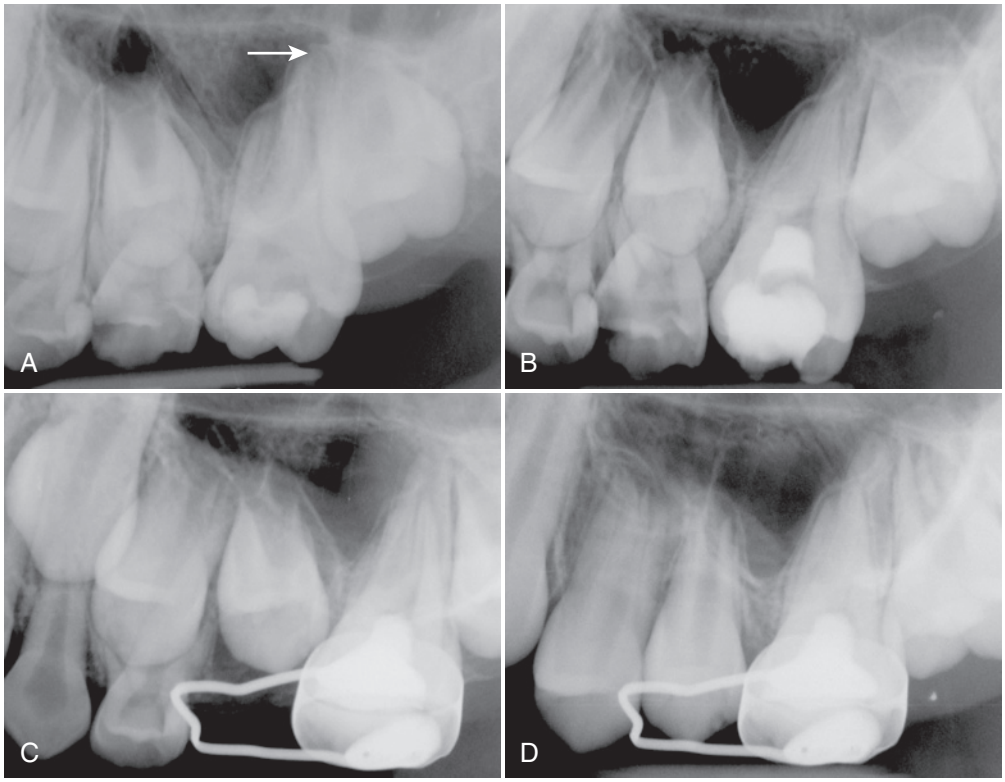

FIG. 23-16 Seven-year-old patient who presented with a symptomatic maxillary left first molar. **A,** Periapical radiograph showing open palatal apex and a coronal provisional restoration. **B,** Radiograph taken after sodium hypochlorite hemostasis and mineral trioxide aggregate (MTA) pulpotomy. **C,** One-year radiographic recall showing advancing apical maturation and a banded space maintainer. The patient was asymptomatic. **D,** Two-year radiographic recall; the tooth showed no pathosis, and the molar was firm and in full function. (© Dr. George Bogen.)

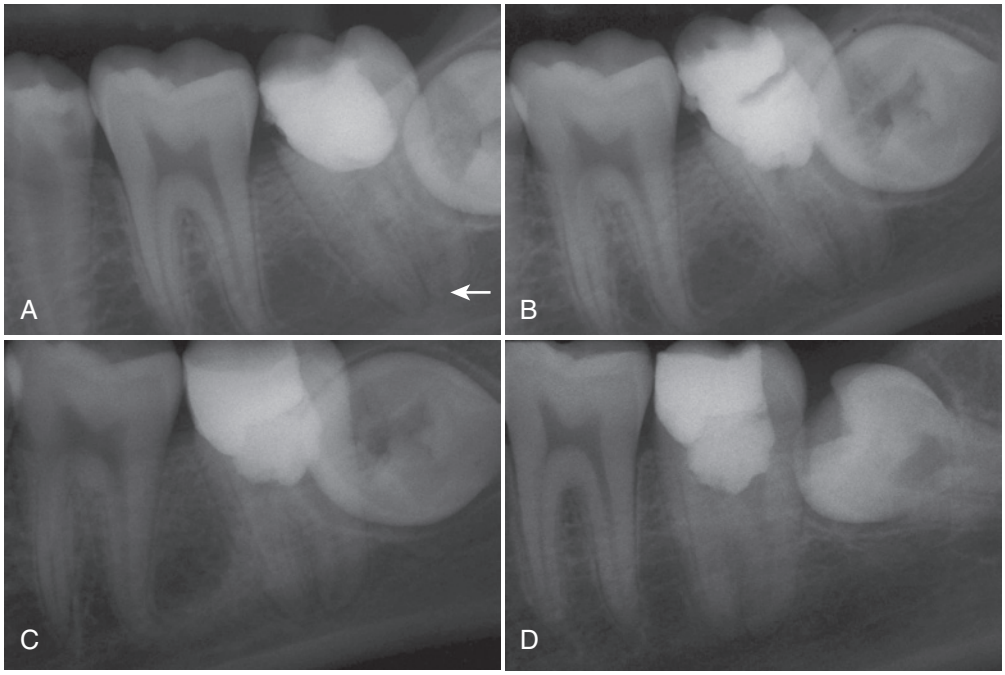

FIG. 23-17 Twelve-year-old patient who received an unsatisfactory previous pulpotomy treatment. **A,** Recently completed pulpotomy in tooth #18 using zinc oxide eugenol (ZOE). Note the carious lesion in the distal aspect of the second premolar and the open apex in the distal root of the second molar *(arrow).* **B,** Radiograph after removal of ZOE and placement of mineral trioxide aggregate (MTA) with a Cavit® provisional. **C,** Radiograph of bonded adhesive composite restoration over cured MTA pulpotomy. **D,** Four-year radiographic control demonstrating maturation and closure of the distal root apex. (© Dr. George Bogen.)

changes to vital pulp tissue when indirectly placed on the remaining dentin.[70,95,159,192]

Resin Modified Glass Ionomer cements have substantial antimicrobial properties, and there are no known physiochemical properties of unset MTA that would adversely affect the set of RMGI materials. However, the challenge during one-step pulp capping procedures with RMGI cements in conjunction with a larger volume of unset MTA is adequate adhesion of the RMGI to the remaining dentin and wet MTA during composite placement.[26] This situation creates great difficulty when contemporary bonding protocols require air blasting during primer and resin placement because the RMGI can be easily dislodged from the unset MTA. In this instance, it may be advisable to place an unbonded amalgam restoration over the RMGI/MTA/dentin interface, if feasible.[250] A reduction in MTA coverage and probable thickness to accommodate RMGI adhesion can compromise favorable outcomes in vital pulp therapy, and either a two-step treatment sequence or fast-set CSCs should be considered.

TREATMENT RECOMMENDATIONS*

Two-Visit MTA Pulp Capping

1. After differential diagnosis, it is determined that the tooth demonstrates either a normal pulp or reversible pulpitis. The tooth should have a normal radiographic appearance and be restorable without crown lengthening procedures. After profound local anesthesia has been obtained, the tooth is isolated with a dental dam and additionally sealed with an agent, such as Oraseal (Ultradent Products, South Jordan, Utah) or a similar product, if saliva leakage is present. The clinical crown is then disinfected with NaOCl or chlorhexidine. Optical magnification and illumination are highly recommended. A high-speed diamond or carbide bur is used to remove undermined enamel, and soft debris is removed with a spoon excavator.
2. After the carious dentin has been exposed and air-dried, a caries detector dye is applied for 10 seconds, and the tooth is washed and dried with a two-way syringe. Caries removal is completed with spoon excavators and/or slow-speed #6-2 carbide round burs until minimal (light pink) or no deeply stained dentin is evident. The dentin is washed and dried again, and the caries detector is reapplied on the dentin for 10 seconds. The process is repeated meticulously until no or only light pink staining is evident (usually 5 to 7 applications).
3. Bleeding can be controlled by the placement of a cotton pellet moistened with 3% to 6% NaOCl for 20 to 60 seconds if pulp exposure occurs during the caries removal process. The staining and caries removal are continued carefully around the exposure site(s) until minimal or no staining is visible. Areas of reparative dentin should be left intact.
4. Direct pulp exposures that occur during caries removal should be hemorrhaging to some degree. A 3% to 6% NaOCl solution, with or without a cotton pellet, is placed directly against the exposure or exposures for a contact time of 1 to 10 minutes. If the tissue appears normal while hemorrhaging, no tissue amputation is indicated. The diagnosis is changed to irreversible pulpitis when hemostasis cannot be obtained within 10 minutes; more aggressive treatment should then be initiated. If bleeding is not evident after pulpal exposure, a high-speed round diamond bur is used to remove tissue until bleeding and healthy tissue are evident. A second application of NaOCl after tissue removal should stop any hemorrhaging. A pulpotomy or pulpectomy must also be considered if the entire axial wall or pulpal roof is removed during caries excavation (Fig. 23-18).
5. The dentin should be gently washed with water and dried to remove excess NaOCl before MTA is applied. The material is mixed according to the manufacturer's instructions (3:1, MTA:H_2O) to a consistency similar to wet sand. An MTA carrier gun or a hand instrument (spoon excavator or Glick) is used to bring the cement to the site in bulk. The MTA should be placed directly over the exposed pulp tissue, including all surrounding dentin. The material is gently patted down with a small moist cotton pellet or a dry pellet if the mixture is too wet. The cement placed should have a minimum thickness of 1.5 mm. MTA inadvertently forced into the pulp chamber will not affect the outcome adversely. For the final bonded restoration to provide an effective seal, a region of dentin and enamel measuring approximately 1.5 to 2 mm should be cleared circumferentially around the MTA with a small (2 mm) moist cotton pellet or sponge brush.
6. A custom-fabricated, flat (1 to 2 mm), moist cotton pellet or gauze is placed over the entire mass of the MTA. The moist pellet or gauze may require placement in two sections if the cavity involves a proximal preparation with an exposure on the axial wall. If the decision is made to complete the restorative treatment on the same day, a large moist cotton pellet can be placed; the patient is instructed not to eat or chew because this may disrupt the unset MTA. A bonded restoration can be placed after a 4-hour waiting period.
7. After placement of MTA and coverage with a wet, flat cotton pellet or gauze, a durable and removable provisional material is used. Unbonded Photocore® (Kuraray America, Inc., New York, New York) is recommended as a reliable option because of its ease of handling and unique polymerization characteristics. Teeth should be conservatively removed from occlusion if thin cusps are present as a result of extensive caries removal. Unless amalgam is the designated restorative material, restorations such as IRM or zinc oxide eugenol (ZOE) (eugenol based) may reduce the bond strengths of adhesive resins and their use with bonding materials remains controversial.[56a,269a]
8. The return appointment can be scheduled 1 to 10 days after MTA placement. The patient is asked about sensitivity, mastication comfort, and pain before the local anesthetic is given. To confirm continued normal vitality before anesthesia, the tooth is cold tested with carbon dioxide (CO_2) ice or Hygienic Endo-Ice (Coltène/Whaledent, Cuyahoga Falls, Ohio). After profound anesthesia is obtained, the tooth is isolated with a dental dam. The provisional material is removed with a high-speed diamond or carbide bur using water coolant. The cotton pellet or gauze is removed, and the embedded cotton fibers are removed with a spoon excavator or similar hand instrument. Magnification and

*Adapted from Bogen and Chandler.[38]

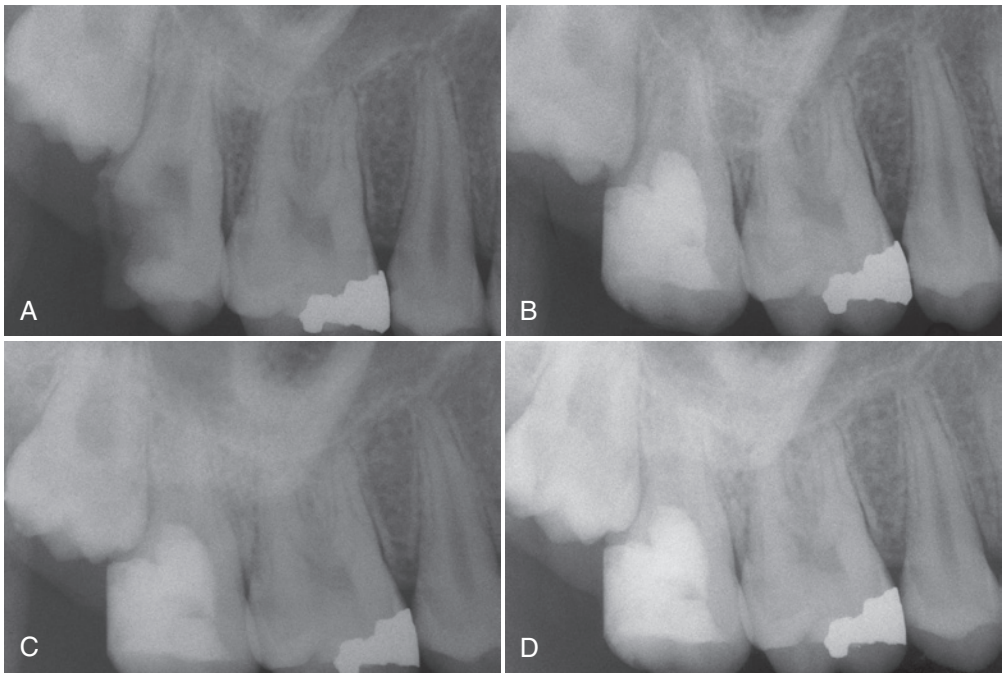

FIG. 23-18 Radiographic sequence of treatment of a maxillary right second molar in a 14-year-old patient, previously recommended for extraction because of advanced caries. **A,** Preoperative radiograph showing extensive caries at the occlusal level of the erupting third molar. The patient was asymptomatic but had pain on cold testing. **B,** Postoperative radiograph after complete exposure of axial wall during caries excavation, mineral trioxide aggregate (MTA) pulpotomy, and placement of flowable resin-modified glass ionomer cement over wet MTA with bonded composite placement. **C,** One-year radiographic recall. **D,** Two-year radiographic control showing absence of apical disease. The molar was functioning normally and without symptoms. (© Dr. George Bogen.)

illumination are strongly recommended to guide the operator. A bonded composite restoration is placed, following the manufacturer's recommendations, after the MTA has been checked to ensure proper curing.

9. After completion of the permanent bonded restoration, the occlusion is checked and adjusted as required. Subjective symptomatology and cold testing should be evaluated at 6 weeks. Radiographic follow-up, cold testing, and subjective symptomatology can be reevaluated at 6 and 12 months if the initial follow-up appears favorable. Yearly or biannual recalls are recommended.

One-Step Pulp Capping

The manufacturer of MTA (ProRoot MTA, DENTSPLY Tulsa Dental Specialties) recommends that pulp capping be completed in one visit. In some instances, treatment of the immature permanent tooth can be difficult, particularly in young patients with challenging medical or behavioral problems that require treatment under sedation. The manufacturer of MTA recommends the following protocol when one-step treatment is indicated.

1. With dental dam isolation, complete a cavity preparation outline using high-speed burs under constant water cooling.
2. If caries is present, excavate using a round bur in a handpiece at low speed or use hand instruments.
3. Rinse the cavity and exposure site (or sites) with 2.6% to 5% NaOCl. Heavy bleeding may be controlled with a cotton pellet moistened with NaOCl.
4. Prepare ProRoot MTA according to mixing instructions.
5. Using a small ball applicator or similar device, apply a small amount of ProRoot MTA over the exposure.
6. Remove excess moisture at the site with a dry cotton pellet.
7. Apply a small amount of Dyract flow flowable compomer (Dentsply International, York, Pennsylvania) (or an equivalent light-cured resin–glass ionomer liner) to cover the ProRoot MTA and light cure according to the material's instructions.
8. Etch the remaining cavity walls with 34% to 37% phosphoric acid gel for 15 seconds. Rinse thoroughly.
9. Dry the cavity gently, leaving the dentin moist but not wet. Apply Prime and Bond NT material or an equivalent bonding material. Cure according to its instructions.
10. Place TPH Spectrum (Dentsply Caulk, Milford, Delaware) composite material or an equivalent composite resin to complete the restoration. Cure according to its instructions.
11. At the next appointment, assess the pulp vitality. Pulp vitality and status should be assessed radiographically every 3 to 6 months or as needed.

The authors recommend the following modifications: Caries removal can be completed under magnification and illumination with the aid of a caries detector dye (step 2). During MTA placement against the exposure site (step 5), a larger bulk of MTA be placed that includes most of the surrounding dentin at a thickness of at least 1.5 mm. One-step pulp capping and pulpotomy procedures can also be completed with the faster-setting hydraulic tricalcium silicate materials.

THE PERMANENT RESTORATION

The placement and quality of the permanent restoration can be crucial to the long-term maintenance of pulp vitality and may be more significant than the actual pulp treatment.[11,114,157,221,227] The aim of the final restoration is to complement the sealing ability of the pulp capping/pulpotomy material and effectively defend the pulp from further microbial challenges. The selection of the restorative material, procedure execution, and adherence to proper restorative protocols can all contribute to minimizing potential restorative microleakage. It has been proposed that microleakage around restorations may be more detrimental to vital pulp tissue than unfilled cavity preparations directly exposed to the oral environment.[284] An important consideration in both mature and immature permanent teeth is the conservation of the remaining tooth structure, which ultimately contributes to favorable long-term retention and function.[69,93,167,301] The incorporation of adhesive restorative materials as definitive final restorations minimizes tooth reduction, encourages anatomic preservation, and thus provides better pulpal protection and repair potential.[124,143]

Technologic advances in dental materials have increased treatment options in the restoration of teeth that have undergone vital pulp procedures. Permanent restorations for pulp capped or pulpotomized permanent teeth can include composite resins, bonded or unbonded amalgam restorations, and cuspal coverage restorations. However, the more conservative the restorative treatment, the greater the probability of pulp survival.[124] Vital pulp therapy must be considered an injury to an already challenged connective tissue, and every effort should be directed toward minimizing further injurious procedures.[1] Factors that affect the repair mechanisms in pulp tissue can include the age of the patient, the depth and size of the cavity preparation, and the choice of restorative material.[246]

Amalgam remains the most widely used restorative material because of its ease of placement, proven durability, and low cost. However, drawbacks include potential health risks to dental personnel, esthetic limitations, and a high modulus of elasticity that can increase the incidence of cuspal fracture or coronal infractions.[9,114] This is a major concern in immature permanent teeth, in which cusps are unprotected, requiring further tooth reduction by placement of stainless steel crowns.[75,106,297,348,350] The implementation of bonding resins or RMGI liners in conjunction with amalgam placement can improve sealing ability and reduce potential microleakage. Even though amalgam restorations remain a safe and predictable long-term restorative option, the technology driving adhesion dentistry continues to improve as new composites and bonding resins evolve.[115,263]

Total-etch and self-etching systems produce excellent bond strength to enamel, dentin, and cured tricalcium silicate cements.[20,249] The most durable bond strengths generated with contemporary materials are achieved using selective etching of the enamel with 40% phosphoric acid, followed by two-step self-etching adhesive systems.[56,253,341] Current advances in adhesive technology have also shown that beneath the resin-interfused hybrid layer, an acid-base–resistant zone is formed that enhances the resistance of normal dentin to recurrent caries. This reinforced layer (termed "super dentin") forms when monomer penetration and polymerization occur, and it demonstrates an ability to restrict (inhibit) primary and secondary caries.[252] As a result of their simplifications and improved bonding strengths, modern adhesives have proved to be a predictable partner that complements vital pulp therapy. It is imperative that all bonding procedures strictly follow the manufacturer's recommendations and include dental dam placement.[56]

POSTOPERATIVE FOLLOW-UP AND RECALL

After treatment completion, the pulp status must be assessed periodically to ensure continued pulp vitality, normal function, and apical closure in immature teeth. Radiographic evaluation and cold testing most accurately assess continued pulp health and are excellent predicators for measuring survival rates. Recalls can address postoperative sensitivity, pulpal degeneration or necrosis, and indications for more extensive endodontic care, such as pulpectomy and root canal treatment Radiographic and clinical review also allows detection of emerging complications, including recurrent caries, poor hygiene, restoration failures, cuspal fractures, and other potentially adverse conditions.

Patient compliance for recall in asymptomatic cases can be challenging compared to patients who had unsuccessful pulp treatments that became irreversibly inflamed or advanced to symptomatic apical periodontitis. Because some parents do not practice regular preventive care and may not have a strong background in basic oral health care, recall compliance rates for children can be unpredictable.[176,270] Although recall intervals have traditionally been based on the conventional 6-month checkup and oral prophylaxis period, this generally accepted practice has come into question for accurate follow-up assessment.[228] It may be preferable to establish recall rates individually, based on patient need, symptomatology, the caries index, periodontal status, and craniofacial development assessment in younger patients.[254,344]

A tentative diagnosis of tooth survivability can be made at the 3-month recall.[215] One study demonstrated that the prognosis for long-term pulp survival can be established at an observation period of 21 to 24 months.[215] A 5- to 10-day review period can be beneficial diagnostically when the patient returns for the final restoration in the two-visit MTA pulp capping protocol. Thereafter, if cold testing reveals normal vitality, recall reviews can be scheduled, if possible, at 6 weeks and at 6 and 12 months depending on patient compliance.[40]

The paramount objective in vital pulp therapy is to promote the physiologic formation and maturation of the root end (apexogenesis). Radiographic evidence of root-end closure in immature adult teeth is a reliable prognostic marker of continued pulp vitality (Fig. 23-19).[23] Apexogenesis after direct pulp capping or pulpotomy procedures using MTA should proceed normally in healthy patients at a progressive rate.[27,269,347,353] The observed root maturation should also follow a predictable pattern of tooth development that coincides with the contralateral teeth in the same patient when the teeth are compared radiographically and viewed chronologically.[23]

Observation of contralateral tooth development can be an invaluable method of measuring the success of vital pulp therapy when using MTA. Alternatively, in traumatized teeth that exhibit necrotic pulps and periradicular pathosis, MTA and CH can be used as an apical plug to stimulate the apical papilla to promote barrier formation, which may otherwise require 5 to 20 months for completion (apexification).[7,65,294]

FIG. 23-19 **A,** Radiograph of a symptomatic mandibular first molar with deep caries in a 7-year-old patient who presented with an open distal root apex *(arrow).* **B,** The patient received a mineral trioxide aggregate (MTA) direct pulp cap and bonded restoration in a two-visit sequence. **C,** Radiographic recall after 14 months showing maturation and apical closure. The tooth showed a positive response to carbon dioxide (CO_2) ice testing. (© Dr. George Bogen.)

If MTA is substituted for CH in vital pulp therapy procedures, similar time periods for apical maturation can be anticipated.[103,223,224]

In the absence of microbial challenges, the human dental pulp demonstrates an exceptional regenerative capacity when treated with MTA and similar bioactive substances. Together with our advances in pulp biology and dental materials, changes in treatment protocols and delivery that encourage dental pulp preservation and survival will ultimately contribute to improved dental health for all patients needing vital pulp therapy.

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CHAPTER 24

Pediatric Endodontics: Endodontic Treatment for the Primary and Young Permanent Dentition

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CHAPTER OUTLINE

The Pulp-Dentin Complex in Primary and Young Permanent Teeth
Differences in Primary and Permanent Tooth Morphology
Root Formation
Primary Root Canal Anatomy
Primary Anterior Teeth
Primary Molars
Clinical Pulpal Diagnosis in Children
History and Characteristics of Pain
Clinical Examination
Palpation, Percussion, and Mobility
Pulp Tests
Radiographic Examination
Restorative Diagnosis: Pulpal Exposures and Hemorrhage
Diagnosis After Traumatic Injuries in Children
Pulp Diagnosis and Treatment Planning After Trauma
Principles of Endodontic Treatment in Children
Pulp Therapy for the Primary Dentition
Indirect Pulp Therapy in Primary Teeth
Direct Pulp Capping in Primary Teeth
Pulpotomy in Primary Teeth
Formocresol Pulpotomy
Alternatives to Formocresol Pulpotomy
Glutaraldehyde Pulpotomy
Ferric Sulfate Pulpotomy
Mineral Trioxide Aggregate Pulpotomy
Electrosurgical Pulpotomy
Laser Pulpotomy
Nonvital Pulp Therapy on Primary Teeth
Pulpectomy in Primary Teeth
Access Openings for Pulpectomy
Root-Filling Materials for the Primary Root Canals
Follow-Up After Pulpectomy in the Primary Dentition
Pulpal Therapy for the Young Permanent Dentition
Indirect Pulp Therapy: Avoiding Pulp Exposure
Managing Pulp Exposure
Pulpotomy
The Cvek Pulpotomy on Immature Permanent Teeth
Partial Pulpotomy on Asymptomatic Young Permanent Posterior Teeth
Pulpotomy on Symptomatic Young Permanent Teeth
Follow-Up After Pulp Capping and Pulpotomy
Formocresol Pulpotomy on Young Permanent Teeth
Apexogenesis
Apexification
Apexification Technique
Artificial Apical Barrier Techniques
Restoration After Apexification
New Horizons for Pulp Regeneration

Despite the dental profession's emphasis on prevention, threats to pulp survival, such as caries, restorative dental treatment, and traumatic injuries, have not been eliminated. As a consequence, children continue to lose teeth prematurely, and procedures aimed at preventing and treating pulp disease in the primary and immature permanent dentitions remain an integral part of contemporary dental practice.

Some may question why efforts to preserve pulpally involved primary teeth are important. These practitioners maintain that such efforts may present a risk to developing permanent successors and, in any case, the primary teeth will be lost before long. Preservation of arch space is one of the principal objectives of pediatric dentistry. Premature loss of primary teeth may cause aberration of the arch length, resulting in mesial drift of the permanent teeth and consequent malocclusion. Whenever possible, the tooth with pulp involvement should be maintained within the dental arch in a functional and disease-free condition.

Other objectives of preserving the primary teeth are to safeguard aesthetics and mastication, prevent aberrant tongue

habits, aid in speech, and prevent the psychological effects that may be associated with tooth loss. Premature loss of the maxillary incisors before 3 years of age has been shown to cause speech impairment that may persist in later years.[229]

It is equally undesirable for children to suffer the unplanned loss of permanent teeth, and it should be noted that the prognosis for lifelong retention of an immature tooth with a short root and fragile dentinal walls is far worse than for a mature permanent tooth. Special treatments for the immature permanent tooth, therefore, focus on preserving vital pulp functions, at least until dental development is complete (see also Chapter 10).

This chapter attempts to provide a review of current therapies for the prevention and treatment of pulpal disease in primary and young permanent teeth. The biologic basis of these procedures is emphasized and supported with clinically relevant evidence to help clinicians preserve functional and disease-free teeth in children.

THE PULP-DENTIN COMPLEX IN PRIMARY AND YOUNG PERMANENT TEETH

Pulp therapies should be based on an understanding of dental tissues and their innate reaction patterns. Some fundamentals of tissue structure and behavior merit review, and the reader is encouraged to see Chapter 12.

DIFFERENCES IN PRIMARY AND PERMANENT TOOTH MORPHOLOGY

Successful pulpal therapy in the primary dentition requires a thorough understanding of primary pulp morphology, root formation, and the special features associated with physiologic resorption of primary tooth roots. These factors are considered in the following sections.

According to Finn[74] and Nelson and Ash,[196] there are 12 basic differences between primary and permanent teeth (Fig. 24-1):

1. Primary teeth are smaller in all dimensions than corresponding permanent teeth.
2. Primary crowns are wider in the mesial-to-distal dimension compared with crown length than are permanent crowns.
3. Primary teeth have narrower and longer roots compared to crown length and width in permanent teeth.
4. The facial and lingual cervical thirds of the crowns of anterior primary teeth are much more prominent than those of permanent teeth.
5. Primary teeth are markedly more constricted at the dentinoenamel junction (DEJ) than are permanent teeth.
6. The facial and lingual surfaces of primary molars converge occlusally; therefore, the occlusal surface is much narrower in the faciolingual dimension than the cervical width.
7. The roots of primary molars are comparatively more slender and longer than the roots of permanent molars.
8. The roots of primary molars flare out from the cervical area, and more at the apex, than do the roots of permanent molars.
9. The enamel is thinner (approximately 1 mm) on primary teeth than on permanent teeth, and has a more consistent depth.
10. The thickness of the dentin between the pulp chamber and enamel in primary teeth is less than in permanent teeth.
11. The pulp chambers in primary teeth are comparatively larger than those in permanent teeth.
12. The pulp horns, especially the mesial horns, are higher in primary molars than in permanent molars.

FIG. 24-1 Cross section of a primary and permanent molar for comparison. Divergence of the primary molar roots allows space for the developing permanent premolar.

Root Formation

According to Orban,[205] development of the roots begins after enamel and dentin formation has reached the future cementoenamel junction (CEJ). The epithelial dental organ forms Hertwig's epithelial root sheath, which initiates formation and molds the shape of the roots. Hertwig's sheath takes the form of one or more epithelial tubes (depending on the number of roots of the tooth, one tube for each root). During root formation, the apical foramen of each root has a wide opening limited by the epithelial diaphragm. The dentinal walls diverge apically, and the shape of the pulp canal is like a wide-open tube. Each root contains one canal at this time, and the number of canals is the same as the number of roots (Fig. 24-2, *A*). When root length is established, the sheath disappears, but dentin deposition continues internally within the roots.

Differentiation of a root into separate canals, as in the mesial root of the mandibular molars, occurs by continued deposition of dentin. This narrows the isthmus between the walls of the canals and continues until the formation of dentin islands in the root canal and eventual division of the root into separate canals. During the process, communications exist between the canals as an isthmus and then as fins connecting the canals (Fig. 24-2, *B*). (See Chapter 12 for a complete description of pulp and dentin formation.)

As growth proceeds, the root canal is narrowed by continued deposition of dentin, and the pulp tissue is compressed. Additional deposition of dentin and cementum closes the apex of the tooth and creates the apical convergence of the root canals common to the completely formed tooth (Fig. 24-2, *C*).

Root length is not completed until 1 to 4 years after a tooth erupts into the oral cavity. In the primary dentition, root length is completed in a shorter period than in the permanent tooth because of the shorter length of the primary roots.

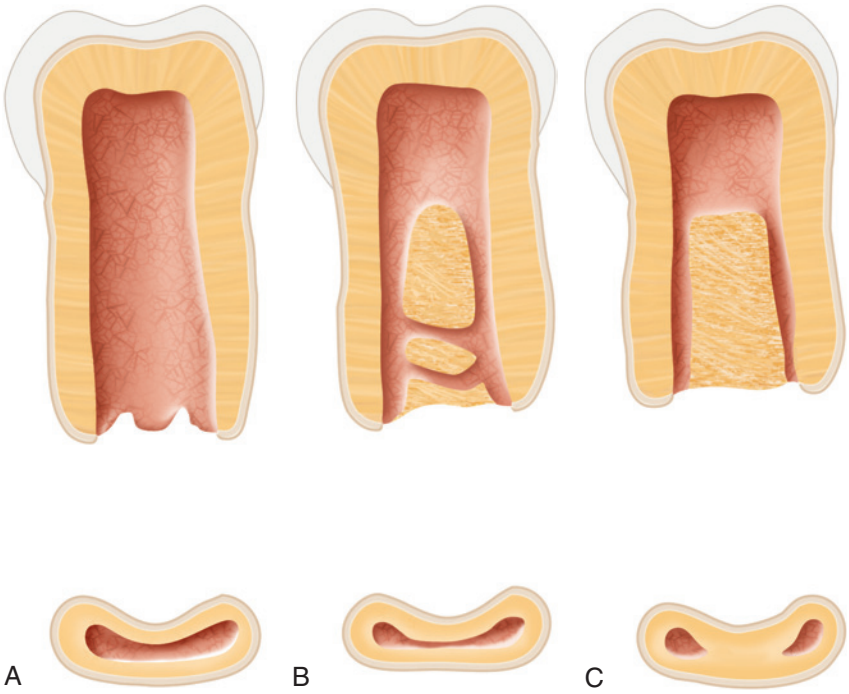

FIG. 24-2 **Faciolingual cross section of the mesial root of a mandibular primary molar. A,** Formation of the root at the time the root length is completed; only one canal is present. **B,** Differentiation of the root into separate canals by the continued, but incomplete deposition of dentin in central areas. Small fins and branches are present between the two canals. **C,** Canals are completely divided; root resorption has begun.

The root-to-crown ratio of the primary teeth is greater than that of the permanent teeth. The primary roots are narrower than the permanent roots. The roots of the primary molars diverge more than those of the permanent teeth; this feature allows more room for the development of the crown of the succeeding premolar (see Fig. 24-1).[194]

The primary tooth is unique insofar as resorption of the roots begins soon after formation of the root length has been completed. At this time, the morphology of the root canals roughly corresponds to the form and shape of the external anatomy of the teeth. Root resorption and the deposition of additional dentin within the root canal system, however, significantly change the number, size, and shape of the root canals within the primary tooth. A more detailed description of how this affects the primary teeth is presented later in this section.

It should be noted that most variations within the root canals of primary and permanent teeth are in the faciolingual plane; dental radiographs do not visualize this plane but show the mesiodistal plane. Therefore, when reviewing clinical radiographs, many of the variations that may be present are not visible. Figures 24-3 and 24-4 show silicone models of root canals, and Figure 24-5 shows a reconstructed digital image to illustrate morphology not visible with plain radiographs. The increasing availability of three-dimensional imaging techniques, such as cone beam computed tomography,[211] is bringing greater understanding of such anatomic features to practicing clinicians.

Primary Root Canal Anatomy

To treat the pulps of primary teeth successfully, the clinician must have a thorough knowledge of the anatomy of the primary root canal systems and the variations that exist within them.[196]

BOX 24-1

Root Canal Anatomy Considerations in Primary Teeth

- After root-length completion, dentin deposition continues in the root canal.
- After root-length completion, dentin deposited in a root canal may change the number, size, and shape of the root canals.
- Often root canal variations are not visible on clinical radiographic images.
- In anterior teeth, one root canal is usually present, although mandibular incisors occasionally have two.
- In anterior teeth, accessory and lateral canals and apical ramifications are rare.

Understanding some of the variations in the primary root canal systems requires an understanding of root formation (see previous discussion and Box 24-1).

Primary Anterior Teeth

The morphology of root canals in primary anterior teeth resembles the form and shape of the roots of the teeth themselves (see Figs. 24-3 and 24-4). The permanent tooth bud lies lingual and apical to the primary anterior tooth, so physiologic root resorption of primary incisors and canines begins on the lingual aspect of the apical third of the roots (see Fig. 24-3, *A*).

Maxillary Incisors

The root canals of maxillary primary incisors are almost round in cross section but somewhat compressed in a faciolingual

FIG. 24-3 Primary central incisors and silicone models of pulp canals. **A,** Facial surfaces. **B,** Beginning resorption of roots on apical third of lingual surfaces. **C,** Models. Pulp canals were injected with silicone, and the tooth structure decalcified away, leaving a model of the root canal systems. Note the division of the canal in the model on the left.

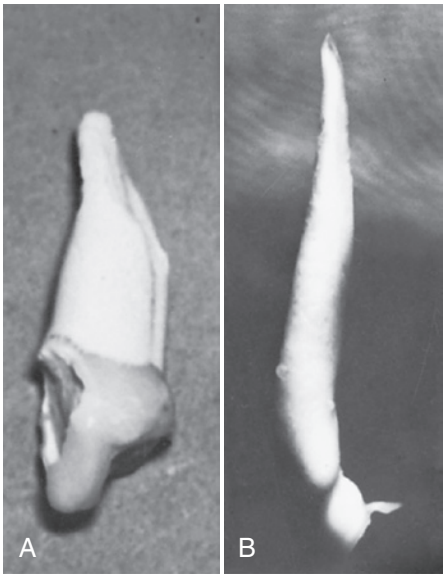

FIG. 24-4 Maxillary primary canine and silicone model of the root canal. **A,** Mesial surface. **B,** Model of the root canal.

direction. Commonly these teeth have one canal without bifurcations. Apical ramifications and accessory and lateral canals are rare but may occur (see Fig. 24-3).[309]

Mandibular Incisors

The root canals of primary mandibular incisors are flattened on the mesial and distal surfaces and are sometimes grooved, progressing in an apical direction to an eventual division into two canals (facial and lingual). The presence of two canals is seen less than 10% of the time. Occasionally, lateral or accessory canals are observed.[309]

Maxillary and Mandibular Canines

The root canals of primary maxillary and mandibular canines correspond to the exterior root shape: a rounded, triangular shape with the base toward the facial surface. Sometimes the lumen of the root canal is compressed in the mesiodistal dimension. The canines have the simplest root canal systems of all the primary teeth and offer few challenges during endodontic treatment. Bifurcation of the canal does not normally occur. Lateral canals and accessory canals are rare (see Fig. 24-4).[309]

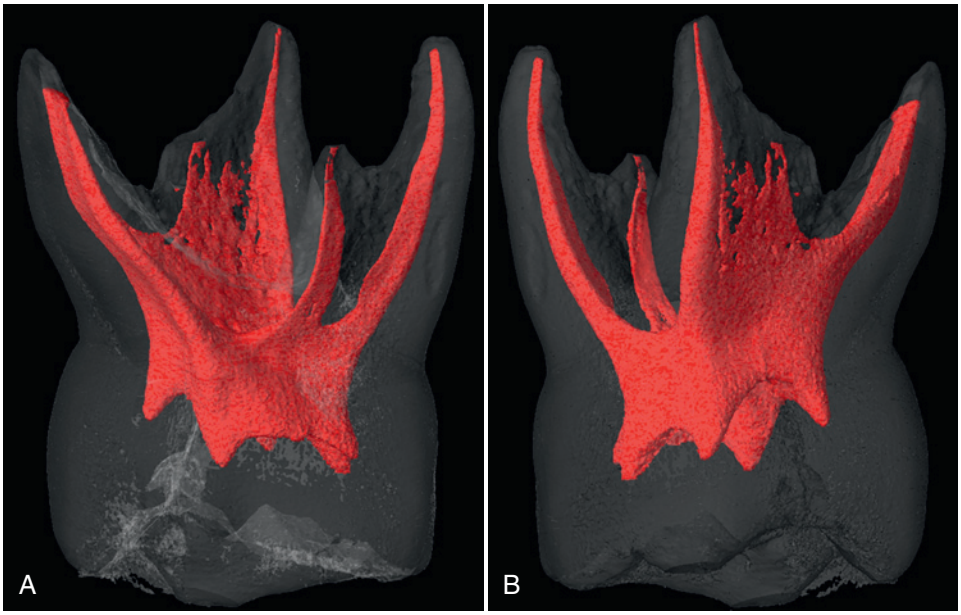

FIG. 24-5 Three-dimensional tomographic images of a maxillary second primary molar. A, Mesial view of primary molar with four root canals. Note the fins of pulp tissue between the distofacial and palatal canals. B, Same tooth from the distal view. Note the wide area of pulp tissue connection between the distofacial and palatal canals. (Courtesy Paul Dummer and Sue Bryant, Cardiff University, Wales, UK.)

Primary Molars

Often, primary molars have the same number and position of roots as the corresponding permanent molars (see Fig. 24-5). Maxillary molars have three roots, two facial and one palatal; mandibular molars have two roots, mesial and distal. The roots of the primary molars are long and slender compared with crown length and width, and they diverge to allow for permanent tooth bud formation.

When full-length root formation has just been completed in the primary molars, only one root canal is present in each of the roots. The continued deposition of dentin internally may divide the root into two or more canals. During this process, communications exist between the canals and may remain in the fully developed primary tooth as isthmi or fins connecting the canals (see Fig. 24-2, *B*, and Fig. 24-5).

In primary teeth, the deposition of secondary dentin has been reported to occur after root-length completion.[21,111,127] This may result in changes to the basic root canal morphology, producing variations and alterations in the number and size of the root canals. This deposition begins at about the time root resorption begins. Variations in form are more pronounced in teeth that show evidence of root resorption.[111]

The greatest variation in morphology of the root canals occurs in the mesial roots of maxillary and mandibular primary molars. This variation originates in the apical region as a thinning of the narrow isthmus between the facial and lingual extremities of the apical pulp canals. Subsequent deposition of secondary dentin may produce complete separation of the root canal into two or more individual canals. Many fine connecting branches or lateral fibrils form a connecting network between the facial and lingual aspects of the root canals (see Fig. 24-5).

The variations found in the mesial roots of the primary molars are also found in the distal and lingual roots but to a lesser degree. Accessory canals, lateral canals, and apical ramifications of the pulp are common in primary molars, occurring in 10% to 20%.[111,309]

In the primary molars, resorption usually begins on the inner surfaces of the roots next to the interradicular septum. The effects of resorption on canal anatomy and root canal filling on the primary teeth are discussed in detail later in this chapter.

Maxillary First Primary Molar

The maxillary first primary molar has two to four canals that correspond approximately to the exterior root form, with much variation. Usually, the palatal root is round and often longer than the two facial roots. Bifurcation of the mesiofacial root into two canals is common and occurs in approximately 75% of maxillary first primary molars.[194,309]

Fusion of the palatal and distofacial roots occurs in approximately one third of maxillary first primary molars. In most of these teeth, two separate canals are present, with a very narrow isthmus connecting them. Islands of dentin may exist between the canals, with many connecting branches and fibrils.

Maxillary Second Primary Molar

The maxillary second primary molar has two to five canals roughly corresponding to the exterior root shape. The mesiofacial root usually bifurcates or contains two distinct canals. This occurs in approximately 85% to 95% of maxillary second primary molars.[111,309]

Fusion of the palatal and distofacial roots may occur. These fused roots may have a common canal, two distinct canals, or two canals with a narrow connecting isthmus of dentin islands between them and many connecting branches or fibrils.

Mandibular First Primary Molar

The mandibular first primary molar usually has three canals, again corresponding approximately to the external root

anatomy, but it may have two to four canals. It is reported that approximately 75% of the mesial roots contain two canals, whereas only 25% of the distal roots contain more than one canal.[111,309]

Mandibular Second Primary Molar

The mandibular second primary molar may have two to five canals, but it usually has three. The mesial root has two canals approximately 85% of the time, whereas the distal root contains more than one canal only 25% of the time.[111,309]

CLINICAL PULPAL DIAGNOSIS IN CHILDREN

Restorative dental treatment should never commence without a working diagnosis and treatment plan. Chapter 1 provides a comprehensive account of diagnostic procedures, but certain points should be emphasized for examination of the child patient. As always, the diagnostic process should follow an orderly pattern, with attention to the medical and dental history, clinical examination, and special tests, including radiographs where appropriate. Parents or caregivers may be helpful in clarifying the case history and may need to be involved if any systemic condition is likely to influence clinical management. The medicolegal dimensions of careful history taking, diagnostic procedures, and case documentation should also be recognized.

Every effort should be made to establish a working pulp diagnosis before anesthetizing the suspect tooth and isolating it with a rubber dam. These conditions are rarely conducive to accurate history taking or diagnostic reliability, especially in young children. The opportunity should also be taken before treatment to discuss contingent alternatives that may become necessary as a result of an unexpected event, such as the exposure of a pulp during deep caries excavation.

Established tests to determine the extent of pulpal inflammation are crude at the best of times and may be of little or no value in young and anxious children and on primary or immature permanent teeth. The literature on pulp diagnosis in children is mostly outcome reports based on empiric treatment and anecdotal case reports. Assumptions about pulpal status before treatment have often been based on retrospective findings rather than histologic or microbiologic data to support the prerestorative diagnosis. Histologic investigation remains the only true method of determining the nature and extent of pulp inflammation, and any correlation with clinical signs and symptoms is limited.[53,172,264]

Although every effort should be made to discern the condition of the pulp before treatment, exhaustive pulp provocation tests may not be helpful in children. From a clinical standpoint, the characteristics of the presenting pain are often critical to the working diagnosis, especially when summated with additional information from the clinical and radiographic examination. Further evidence can be provided by clinical observations during the procedure, such as the nature, volume and ability to control hemorrhage from an exposed pulp. It should be recognized that pulp diagnosis in children is as much an art as a science.

History and Characteristics of Pain

The character of any presenting pain is first identified in the history. Wherever possible, the distinction should be made between provoked and spontaneous pain.

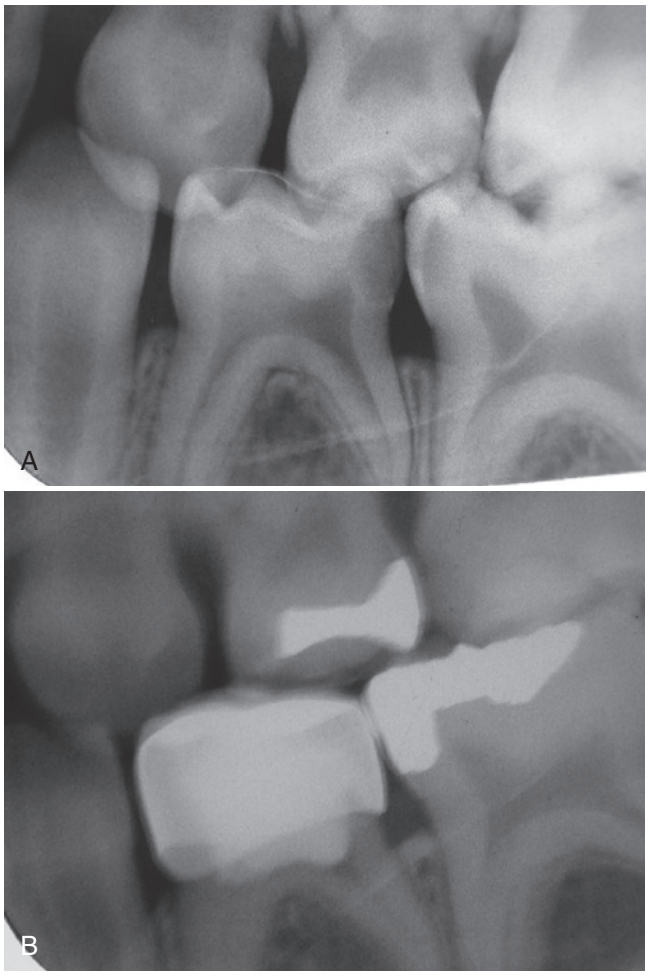

FIG. 24-6 Bone loss (and pain) caused by food impaction in interproximal space between primary molars. **A**, First and second mandibular primary molars with caries and marginal bone loss caused by food impaction. **B**, Healthy bone after restoration of teeth.

Provoked pain is usually triggered by a thermal or an osmotic stimulus (e.g., cold drinks, eating candy) and usually ceases when the stimulus is removed. This history is indicative of minor, reversible pulp inflammation. Provoked pain may sometimes be confused with that caused by interproximal impaction of food (Fig. 24-6), soreness associated with tooth exfoliation, or eruption of permanent teeth.

By contrast, spontaneous pain is not consistently associated with an external stimulus, may arise at any time of the day, or may wake the child from sleep. In both primary and young permanent teeth, spontaneous pain and provoked pain that continues long after the causative factor has been withdrawn are usually associated with extensive, irreversible pulpal inflammation that extends into the root canals.[101] Primary teeth with a history of spontaneous toothache are unreliable candidates for vital pulp therapy and should not be considered for any form of treatment short of pulpectomy or extraction. The situation is quite different for immature permanent teeth. Because the consequences of losing vital pulp functions are so serious, immature permanent teeth with a similar pain history should always be considered for pulpotomy, apexogenesis, or even regenerative techniques in an effort to safeguard tooth development (see later sections on immature permanent teeth).

Clinicians faced with an apparently graphic pain history should not neglect to conduct a proper clinical examination because other conditions, such as papillitis caused by interproximal food impaction, can mimic pulpal pain.

Equally, the absence of pain should not encourage clinical complacency because varying degrees of pulp degeneration or even complete necrosis can be encountered without any report of pain. Consequently, children may present without any complaint, despite extensive carious lesions and a draining sinus tract. Those who have developed early childhood caries (e.g., nursing bottle caries) may have no experience of their teeth feeling any other way and thus no special pain history to report.[214]

Clinical Examination

A careful extraoral and intraoral examination is of great importance in identifying teeth with pulpal involvement. Signs such as tooth discoloration, gross caries, redness and swelling of the vestibulum, or a draining sinus tract may strongly suggest pulpal pathoses. In addition, attention should be paid to teeth with extensive fractured or missing restorations or restorations with recurrent caries. All may indicate involvement of the pulp through the relatively thin and porous dentin of the cavity floor.

Palpation, Percussion, and Mobility

Fluctuation, felt by palpating a swollen mucobuccal fold, may be the expression of an acute dentoalveolar abscess that may not be visualized yet externally. Bone destruction associated with a chronic dentoalveolar abscess may also be detected by palpation.

Although significant inflammatory bone loss can make primary teeth mobile, this is not a reliable, objective test of pulpal status. Teeth with varying degrees of pulpal inflammation may have very little mobility, whereas mobility can be significant during phases of active physiologic root resorption in primary teeth with healthy pulps.

Comparing the mobility of a suspicious tooth with its contralateral equivalent can be especially helpful in clarifying such quandaries. If a significant difference in mobility is noted, this, along with other diagnostic information, may suggest pulpal inflammation or necrosis.

Sensitivity to percussion may reveal a painful tooth in which pulpal breakdown has resulted in acute periradicular periodontitis. Exceptions include recently traumatized teeth. Care should be taken in percussing the teeth of children; Pinkham et al.[214] recommend gentle use of a fingertip rather than the end of a dental mirror.

Pulp Tests

Standard electrical and thermal pulp provocation tests are of limited value in the primary dentition and in young permanent teeth with incompletely developed apices.[7] Although these tests may indicate the presence of some vital responsive tissue, they do not give reliable data on the extent of pulpal inflammation. Many children with perfectly normal teeth do not respond to the electrical pulp tester, even at the higher settings. In addition, young patients may give unreliable responses to such provocation tests because of apprehension, fear, or general management problems.

The unreliability of electrical pulp testing in immature permanent teeth has been reported in several studies.[86,87,89,139] Klein[139] showed responses ranging from 11% in 6- to 11-year-olds with completely open apices to 79% in older children with complete root formation.

Thermal tests may be more reliable than electrical methods for determining the presence of vital responsive tissue in immature permanent teeth, and carbon dioxide snow has been shown to be more effective than ice and ethyl chloride.[86,87,89,139] Heat is unreliable as a diagnostic test in young children.[87]

Laser Doppler flowmetry has been reported to be reliable for diagnosing pulpal vitality in immature permanent teeth,[66,256,306] but the equipment has not been perfected for clinical use[66] and is cost prohibitive. It is also affected by blood pigmentation of the crown, or by the presence of large or full-coverage coronal restorations.

Radiographic Examination

The clinical examination should be supplemented where appropriate with high-quality radiographs. Interradicular (furcal) radiolucencies, a common finding in primary posterior teeth with pulpal pathoses, are best observed in bite wing radiographs. If the apical area cannot be clearly observed, a periapical view should be obtained. The integrity of the lamina dura of the affected tooth should be compared with that of adjacent or contralateral teeth.[214]

Current radiographs are essential in examining for caries, restoration integrity, previous endodontic treatments, and resorptive and periapical changes in primary and young permanent teeth. In children, interpretation of radiographs is complicated by physiologic root resorption of primary teeth and incompletely formed roots of permanent teeth. If the clinician is not familiar with interpreting radiographs of children or does not have radiographs of good quality, these normal conditions can easily be misinterpreted as pathologic changes in need of treatment. In the case of immature permanent teeth, comparison of root formation with the contralateral tooth should always be considered.

The radiograph does not always demonstrate periapical pathosis, nor can the proximity of caries to the pulp always be accurately determined (see also Chapter 2). What may appear as an intact barrier of secondary or tertiary dentin overlying the pulp may actually be a perforated mass of irregularly mineralized and/or carious dentin overlying a pulp with extensive inflammation.[53]

The presence of calcified masses within the pulp is an important diagnostic sign (Fig. 24-7). Mild, chronic irritation to the pulp stimulates tertiary reactionary dentin formation. When the irritation is acute and rapid in onset, the defense mechanism may not have a chance to lay down reactionary dentin. When the disease process reaches the pulp, the pulp may form calcified masses away from the exposure site. These calcified masses can be associated with degeneration of the coronal pulp and inflammation of the radicular pulp in primary teeth.[53] In the absence of other clinical evidence, it is unclear whether this warrants invasive treatment in all cases. Certainly for immature permanent teeth, the presence of calcific metamorphosis within the coronal pulp chamber would not in isolation warrant pulpectomy and root canal treatment.

Pathologic changes in the periapical tissues surrounding primary molars are most often apparent in the bifurcation or trifurcation areas rather than at the apices (as generally seen in permanent teeth) (see Fig. 24-7). Pathologic bone and root resorption indicates advanced pulpal degeneration that has

FIG. 24-7 Calcified mass in the pulp chamber. Internal and external root resorption has occurred. The calcified mass *(arrow)* is an attempt to block a massive carious lesion. Because of the root resorption, this tooth should be extracted. Note the bone loss in the bifurcation area.

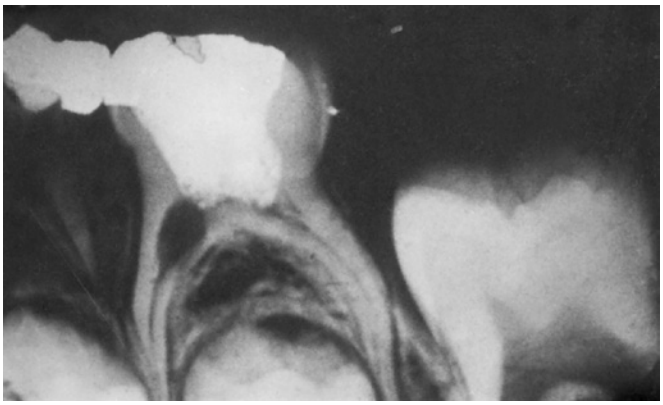

FIG. 24-8 Pulpotomy failure. Note the internal resorption, which has perforated the pulp chamber floor and resulted in a furcal radiolucency.

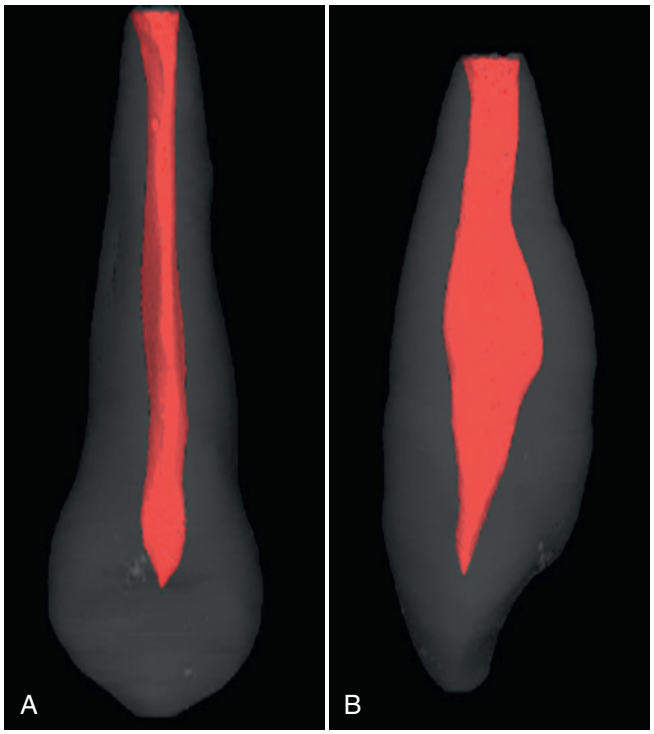

FIG. 24-9 Three-dimensional tomographic images of an immature maxillary canine. A, Faciolingual view. **B,** Mesiodistal view. Such imaging techniques are bringing new anatomic awareness to both the research laboratory and the clinic. (Courtesy Paul Dummer and Sue Bryant, Cardiff University, Wales, UK.)

spread into the periapical tissues. The pulpal tissue may remain vital even with such advanced degenerative changes. Periapical radiolucencies of primary anterior teeth, like those in permanent teeth, are usually at the apices.

Internal resorption occurs frequently in the primary dentition after pulpal involvement. It is always associated with extensive inflammation,[101] and it usually occurs in the molar root canals adjacent to the bifurcation or trifurcation area. Because of the thinness of primary molar roots, once the internal resorption becomes visible on radiographs, it has usually advanced to root perforation (Fig. 24-8). In some instances, however, the process is reversible and self-correcting, and the resorbed area becomes filled with mineralized tissue.[105,208] If a perforation of the root occurs in a primary tooth because of internal resorption, all forms of pulp therapy are contraindicated. The treatment of choice is observation (if the area of the resorption is confined to the tooth) or extraction (if the process has reached the bone).[105,208]

In immature permanent teeth, it is often difficult to assess the extent of apical closure. Plain-film images are frequently misleading because under normal conditions, they show the mesiodistal plane of the tooth but give little information about the faciolingual dimension. Except for the maxillary central and lateral incisor, all other root canals of the permanent teeth are wider in the faciolingual plane than in the mesiodistal plane. The faciolingual aspect of the root canal is two to three times as wide as the mesiodistal width and is the last to become convergent apically as the root develops. Therefore, it is possible to have a dental radiograph showing an apically convergent root canal, whereas in the faciolingual plane, the root canal is divergent. Equally, canals that are apically diverging in the faciolingual view may be parallel or converging in the mesiodistal view. As mentioned, contemporary three-dimensional imaging techniques are beginning to improve understanding, both in the research laboratory (Fig. 24-9) and in the clinic.[211]

In summary, radiographs add to the diagnostic process by visualizing the presence or absence of the following:

1. Deep caries with possible or definite pulp involvement
2. Deep restorations close to a pulp horn
3. A successful or failing pulp cap, pulpotomy, or pulpectomy
4. Changes within the pulp, such as calcific barrier formation, calcific metamorphosis, and pulp stones (denticles)
5. Pathologic root resorption, which may be internal (within the root canal) or external (affecting the root or the surrounding bone). Internal inflammatory resorption indicates inflammation of a vital pulp, whereas external inflammatory resorption demonstrates a nonvital pulp

with extensive inflammation, including resorption of the adjacent bone. External, replacement resorption usually follows trauma and is discussed more fully in Chapters 16 and 20.

6. Periapical and interradicular radiolucencies of bone. In primary molar teeth, any radiolucency associated with a nonvital tooth is usually located in the furcation area, not at the apices. This is because of the presence of accessory canals on the pulpal floor area. A bite wing film is frequently a useful diagnostic aid, particularly in maxillary molars where the developing premolar obscures the furca in a periapical radiograph.
7. The degree of root formation in young permanent teeth

It is important to emphasize once again that the clinician should be familiar with the normal factors that complicate interpretation of radiographs in children: larger bone marrow spaces, superimposition of developing tooth buds, normal resorption patterns of the teeth, and immature root apices.[214]

Restorative Diagnosis: Pulpal Exposures and Hemorrhage

It has been reported that the size of the exposure, the appearance of the pulp, and the color and amount of hemorrhage are important factors in diagnosing the extent of inflammation in a pulp exposed by caries. The presence of excessive[53,214,264] or deep purple[214] hemorrhage from an exposed or amputated pulp is evidence of extensive inflammation in both primary and young permanent teeth. A true carious exposure is always accompanied by pulpal inflammation (see Chapter 13),[53,264] and even a pinpoint carious exposure can be associated with pulpal inflammation ranging from minimal to extensive or even complete necrosis. However, massive exposure in primary teeth is always associated with widespread inflammation or necrosis and makes the tooth a poor candidate for any form of vital pulp therapy. As discussed previously, this rule does not apply for young permanent teeth with incomplete root development, where the premature loss of vital pulp functions is so catastrophic that every effort should be made to safeguard tooth development (see later section on the management of pulpal exposure in immature permanent teeth).

Sometimes a final working diagnosis can be reached only by direct evaluation of the pulp tissue, and a decision about treatment is made accordingly. For example, if a pulpotomy is planned in a primary molar, the bleeding from the amputation site should be normal, and hemostasis should be evident after 2 to 3 minutes of light pressure with a moistened cotton pellet. Significant bleeding beyond this point indicates inflammation of the radicular pulp, and a more radical treatment, such as pulpectomy or extraction, should be considered. Conversely, if a pulp polyp is present and bleeding stops normally after coronal pulp amputation, a pulpotomy may be performed instead of a more radical procedure.[214]

In the case of an immature permanent tooth, persistent hemorrhage after several minutes of sodium hypochlorite application is an indication of serious pulp inflammation, and a tooth initially scheduled for a direct mineral trioxide aggregate (MTA) pulp cap may be a better candidate for a pulpotomy, apexification, or pulp regeneration (see later section on immature permanent teeth).

Guthrie et al.[101] attempted to use the first drop of hemorrhage from an exposed pulp site as a diagnostic aid for determining the extent of degeneration within the pulp. A white blood cell differential count (i.e., hemogram) was made for each of 53 teeth included in the study. A detailed history was obtained, including percussion, electrical pulp testing, thermal tests, mobility, and history of pain. The teeth were extracted and histologically examined. On correlation of the histologic findings with the hemogram and history, it was determined that percussion, electrical and thermal pulp tests, and mobility were *unreliable* in establishing the degree of pulpal inflammation. The hemogram did not give reliable evidence of pulpal degeneration, although teeth with advanced degeneration of the pulp involving the root canals did have an elevated neutrophil count. A consistent finding of the study, however, was advanced degeneration of pulpal tissue in teeth with a history of spontaneous toothache.

The outward clinical signs of inflammation represent a succession of cellular, vascular, and immunologic processes involving many endogenous mediators. Inflammatory mediators (vascular mediated and cell released) and their role in pulpal inflammation have been the subject of much research. The relationship between the concentration of a known cell-released inflammatory mediator (prostaglandin E_2 [PGE_2]) in pulpal blood samples and treatment outcome after vital pulpotomy in extensively carious primary molars has been reported.[297] Thirty-nine primary molars with no history of spontaneous pain had blood samples harvested from the radicular pulp stumps immediately after coronal pulp amputation. Enzyme immunoassay of the samples for PGE_2 detected the inflammatory mediator in all samples. A wide range of concentrations was detected, and it was shown that the concentration of PGE_2 detected correlated positively with radiologic signs of failure after treatment. The authors tempered their findings by describing the dependence upon a single inflammatory mediator to predict prognosis as oversimplistic and suggested the "trauma" of pulp amputation would stimulate prostaglandin production irrespective of the underlying inflammatory status of the tissue. Similarly, a rapid and low-cost, chairside diagnostic kit to assess the extent of pulp inflammation does not yet exist. Therefore, despite research in this area, clinicians still rely upon empiric clinical findings to diagnose the inflammatory status of the pulp.

Diagnosis After Traumatic Injuries in Children

The details of trauma management are considered in Chapter 20. Injuries to the primary dentition are common, occurring in 1 in 3 children by the age of 5 years.[9,94] Diagnosis after traumatic injuries requires consideration of other factors in addition to those previously discussed. The most frequent injury in the primary dentition is tooth displacement that occurs because the bone is less dense and the roots are shorter. Healing varies from normal without sequelae to canal calcification or pulpal necrosis. Canal calcification may vary from an amorphous material resembling osteodentin to partial or complete closure of the canal.[115,234]

Pulp Diagnosis and Treatment Planning After Trauma

Treatment guidelines are almost nonexistent concerning healing and complications after trauma in primary teeth. The

literature is devoid of histologic or microbiologic studies in this area. In a study of 545 traumatized primary maxillary incisors, Borum and Andreasen[27] found that 53% developed pulpal necrosis, and 25% developed canal obliteration. The age of the patient, degree of tooth displacement, concurrent crown fracture, and amount of root resorption were factors influencing pulpal necrosis and calcification. Teeth with a coronal fracture were less likely to suffer mineralized obliteration of the root canal than those that had been luxated.

In deciding on the appropriate treatment, the proximity of the primary tooth to the permanent successor is an important consideration. The treatment least likely to damage the permanent tooth should be chosen.[7,27] Studies present conflicting data on the merits of treating or extracting traumatized primary teeth. Some have suggested no relationship,[8,9] whereas others have shown more extensive developmental disturbances if the primary tooth is treated and preserved.[250]

Transient or permanent discoloration of the crown occurs in approximately 50% of traumatized primary incisors, varying from yellow to dark gray and usually becoming evident in 1 to 3 weeks. The yellow discolorations are frequently associated with canal calcification but are not commonly associated with pulp necrosis.[27,114]

Pulpal necrosis ranging from 50% to 82% has been reported in traumatized primary incisors with dark gray discoloration, compared to 25% in those with no discoloration.[27,114,116,247] Croll et al.[43] pointed out that color change in the absence of other clinical findings is unreliable. The diagnosis of pulpal necrosis is usually based on dark gray color and radiographic evidence of periapical pathology or cessation of root development.

In immature permanent teeth, discoloration of the crown is also one of the best diagnostic indicators after traumatic injuries.[124,129] Yellow or brown-tinted discoloration usually indicates calcification of the pulp space; a gray color is usually associated with pulpal necrosis. A return to normal after transient coronal discoloration[6,38] and transient apical breakdown up to 4 months' duration[38] has also been reported.

PRINCIPLES OF ENDODONTIC TREATMENT IN CHILDREN

Endodontic procedures are undertaken to preserve teeth in a comfortable, functional, and ideally disease-free condition. The following sections describe a range of clinical procedures that aim to achieve these goals in the primary and young permanent dentitions, but they should not be seen merely as technical exercises. Readers should bear in mind that clinical management goes beyond the simple restorative procedure. The general care of anything from a preschool child requiring pulp therapy on a carious primary molar to an 8-year-old who has sustained trauma to an immature maxillary incisor requires special skills. In purely dental terms, the principle adopted is that the best root filling is a healthy pulp, and emphasis is given to methods of pulp preservation in both the primary and young permanent dentitions. Pulpotomy techniques for the partial preservation of pulp tissue are also presented as legitimate therapies—in primary teeth where young and well-perfused tissues combine with the relatively transient nature of the dentition to win success, and in young permanent teeth where therapies strive to maintain the well-perfused and resilient apical pulp, at least until root formation is complete. None of this diminishes the potential for pulpectomy and root canal treatment, should this become necessary, but as new opportunities open for regenerative therapies, traditional approaches to the root canal treatment of pulpless immature teeth may be coming into question.

PULP THERAPY FOR THE PRIMARY DENTITION

Indirect Pulp Therapy in Primary Teeth

Indirect pulp treatment (IPT) in the primary dentition is considered a contemporary, effective approach to the management of a deep carious lesion in the absence of signs or symptoms of irreversible pulp pathosis.[249] It involves the removal of caries to leave a layer of stained dentin at the cavity floor in areas where its removal would result in exposure of pulp tissue. A decision to use this treatment is derived from a thorough pain history, clinical and radiographic examination, direct evaluation of the cavity preparation, and a good knowledge of tooth anatomy and the caries process.[11]

The aims of IPT are to arrest the carious process, provide conditions conducive to the formation of reactionary dentin, and promote remineralization of the altered dentin remaining.[235] This in turn is expected to promote pulpal healing and preserve the vitality of the pulp.

Technique

The clinical steps in IPT may be divided into stages involving partial caries removal, placement of an antibacterial agent, and restoration of the crown in a way that provides the optimum coronal seal.

Some studies of IPT in primary teeth have advocated a two-stage approach.[67,284] After initial partial caries removal without local anesthetic, the cavity was restored for 1 to 3 months, using a reinforced zinc oxide–eugenol (ZOE) cement or glass ionomer restoration. After this, further caries removal and definitive restoration were undertaken under local anesthesia. It has been suggested that this approach may have a use in the very young or very anxious child, but it may be argued that a single-visit approach is more appropriate and successful.[68] Thinner dentin in the primary tooth compared with the permanent tooth may result in a higher risk of pulp exposure if a primary tooth is reentered to remove residual caries.[262]

The following technique is based on a recommended approach[235]:

1. Local anesthetic
2. Isolation with a rubber dam
3. Removal of all caries at the enamel-dentin junction of the cavity, ensuring caries-free walls
4. Judicious removal of soft, deep carious dentin, using large, round steel burs (#6 to #8). Hand excavators should be used only to remove caries at the dentinoenamel junction and should be angled outward at the DEJ, with care taken not to produce a pulp exposure.
5. Placement of an appropriate lining material, such as glass ionomer cement, hard-setting calcium hydroxide ($Ca(OH)_2$). ZOE, or a directly bonded restoration
6. Definitive restoration providing the optimal coronal seal, such as an adhesive restoration or preformed metal (stainless steel) crown

Unfortunately, the evidence currently is insufficient to promote a definitive choice for the lining material placed over the residual stained dentin.[235] Studies show good success rates for resin-modified glass ionomer lining/restoration, self-etching adhesive systems, and $Ca(OH)_2$ linings.[35,36,142]

Although additional prospective clinical evaluation of IPT is required, studies involving the primary dentition show good success rates (over 90% at 3 years).[4,67,68,295] However, it has been suggested that a successful outcome is highly dependent upon obtaining the optimal coronal seal to eradicate the nutrient supply to residual cariogenic microorganisms. When intracoronal amalgam restorations were compared with extracoronal preformed metal (stainless steel) crowns after IPT, failure was 7.7 times more likely with the amalgam restorations. Adhesive restorations also were reported to provide the optimal coronal seal after pulp therapy.[103] Therefore, to secure the best possible outcome after IPT, or indeed after any pulp therapy for the primary tooth, definitive restoration should involve a bonded restoration and/or preformed metal (stainless steel) crown.

The Hall Technique

The Hall technique, an emerging method of managing dental caries in primary molars, is noteworthy in this section on indirect pulp therapy. The technique was introduced by Dr. Norna Hall, a primary care clinician in Scotland, who was overwhelmed by the number of children with dental caries. In Scotland, by the age of just 5 years, 55% of children had visible decay into dentin, and 16% had experienced tooth extraction. In response, this clinician decided to manage lesions in primary molars (that were symptom free and free of radiographic signs of periradicular pathology) by cementing a preformed metal (stainless steel) crown in place without local anesthesia, tooth preparation, or any attempt at caries removal. This is viewed in the United Kingdom as an undoubtedly novel approach to caries management and caught the attention of clinical academics in Scotland. Audit data from Hall's records indicate that her technique may have results similar to those for more conventional approaches in the primary care setting.[126]

Subsequently, a randomized, controlled clinical trial was undertaken comparing the Hall technique with conventional restorations in carious primary molars in primary care.[125] Interestingly, the Hall technique was preferred to conventional restorations by most children, guardians, and clinicians. After a review period of 2 years, in which the teeth managed using Hall preformed metal (stainless steel) crowns were compared with conventional restorations, the "Hall crowns" showed better treatment outcomes for both pulpal health and restoration longevity. In the United Kingdom, this has further stimulated an ongoing debate over whether restorative treatment provided by general clinicians in primary care is an effective way of managing caries in the primary dentition.[70,179]

Certainly this new and novel approach would appear to encompass present-day theory. Potentially cariogenic microorganisms require a very specific environment to start or progress a carious lesion. By sealing the lesion within the tooth, this removes the nutrient supply and stops or slows the lesion's progress.[168] Nevertheless, obtaining an effective and complete coronal seal should be of utmost concern.

Direct Pulp Capping in Primary Teeth

The life span of the average primary tooth from initial development to exfoliation is significantly shorter than that of a permanent tooth. Primary teeth undergo dramatic physiologic and physical changes over a relatively short period. Clinicians should keep in mind that pulp tissue is not static in nature, and outcomes for the same procedure may differ, depending on the age of the patient. Furthermore, because of the aging process within dental pulp, the likelihood of successful pulp capping diminishes with age. This may be explained by the increase in intrapulpal fibrous and calcific deposits seen with aging, together with a reduction in both pulpal volume and pulp fibroblast proliferation.[105]

Direct pulp capping should not be performed on carious pulpal exposures in primary teeth. *Guidelines developed by both the American Academy of Pediatric Dentistry (AAPD) and the British Society of Paediatric Dentistry (BSPD) recommend that direct pulp capping should be reserved only for small mechanical or traumatic exposures in primary teeth.*[54,235] Under these circumstances, it is presumed that the conditions for a favorable pulpal response are optimal.

Pulpotomy in Primary Teeth

The AAPD guidelines for pulp therapy for primary and young permanent teeth describe the pulpotomy procedure in primary teeth as the amputation of the affected or infected coronal portion of the dental pulp, preserving the vitality and function of all or part of the remaining radicular pulp.[54] Evidence of successful pulp therapy includes the features listed in Box 24-2.

Complete amputation of the coronal pulp is the norm for pulpotomy procedures performed on the cariously exposed vital pulps of primary teeth. After this, and once hemostasis has been achieved, a decision is made on the wound dressing or technique to apply to the pulp stumps.

The following are some of the available options. The success of all procedures depends upon ensuring that the residual pulp tissue is correctly diagnosed as healthy or reversibly inflamed.

- Hemostasis and maintenance of vital tissue (e.g., ferric sulfate solution, electrosurgery, laser)
- Dentin bridge formation and maintenance of vital tissue (e.g., MTA)
- Superficial (partial) pulp tissue fixation and maintenance of vital tissue (e.g., dilute formocresol solution, glutaraldehyde solution)

Many pharmacotherapeutic agents have been used for pulp therapy in the past. Formocresol has been the most popular agent, mainly because of its ease of use and good clinical success. Nonetheless, formocresol, and in particular one of its constituents, formaldehyde (FAD), has come under close

BOX 24-2

Evidence of Successful Pulp Therapy

- Vitality of most of the radicular pulp
- No prolonged adverse clinical signs or symptoms (e.g., sensitivity, pain, or swelling)
- No radiographic evidence of internal resorption reaching the alveolar bone
- No breakdown of periradicular tissue
- No harm to permanent successor teeth
- Pulp canal obliteration (abnormal calcification)—not considered a failure

scrutiny because of reported concerns related to the systemic distribution of FAD and its potential for toxicity, allergenicity, carcinogenicity, and mutagenicity. Other medicaments (e.g., glutaraldehyde, $Ca(OH)_2$, collagen, ferric sulfate, MTA) have been suggested as possible replacements. However, varying success rates and questions about the safety of these materials make it clear that additional research is required on the use of these and other pharmacotherapeutic agents.

Nonpharmacologic hemostatic techniques have been recommended, including electrosurgery[163,197,237,253,254] and laser therapy.[62] Research involving human clinical trials on both these techniques is sparse; nevertheless, electrosurgical pulpotomy currently is taught in several dental schools. Comprehensive reviews of the techniques and agents used in vital pulp therapy and discussion of possible new modalities are available in the literature.[223,262,296]

Indications and Contraindications for Pulpotomy

Pulpotomy is indicated for *pulp exposure on primary teeth in which the inflammation or infection is judged to be confined to the coronal pulp.*[137,278] If inflammation has spread into the tissues within the root canals, the tooth should be considered a candidate for pulpectomy and root canal filling or extraction. The contraindications to pulpotomy on a primary tooth are outlined in Box 24-3.

Technique

Pulpotomy is used for primary teeth with radicular pulp tissue judged to be free of inflammation and infection. Compromise on this principle leads to a diminished success rate. If inflamed vital (coronal) pulp tissue is amputated to leave residual healthy pulp tissue (radicular), the tissue remaining has the capacity to remain healthy if managed correctly. The overall success of vital pulp therapy in the primary dentition depends upon several factors:

- Effective control of infection
- Complete removal of inflamed coronal pulp tissue
- Appropriate wound dressing
- An effective coronal seal during and after treatment

An accurate diagnosis of pulp status is very important in aiding appropriate pulpal management. However, this can be challenging, particularly in young children. The following points highlight the diagnostic aids available.

- An accurate pain history is helpful for determining the possible stage or extent of pulpal inflammation, but it may be difficult to elicit such information from a child.
- Radiographic findings can guide treatment decisions. Pretreatment radiographs are essential to assess the extent of the carious lesion and its proximity to the pulp horns or chamber. They also provide information about periradicular pathosis.
- Clinically, pulpal involvement can be assessed immediately after caries removal by a very careful search for evidence of a pulp exposure.

BOX 24-3

Contraindications to Pulpotomy in a Primary Tooth

- History of spontaneous toothache (not caused by papillitis resulting from food impaction)
- Nonrestorable tooth where a postpulpotomy coronal seal would be inadequate
- Tooth near exfoliation; or, no bone overlies crown of the permanent successor tooth
- Evidence of periapical or furcal pathosis
- Evidence of pathologic root resorption
- Pulp that does not bleed (necrotic)
- Inability to control radicular pulp hemorrhage after coronal pulp amputation
- Pulp with serous or purulent drainage
- Presence of a sinus tract

Technique for Coronal Pulp Amputation

Removal of the coronal pulp tissue is a process common to whichever pulpotomy procedure is chosen for the cariously exposed vital primary tooth. After successful coronal amputation and hemostasis (Fig. 24-10, *A* and *B*), the subsequent management of the radicular pulp stumps is defined by which pulpotomy technique the clinician chooses. For example, once hemostasis has been achieved, a wound dressing may be applied to the pulp wounds or, alternatively, the exposed tissue might be subjected to electrosurgery or laser application. Figure 24-10, *C* to *H* provides a schematic overview of the options and expected outcomes.

Stages of the Technique for Coronal Pulp Amputation

1. After the initial diagnosis of probable vital pulpal involvement, the primary tooth is anesthetized and isolated with a rubber dam.
2. All caries is removed, and the observation of bleeding from exposure sites indicates vital (if inflamed) coronal pulp tissue (Fig. 24-11, *A* and *B*).
3. The entire roof of the pulp chamber is removed using a high-speed non–end cutting bur and copious water spray.
4. All the coronal pulp is amputated with a slow-speed #6 or #8 round bur or spoon excavator. Care must be exercised to fully unroof the chamber and extirpate all filaments of the coronal pulp (Fig. 24-11, *C* and *D*). If any filaments remain in the pulp chamber, hemorrhage will be impossible to control.
5. The pulp chamber is thoroughly washed with sterile water or saline to remove all debris, and the site is dried by vacuum and sterile cotton pellets.
6. Hemorrhage is controlled by slightly moistened cotton pellets (wetted and blotted almost dry) placed against the stumps of the pulp at the openings of the root canals. Completely dry cotton pellets should *not* be used; fibers of the cotton will be incorporated into the clot, and when the pellet is removed, hemorrhage will result. Dry cotton pellets are placed over the moist pellets, and pressure is exerted on the mass. Hemorrhage should be controlled in this manner within 3 minutes. It may be necessary to change the pellets to control all hemorrhage.
7. If bleeding persists, the clinician should carefully check that all tissues of the pulp were removed from the pulp chamber and that the amputation site is clean. If the bleeding persists from one of the canals, that canal can be reentered with a small round bur to amputate the suspected inflamed tissue; the canal then is rinsed again, and cotton pellet pressure is applied.

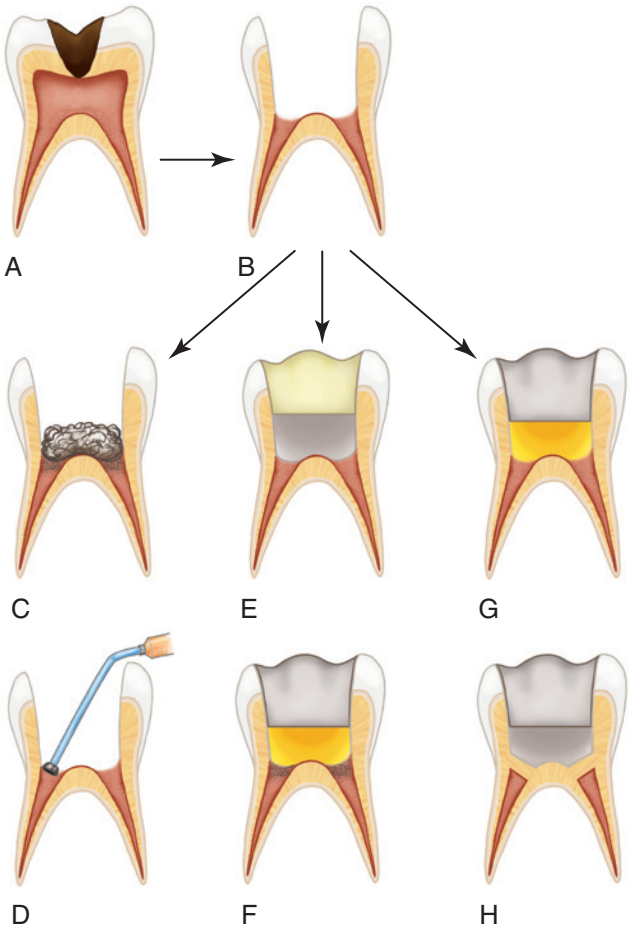

FIG. 24-10 Three different approaches to the radicular pulp after coronal pulp amputation during pulpotomy in an extensively carious primary molar. **A,** Primary molar with caries extending to the pulp. Note the inflammatory response in the coronal pulp. **B,** The tooth has undergone removal of the coronal pulp. The radicular pulp tissue is healthy and has stopped bleeding. **C,** A 1:5 dilute solution of formocresol may be applied to the pulp stumps for 5 minutes. This produces partial tissue fixation, which is greatest near the point of application. **D,** Intracoronal restoration: zinc oxide eugenol (ZOE) is placed directly over the pulp stumps, and an intracoronal restoration is placed. Vital radicular pulp may remain apically. **E,** A 15.5% ferric sulfate solution is applied to the pulp stumps for 15 seconds. This produces mechanical blockage of open capillaries by a protein-iron complex. **F,** Intracoronal restoration has been provided as in **D.** Healthy, vital pulp tissue remains in the root canals. **G,** Mineral trioxide aggregate (MTA) is placed directly over the radicular pulp stumps, and an intracoronal restoration is provided as before. **H,** The radicular pulp tissue remains vital beneath the MTA; a calcific bridge may form over time beneath the MTA.

8. If hemostasis is not achieved within 2 to 3 minutes, the pulp tissue within the canals is probably inflamed, and the tooth is not a candidate for a pulpotomy. The clinician should then proceed with pulpectomy, or the tooth should be extracted.

Once bleeding has stopped at the radicular pulp stumps, the wounds are managed according to one of the following pulpotomy techniques (see Fig. 24-10):

- Application of dilute formocresol solution for 5 minutes
- Application of 15.5% ferric sulfate solution for 15 seconds (Fig. 24-11, *E* and *F*)

BOX 24-4

Formulation of a One-Fifth Dilution of Formocresol Solution

Mix 1 part Buckley's formocresol solution with:

- 1 part distilled water
- 3 parts glycerin

- Permanent placement of MTA
- Electrosurgical manipulation of the wound surfaces
- Laser manipulation of the wound surfaces

Figure 24-11, *G* shows the treated tooth restored with a preformed metal crown.

Formocresol Pulpotomy

Technique (See Fig. 24-10, *C* and *D*)

1. After coronal pulp amputation and once hemostasis has been achieved, a cotton pellet moistened with *one-fifth dilution formocresol solution* (Box 24-4) is blotted to remove excess formocresol and then placed in direct contact with the pulp stumps for 5 minutes. Formocresol is caustic and creates a severe tissue burn if allowed to touch the gingiva.
2. When the pellet is removed, the tissue appears brown, and no hemorrhage should be evident.
3. If an area of the pulp was not in contact with the medication, the procedure must be repeated for that tissue. Small cotton pellets for applying the medication usually work best because they allow closer approximation of the material to the pulp.
4. A cement base of ZOE is placed over the pulp stumps and allowed to set. The tooth may then be restored permanently.
5. The restoration of choice is a preformed metal (stainless steel) crown for primary molars. On anterior primary teeth, a composite tooth-colored restoration is the treatment of choice unless the tooth is so badly broken down that it requires a crown.

The use of formocresol in dentistry remains controversial. Formaldehyde, a volatile organic compound, is toxic and corrosive, especially at the point of contact. Formocresol's other active constituent, cresol, is also an irritant and corrosive in nature.[144]

Local and Systemic Accumulation of Formaldehyde

Localized accumulation of formocresol or FAD has been demonstrated in pulp, dentin, periodontal ligament, and bone surrounding the apices of pulpotomized teeth.[88,190]

Although animal studies have identified radioisotope-labeled formocresol or FAD in major organs after systemic injection or multiple pulpotomies, researchers concluded that the doses of formocresol were far in excess of those used in normal clinical practice. Therefore, it has been suggested that the findings should not be extrapolated to clinical use in humans.[187-189] More recently, a review of the safety of formocresol, including FAD metabolism, suggested that FAD is rapidly metabolized, so the findings of previous studies may have been identifying FAD metabolites systemically and not FAD itself.[178] Notwithstanding this, the amount of formocresol

FIG. 24-11 Clinical stages of a vital pulpotomy using ferric sulfate solution. A, Extensive dental caries affecting a mandibular first primary molar. Note the proximity of the radiographic lesion to the mesial pulp horn. **B,** Caries removal, showing a carious pulpal exposure; bleeding is evident. **C,** Partial unroofing of the pulp chamber; note the bleeding coronal pulp before amputation. **D,** Roof of pulp chamber removed completely. **E,** A 15.5% solution of ferric sulfate is applied to the radicular pulp stumps with a dento-infuser tip supplied by the manufacturer. **F,** Hemostasis is evident at the radicular pulp stumps. **G,** Definitive restoration involves placement of zinc oxide eugenol, overlaid with glass ionomer intracoronally, followed by a preformed metal (stainless steel) crown. (Courtesy Vidya Srinivasan, Edinburgh Dental Institute, Scotland, UK.)

absorbed systemically by way of the pulpotomy route is small and may not contraindicate use of the drug.[210]

Formocresol as an Allergen

The results of studies investigating the allergenic risks of formocresol are equivocal. Studies have shown no evidence of an allergic response in nonpresensitized animals,[258] and presensitized animals showed only a weak allergic potential.[292] However, demonstration of an immune response to formocresol-fixed, autologous tissue implanted in connective tissues or injected into root canals has been reported.[24,293]

Is Formocresol Carcinogenic?

With respect to mutagenicity and carcinogenicity, it is generally accepted that FAD is genotoxic in vitro, inducing mutations and DNA damage in cells from a variety of organisms, including humans.[96,200,207,272,302] The possible link between FAD and carcinogenesis has been investigated in the field of occupational health medicine. In a review of several large longitudinal studies, the International Agency for Research on Cancer (IARC) reported that *"sufficient evidence"* exists that FAD had caused nasopharyngeal cancer in humans.[32] This was suggested to be linked to a localized combination of irritation and genotoxicity of FAD, which repeat-dose inhalation studies using rodents has corroborated.[209] In the dental context, the amount of FAD in a diluted solution of formocresol is small, but no data are available on the amounts of FAD vapor inhaled by patients or dental personnel during pulpotomy procedures and whether this may constitute a potential risk. Moreover, the often disregarded cresol ingredient itself may pose a genotoxic risk to mammalian cells.[104] Formocresol solution used in pulpotomies in children has been reported as genotoxic after postpulpotomy harvested lymphocyte cultures displaying significantly increased chromosomal aberrations were compared with nonpulpotomized controls.[160]

Despite these concerns, the formocresol pulpotomy continues to be one treatment choice available for primary teeth with vital, carious exposures of the pulp in which inflammation or degeneration is judged to be confined to the coronal pulp. The last reported worldwide survey of dental schools (in 1989)[13] showed that most pediatric dentistry departments and pediatric dentists advocated the formocresol pulpotomy technique, and it may still be widely used in clinical practice. In the United Kingdom, since 2004, a general trend away from using formocresol has been noted, driven by several factors, such as difficulty obtaining the medicament, concerns related to its safety, and promising clinical results for newer, nonaldehyde techniques.[296] However, the recent U.K. clinical guideline, Pulp Therapy for Primary Molars, still includes the formocresol pulpotomy as an option,[235] as do the AAPD guidelines.[54] Although formocresol pulpotomy is still taught in predoctoral pediatric dentistry programs in the United States,[218] consensus is lacking on its use for vital pulp therapy in primary teeth.

The current formocresol pulpotomy technique is a modification of the technique reported by Sweet in 1930.[271] The effect of formocresol on pulp tissue (i.e., the amount of tissue fixation) is controlled by the quantity that diffuses into the tissue and depends on the duration of application, the concentration used, the method of application, or a combination of these factors.[63,90,167,173]

A one-fifth dilution formocresol solution has been widely advocated based on the outcomes of both in vitro and in vivo studies comparing dilute formocresol (see Box 24-4) with the undiluted solution (Table 24-1). Histochemical investigations comparing dilute and undiluted formocresol noted little difference between initial effects on pulp tissue fixation but earlier recovery of enzyme activity and improvement in the rate of recovery from the localized cytotoxic effects of formocresol with diluted formocresol.[158,159,268] Clinical studies have shown diluted formocresol to be as successful as full-strength formocresol.[81,82,180]

There is enough evidence today to conclude that ***if formocresol is to be used at all,*** *the one-fifth concentration should be preferred for pulpotomy procedures because it is as effective as and less damaging than the traditional preparation.*

TABLE 24-1

Formulation of Full-Strength Buckley's Formocresol

Formaldehyde	19%
Tricresol	35%
Glycerin	15%
Water	31%

Formocresol and the Permanent Successor

The fear of damage to the succedaneous tooth has been offered as an argument against formocresol pulpotomy on primary teeth. Results from studies are inconsistent, ranging from the same incidence of enamel defects in treated and untreated contralateral teeth[181,236] to an increase in defects and positional alterations of the underlying permanent tooth.[177] It should be pointed out that studies of this nature are follow-up studies long after treatment, and the researchers had no knowledge of the existing status of the pulp before pulpotomy.

The effect of a formocresol pulpotomy upon the exfoliation time of primary molars is also equivocal; some studies found no consistent effect,[181,290] and others reported early exfoliation.[113,180]

Unlike the tissue response to $Ca(OH)_2$ or MTA, no dentin bridge should be anticipated after formocresol is applied to exposed pulp tissue (see Fig. 24-10, *C* and *D*). However, narrowing of the root canal through the continued deposition of dentin by the preserved radicular pulp may be observed in some cases (Fig. 24-12).

Criteria for Success

Failure of a formocresol pulpotomy is usually detected on radiographs (see Fig. 24-8). The first signs of failure are often internal resorption of the root adjacent to the area where the formocresol was applied. This may be accompanied by external resorption, especially as the failure progresses. Sometimes, however, the internal resorption is self-corrected with deposition of calcified tissue. In the primary molars, radiolucency develops in the bifurcation or trifurcation area. In the anterior teeth, a radiolucent area may develop at the apices or lateral to the roots. With more destruction, the tooth becomes excessively mobile; a sinus tract usually develops. It is rare for pain to occur with the failure of a formocresol pulpotomy. Consequently, unless patients receive follow-up checks after a formocresol pulpotomy, failure may go undetected. When the tooth

loosens and is eventually exfoliated, the parents and child may consider the circumstances normal.

The development of cystic lesions after pulp therapy in primary molars has been reported.[29,243] An amorphous, eosinophilic material shown to contain phenolic groupings similar to those present in medicaments was found in the lesions. Myers et al.[185] have observed furcal lesions in untreated, pulpally involved primary molars containing granulomatous tissue with stratified squamous epithelium, which suggests the potential for cyst formation. In a subsequent study involving failed, pulpotomized primary molars, most specimens were diagnosed as furcation cysts.[186] These findings emphasize the importance of periodic follow-up to endodontic treatment on primary teeth. Figure 24-13 shows a favorable long-term outcome after a formocresol pulpotomy in a mandibular primary molar.

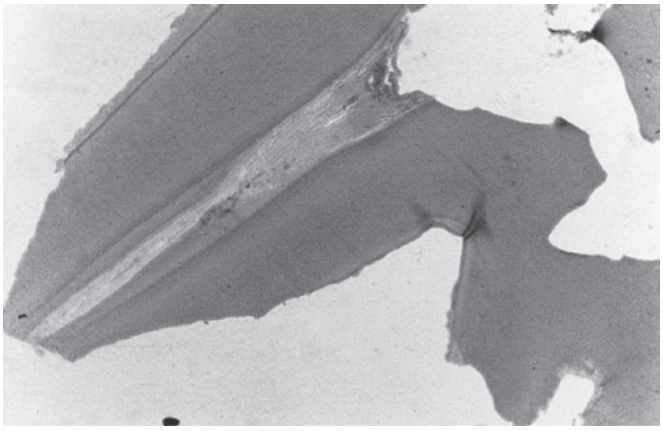

FIG. 24-12 Narrowing of the apical region of the root canal after formocresol pulpotomy. Histologic section of a tooth showing deposition of new dentin along the walls of the apical region of the root canal.

ALTERNATIVES TO FORMOCRESOL PULPOTOMY

A 2006 comprehensive review by Srinivasan et al.[262] is suggested for readers requiring a more detailed look at the numerous alternative techniques available.

Glutaraldehyde Pulpotomy

Glutaraldehyde has been suggested as an alternative to formocresol as a tissue fixative for vital pulpotomy. Histologic studies showed that it produced rapid surface fixation of pulp tissue but with limited depth of penetration, so a larger amount of radicular pulp tissue remained vital.[275,300] Fixed pulp tissue may be replaced with dense collagenous tissue over time.[141] Studies have also demonstrated less systemic distribution than was thought to occur with formocresol.[51,152,187,210,301]

Glutaraldehyde has been shown to be rapidly metabolized with little toxic effect.[112,130,135,187,227] It has also been demonstrated that although glutaraldehyde has antigenic action similar to that of formocresol, it is of a lower potential.[224,226]

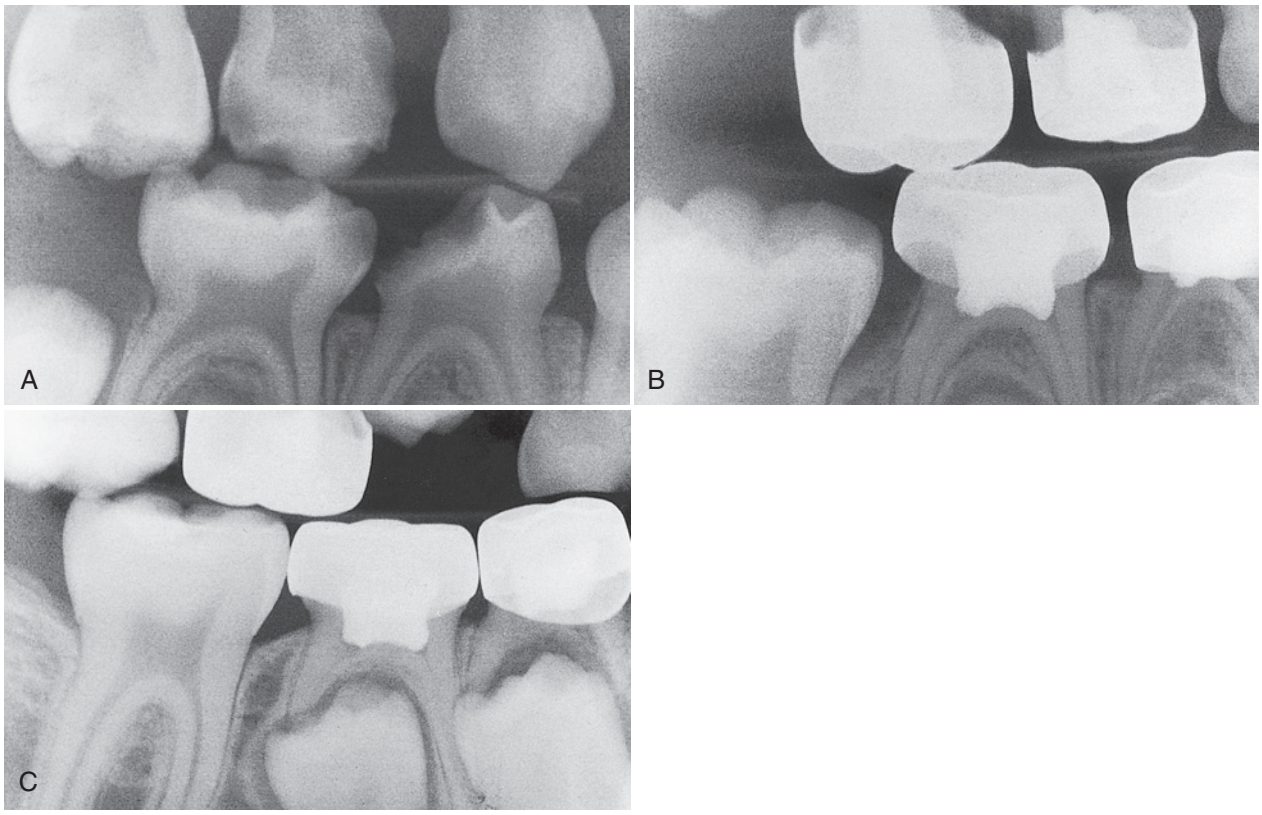

FIG. 24-13 Five-year follow-up of a primary tooth treated by formocresol pulpotomy. A, Pretreatment radiograph showing deep caries on the lower right first and second primary molars. **B,** One-year posttreatment radiograph. **C,** Five-year follow-up radiograph. Note the eruption of the lower right first permanent molar and first bicuspid.

However, there are perceived problems with the use of glutaraldehyde as a pulpotomy agent:

- Glutaraldehyde solutions are reported to be unstable.[222]
- Neither the optimum concentration nor optimal duration of application of glutaraldehyde solution have been established conclusively.[92,156,225]
- Clinical studies reported increasing failure rates with increasing time after pulpotomy,[83] with lower levels of clinical success than with formocresol.[5]

Despite its perceived enhanced safety compared with formocresol, glutaraldehyde has not gained favor over formocresol for pulpotomy in the primary dentition.[13,72]

Ferric Sulfate Pulpotomy

Technique

1. After completion of coronal pulp amputation and achievement of hemostasis with moist cotton pellets, a 15.5% solution of ferric sulfate is applied to the radicular pulp stumps for 10 to 15 seconds.
2. The ferric sulfate may be applied using a cotton pellet or by allowing small droplets of the solution to drip from a burnisher tip onto the surface of the pulp tissue. One manufacturer also supplies a special dento-infuser tip for this purpose (Astringident, Ultradent Products, Salt Lake City, Utah) (see Fig. 24-10, *E*, and Fig. 24-11, *E*).
3. Upon removal of the cotton pellet, the wounds appear brown, and no bleeding should be evident. If a small amount of residual bleeding occurs, one further application of ferric sulfate should be considered.
4. A cement base of ZOE is placed over the pulp stumps and allowed to set. The tooth may then be restored permanently as described for the formocresol pulpotomy (see Fig. 24-10, *F*).

Ferric sulfate is well esablished as a hemostatic agent for crown and bridge impressions.[75] Its hemostatic action occurs by the reaction of blood with ferric and sulfate ions within the acidic pH of the solution. The agglutinated proteins form plugs that occlude the capillary orifices and prevent blood clot formation.[153] Initially the use of ferric sulfate was recommended as a hemostatic agent during $Ca(OH)_2$ pulpotomy on the grounds that it may prevent problems arising from clot formation after coronal pulp amputation and minimize the chances for inflammation and internal resorption[246]; some consider these to be important factors contributing to the failure of pulpotomies using $Ca(OH)_2$. The first documented use of ferric sulfate in this way was in primate teeth.[145]

The use of ferric sulfate itself as a hemostatic pulpotomy agent was investigated by researchers using animal models (rat[40] and baboon[79]). Published clinical studies using ferric sulfate as a pulpotomy medicament have included both prospective comparative[71,79,123,208] and retrospective[28,261] designs.

In all the comparative studies, formocresol was included as the gold standard. The materials and methods used in all these studies are similar, which allows more meaningful comparison of their findings. Pulpotomies were performed in primary molars of healthy children who were selected on the basis of symptomless exposure of vital pulp by caries, absence of clinical or radiographic evidence of pulp degeneration, and possibility of proper restoration.

In all of the aforementioned ferric sulfate studies, the teeth were treated using the pulpotomy technique already outlined, and the teeth were restored with preformed metal (stainless steel) crowns. In a study by Papagiannoulis,[208] half of the teeth were restored with composite resin and the remaining half with preformed metal (stainless steel) crowns.

Prospective Studies

In a study by Fei et al.,[71] 27 teeth were included in the formocresol group and 29 in the ferric sulfate group. Although the sample size was small, the recall rate was excellent, and in the 3- and 6-month intervals, no statistically significant difference was found between the two groups. However, at the end of this 1-year study, a statistically significant difference was seen in the success rate in favor of ferric sulfate (97% for ferric sulfate, 78% for formocresol). The most frequent evidence of failure in this short-term study in both groups was furcation radiolucency. In a study by Fuks,[79] the sample consisted of 37 teeth in the formocresol group and 55 in the ferric sulfate group. The follow-up period ranged from 6 to 34 months, with a mean of 20.5 months. In this study, the sample size was bigger and the evaluation period much longer than in the previous one. The total success rate between the two groups over the total period of 34 months did not present any statistically significant difference; it was 92.7% for ferric sulfate and 83.8% for formocresol. Calcific metamorphosis and internal resorption were the most common radiographic findings. In this study, internal resorption that was stable and unchanged throughout the study was not recorded as failure.

In a later study by Ibricevic and Al-Jame,[123] 34 teeth were used in each group, and identical results were obtained 24 months after treatment for both ferric sulfate and formocresol: a 97% total success rate was recorded. In each group, only one tooth showed internal resorption and was therefore considered a failure.

The results of a larger comparative study[208] were published in 2002. The sample comprised 133 pulpotomy-treated molars of 90 children from 3 to 10 years old, with a mean age of 6.2 years. Sixty teeth (45%) were treated with formocresol and 73 (55%) with ferric sulfate. The number of teeth restored with either a stainless steel crown or a composite resin material was almost equal within each group. After 36 months, the clinical success rate was 97.3% for formocresol and 90.3% for ferric sulfate, but the difference in success rate between the two groups was reported to be statistically insignificant. The radiographic success rate was 78.3% for formocresol and 74% for ferric sulfate, again with no statistically significant difference between them. This success rate seems to be the lowest recorded compared with the previously mentioned comparative studies. The study's author believed that this finding could be due to three reasons: (1) the larger sample size, (2) the very good recall rate, which increased the possibility to observe and locate more failures; and (3) the very strict radiographic assessment. Internal resorption was the most common radiographic finding for both treatments, with no statistically significant difference between ferric sulfate and formocresol. However, most cases classified initially as failures because of internal resorption remained stable throughout the 36-month observation period, and in two cases the respective areas were self-filled with reparative hard tissue. Based on this observation, the data were reevaluated, and only cases of internal resorption that were either extensive in size or progressing with time were considered as failures. After the reevaluation, the overall success rate became 78.7% for ferric sulfate and 85% for formocresol, with no statistically significant difference between

them. The survival rate of the treated teeth after 6 months was 98% for both treatments and then 97% for ferric sulfate and 94% for formocresol after 1 year. These figures decreased with time for both groups, and the survival rate dropped to 81% for ferric sulfate and 78% for formocresol from 25 to 36 months.

Retrospective Studies

In a retrospective study published in 2000 by Smith et al.,[261] ferric sulfate pulpotomies performed over a period of 5 years by a private pediatric dentist were evaluated. The clinical success rate was very high: 99% up to 36 months and no further failures after 36 months. The radiographic success varied from 80% for the 4- to 12-month period, 74% for the 13- to 24-month period, 81% for the 25- to 36-month period, and 74% for the over 36-month period. The estimated tooth survival time was very high, starting with 99% up to 10 months and dropping to 80% after 43 months. The two most common radiographic findings in this study were calcific metamorphosis and internal resorption. The records in this study showed that 13 teeth that presented with internal resorption did not develop osseous lesions for the period of 43 months.

A second retrospective study, published 2 years later in 2002, reported results comparing different pulpotomy treatments; 83 formocresol pulpotomies, 45 ferric sulfate pulpotomies, and 74 pulpotomies treated with a combination of ferric sulfate and formocresol were selected from the records of children treated in a public clinic at different times and by different clinicians.[28] The initial results showed that the total success rate was similar in the three groups, but after a 36-month follow-up, the success rate was better for formocresol than for ferric sulfate, and the combination of the two agents was the worst. The last finding is not surprising because the teeth were treated initially with ferric sulfate for hemostasis and then with formocresol, and they had a poor prognosis (i.e., hyperemic and/or symptomatic). As pointed out by the authors, this study had several shortcomings, the most serious being that the pulpotomies were performed by many clinicians of different levels of experience and expertise; the criteria for selection of cases were not strictly defined; and the radiographs were not always satisfactory, compromising accurate evaluation of the periradicular and furcal areas of the treated teeth.

The studies described demonstrate a higher success rate for both ferric sulfate and formocresol in those with shorter observation times and smaller sample sizes. As the sample size increases and the observation period becomes longer, the success rate drops; this holds true for both ferric sulfate and formocresol pulpotomies. The most important finding is that no statistically significant differences in the success rates between ferric sulfate and formocresol existed, with the exception of one short-term study[71] in which a statistically significant difference was observed in favor of ferric sulfate at the 1-year period. In all the reviewed studies (prospective and retrospective), both ferric sulfate and formocresol treatments gave very good results with high tooth survival rates. Moreover, the type of restoration (stainless steel crown or composite resin) was not reported to influence the success rate for either pulpotomy agent.[208]

Clinical Outcome

In one study, the cases of internal resorption that remained unchanged throughout the 36-month observation period were not considered as failures in the final examination of the results.[208] Others proposed that internal resorption should not be considered as pulpotomy failure and based this proposal on the fact that none of the teeth with internal resorption in their sample developed an osseous lesion.[261] This proposal may be accepted only in cases of minimal or unchanged internal resorption; not for severe or progressive resorption. Internal resorption may indicate pulp inflammation that is expected at the amputation site after pulpotomy. However, if the inflammation can be restricted and confined to a very small part of the pulp while the remainder is healthy, the resorption process will cease or even self-heal by the apposition of hard tissue and therefore will not be a failure. In all the other cases, internal resorption indicates irreversible or extensive inflammation and should be considered a failure.

It can be seen from these studies and the results of a meta-analysis[157] that for human carious primary molars with reversible coronal pulpitis, ferric sulfate and formocresol pulpotomies give similarly good clinical and radiographic results, with high tooth survival rates and no statistically significant differences in success rates. These findings agreed with an earlier Cochrane Review of pulp treatment for extensive decay in primary teeth.[192]

Based on these studies, ferric sulfate can be used instead of formocresol for treatment periods up to 36 months.

Mineral Trioxide Aggregate Pulpotomy

Technique

1. After completion of coronal pulp amputation and achievement of hemostasis with moist cotton pellets, MTA powder is mixed with sterile water until the powder is adherent. Excess moisture is removed from the powder by placing a dry paper point into the mixture to act as a moisture wick.
2. The MTA may be applied to the pulp tissue using an excavator or retrograde amalgam carrier, ensuring enough material to completely cover the exposed pulp tissue to a depth of 3 to 4 mm (see Fig. 24-10, *G*).
3. The MTA mixture is gently packed over the pulp tissue using the blunt end of a large paper point and a broad-ended amalgam compactor. This layer of MTA is a permanent wound dressing.
4. A cement base of ZOE or glass ionomer cement is placed gently over the MTA and allowed to set. The MTA will take several hours to reach its optimum physical strength, so care must be taken to ensure an intact layer of MTA is in contact with the pulp tissue.
5. The tooth may then be restored permanently as described for the formocresol pulpotomy.

MTA used as a pulp capping agent in monkey teeth showed the pulp tissue responses to MTA to be superior to those produced using $Ca(OH)_2$.[215] Similar results were found when human intact third molars were used to compare the effect of pulp capping with MTA and $Ca(OH)_2$.[1] MTA was found to maintain pulp integrity after pulp capping and pulpotomy in animal studies[55,239] and to have a dentinogenic effect on the pulp expressed by the induction of dentin bridge formation (see Fig. 24-10, *H*) where it touches the pulp tissue.[239,241,288]

Clinical Studies

One of the first preliminary studies published comparing MTA with formocresol in humans[60] involved 45 pulpotomy-treated primary molars in 26 children with a mean age of 6 years, 5

months. Clinical and radiographic follow-up ranged between 6 and 30 months, involving 18 children with 32 teeth. Internal resorption was seen in one molar treated with formocresol (17 months after treatment). None of the teeth from the MTA group presented any clinical or radiographic pathosis. Pulp canal obliteration was observed in 9 of 32 (28%) molars evaluated.

Preliminary results of a 3-year prospective follow-up study comparing MTA with formocresol were reported at a scientific congress.[232] Pulpotomies were undertaken on 60 primary molars in 22 children aged 2 to 8 years, each needing at least two pulpotomies. Each child received at least one MTA and one formocresol pulpotomy, followed by application of a preformed metal (stainless steel) crown. Six months after treatment, seven teeth of the formocresol group and two of the MTA group presented abnormal radiographic findings, but these differences were not statistically significant.

More promising results were observed in a more extensive study by Holan et al.[117] that included part of the material presented by Eidelman et al.[60] and a longer follow-up time. Holan and colleagues assessed the long-term success rate of pulpotomy in primary molars with carious pulp exposure using MTA or formocresol as pulp dressing agents. Sixty-four primary molars of 35 children were treated by a conventional pulpotomy technique. After removal of the coronal pulp and hemostasis, the pulp stumps were covered with MTA in the experimental group. In the control group, formocresol was placed with a cotton pellet over the pulp stumps for 5 minutes and then removed; the pulp stumps were then covered with ZOE paste. Eight teeth from each group were restored with an amalgam restoration, and all others were covered with a preformed metal (stainless steel) crown. Thirty-three children with 62 teeth (29 treated with formocresol and 33 with MTA) were available for long-term clinical and radiographic evaluation. Follow-up ranged from 4 to 74 months, with a mean follow-up time of 38 months and with no difference between the groups. Twenty-nine teeth were followed until natural exfoliation (mean, 33 months). Failures were detected after a mean period of 16 months (range, 4 to 30 months). The success rate of pulpotomy was 97% for MTA (one failure) and 83% for formocresol (five failures). Eight teeth showed internal resorption. In four (two from each group), progress of the resorption process stopped, and the pulp tissue was replaced by a radiopaque calcified tissue. Pulp canal obliteration was observed in 55% (34 of 62) of the evaluated molars. This finding, which was not considered a failure, was detected in 58% (19 of 33) of the MTA and in 52% (15 of 29) of the formocresol group. The authors concluded that MTA showed a higher long-term clinical and radiographic success rate than formocresol as a dressing material following pulpotomy in primary molars and recommended it as a suitable replacement for formocresol.

The clinical, radiographic, and histologic effects of gray MTA, white MTA, and formocresol as pulp dressings in pulpotomy-treated primary teeth have been reported.[102] This involved 24 children with a mean age of 6 years (range, 4 to 8 years), each with at least three primary molars requiring pulpotomy, for the clinical and radiographic part of the study. An additional 15 carious primary teeth planned for serial extractions were selected for the histologic part of the study. All the teeth were evaluated periodically for 12 months except those selected for histologic evaluation; these were extracted 6 months after treatment. Sixty pulpotomy-treated teeth in 20 children were reviewed; of these, one tooth (gray MTA) exfoliated normally, and six teeth (four white MTA and two formocresol) failed because of abscesses. All the remaining 53 teeth showed success clinically and on radiographs. Pulp canal obliteration was found in 11 teeth treated with gray MTA and one treated with white MTA. The histologic evaluation demonstrated that both types of MTA induced thick dentin bridges, whereas in the formocresol group these were thin and poorly calcified. The pulpal architecture observed with the gray MTA was closer to normal than the white MTA, which presented a dense fibrotic pattern with isolated pulp calcification. These authors concluded that gray MTA was better than both white MTA and formocresol as a pulp dressing for pulpotomy-treated primary teeth. More recently, MTA pulpotomies were compared with $Ca(OH)_2$[154,273] and ferric sulfate,[56,65] with reported results showing that MTA is a suitable pulpotomy agent.

The growing evidence from clinical studies and a systematic review[257] allows MTA to be recommended as an effective pulpotomy agent in primary teeth.

MTA is commercially available in the United States as ProRoot MTA (DENTSPLY Tulsa Dental Specialties, Tulsa, OK). Angelus MTA (Angelus Indústria de Produtos Odontológicos, Londrina, Brazil) is also available in Latin America and Europe. ProRoot MTA is considered expensive, particularly because it is retailed in cartons containing a number of sealed 1-g packets. The composition of MTA is similar to that of cement used in the building industry, and such material should be kept dry during storage because contact with moist air leads to "air setting," which affects the powder's physical properties. The same could be applied to the "clinical grade" cement, and the manufacturer of ProRoot MTA recommends the 1-g sachet as single use, which would be expensive. The unused MTA remaining in a sachet may actually be stored for up to a further 4 weeks in a water-tight, air-tight container such as an Eppendorf tube (Eppendorf UK, Cambridge, Great Britain, UK), reducing its cost.[265] It has been suggested that the MTA powder can be stored indefinitely if the corner of the packet is folded firmly and the packet is stored in a sealable plastic bag. Angelus MTA is presented in a sealable glass vial, with the recommendation that 1 g may provide up to seven treatments. Care must be taken not to contaminate the MTA powder when it is dispensed.

Electrosurgical Pulpotomy

Technique

The steps in the electrosurgical pulpotomy technique are basically the same as those for the formocresol technique through the removal of the coronal pulp tissue.

1. Large sterile cotton pellets are placed in contact with the pulp, and pressure is applied to obtain hemostasis.
2. The Hyfrecator Plus 7-797 (Birtcher Medical Systems, El Paso, Texas) is set at 40% power (high at 12 W), and the 705-A dental electrode is used to deliver the electric arc. The cotton pellets are quickly removed, and the electrode is placed 1 to 2 mm above the pulpal stump.
3. The electric arc is allowed to bridge the gap to the pulpal stump for 1 second, followed by a cool-down period of 5 seconds. Heat and electrical transfer are minimized by

keeping the electrode as far from the pulpal stump and tooth structure as possible while still allowing electric arcing.
4. If necessary, this procedure may be repeated up to a maximum of three times. The procedure is then repeated for the next pulpal stump.
5. When the procedure is properly performed, the pulpal stumps appear dry and completely blackened.
6. The chamber is filled with ZOE placed directly against the pulpal stumps. Research by Fishman[76] has shown no difference between ZOE and $Ca(OH)_2$ as the dressing. The tooth should then be restored with a preformed metal (stainless steel) crown.

Although electrocoagulation on the pulps of teeth was reported in 1957,[150] it was a decade later that Mack[162] became the first U.S. clinician to routinely perform electrosurgical pulpotomies. Oringer[206] also strongly advocated this technique in his 1975 text on electrosurgery.

Electrosurgery is a nonpharmacologic, hemostatic pulpotomy technique used directly on the radicular pulp stumps after coronal pulp amputation. Depending on the currents used and thus the heat generated, incision, coagulation, or electrofulguration can occur. Electrosurgical pulpotomy carbonizes and denatures pulp tissue, producing a layer of coagulative necrosis. This acts as a barrier between the lining material and healthy pulp tissue below.[262]

Several clinical studies[237,254] have produced results comparable to those found with the use of formocresol. Conflicting results have been reported from histologic studies, ranging from results comparable to formocresol pulpotomy[253] to pathologic root resorption with periapical and furcal involvement.[255] A retrospective human study in 1993[163] showed a success rate of 99% for primary molars undergoing electrosurgical pulpotomies. Compared with a formocresol pulpotomy study of similar design, the success rate of the electrosurgery technique was shown to be significantly higher. A more recent study[197] compared the use of two different base lining materials during electrosurgical pulpotomy. Success rates at 12 months were high for both groups, 98% and 96% for ZOE and zinc polycarboxylate cements, respectively.

Laser Pulpotomy

Several reports have appeared in the literature on the use of the carbon dioxide laser for performing vital pulpotomies on primary teeth.[62,155] Elliott et al.[62] compared the use of the laser with formocresol in caries-free, primary cuspid teeth that were scheduled for extraction in children between 6 and 10 years of age. Thirty teeth were included in the study. No significant differences were found between the formocresol and laser-treated groups. Areas of isolated internal resorption were identified in one of the formocresol-treated teeth and two of the laser-treated teeth. These authors concluded that on the basis of symptomatic, clinical, and histologic findings, the carbon dioxide laser appeared to compare favorably with formocresol treatment. It was suggested that additional studies be conducted to establish the ideal applied laser energy to maximize optimum residual pulpal response and to explore the effects of laser pulpotomy upon pulps previously exposed by carious lesions.

Liu et al.[155] reported the use of the laser on primary teeth with vital carious pulpal exposures. Thirty-three teeth, 21 primary molars and 12 primary canines, were treated and observed for 12 to 27 months. All were clinically successful, and only one showed evidence of internal resorption at the 6-month follow-up visit. The authors observed complete calcification on radiographs after 9 months in approximately half of the treated teeth.

On the basis of these initial studies, the use of the carbon dioxide laser could be considered a viable alternative to formocresol. More randomized controlled human clinical trials are recommended.

In summary, the search for alternatives to the formocresol pulpotomy in cariously exposed vital primary teeth has yet to reveal an agent, instrument, or technique that has unequivocal long-term clinical success rates better than those of formocresol, although MTA is beginning to show evidence of excellent success rates in comparative studies.[56,65,270] Until such an agent, instrument, or technique is found, ferric sulfate, MTA, or formocresol (one-fifth dilution) can be used with equal confidence in primary tooth pulpotomies.[80,235,262]

NONVITAL PULP THERAPY ON PRIMARY TEETH

Pulpectomy in Primary Teeth

The anatomic and normal physiologic features of primary teeth can present challenges to the clinician wishing to undertake pulpectomy procedures. However, with knowledge of these features and how they may affect the clinical technique, pulpectomy and root canal obturation of primary teeth with irreversibly inflamed or necrotic pulp can be a clinically successful option.

In brief, these features are:

- Root anatomy: Apical positions, lateral and accessory canals
- Root physiology: The effect of exfoliation upon root anatomy and choice of root canal filling material
- The permanent tooth bud: Its proximity to the primary root apex

Effect of Resorption on Canal Anatomy and Apical Foramina

In the newly completed roots of the primary teeth, the apical foramina are located near the anatomic apices of the roots. After the deposition of additional dentin and cementum, there are multiple apical ramifications of the pulp as it exits the root, just as in the mature permanent tooth.

The physiologic resorption of the roots of the primary incisors and canines starts on the lingual surfaces in the apical third because of the position of the permanent tooth bud. In primary molars, resorption usually begins on the inner surfaces of the roots near the interradicular septum (Box 24-5).

As resorption progresses, the apical foramen may not correspond to the anatomic apex of the root but be coronal to it. Subsequently, radiographic establishment of the root canal length may be challenging (Fig. 24-14). Resorption may extend through the roots and into the root canals, creating additional communications with the periapical tissues other than through the apical foramina or lateral and accessory canals. This has been shown to occur at all levels of the root.[231] Because of these factors, use of an apex locator is not reliable to establish canal length.

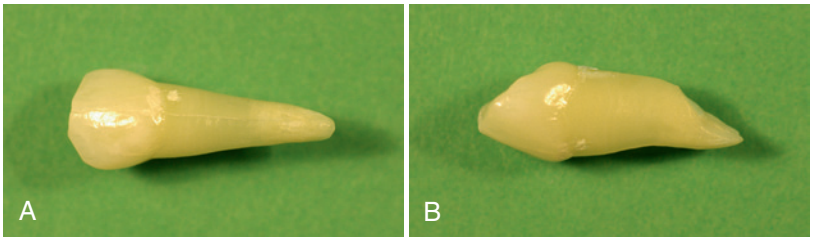

FIG. 24-14 Primary maxillary canine. A, Facial view: the apical foramen appears to be at the apex of the root. B, Physiologic root resorption has occurred at the palatal aspect of the root; the apical foramen is positioned more coronally and will not be coincident with the perceived radiographic apex of the tooth.

BOX 24-5

Physiologic Root Resorption in Primary Teeth

Physiologic root resorption begins:

- Soon after root-length completion
- In primary incisors and canines, on the lingual aspect of the apical third of the roots
- In primary molars, on the inner surfaces of the roots next to the inter-radicular septum

Permanent Tooth Bud

The effects of primary endodontic therapy on the developing permanent tooth bud should be of paramount concern to the clinician. Manipulation through the root apex of a primary tooth and overextension of root canal instruments and filling materials are to be avoided because the permanent tooth bud lies in close proximity. If radiographic signs of resorption are visible, the canal length is usually established by measurement of the diagnostic radiograph, ensuring that the working length of endodontic instruments is 2 or 3 mm short of the radiographic apex. Use of the long cone radiographic paralleling technique for maximal accuracy is recommended. Hemorrhage after pulp removal may indicate overextension into the periapical tissues.

Anesthesia is usually necessary for pulp extirpation and cleaning of the canals but may be unnecessary when primary teeth are filled at a subsequent appointment. The response of the patient can sometimes be used as a guide to the proximity of the apex and as a check of the length of the canal. However, this can only be done in cooperative patients because the discomfort caused by placing the rubber dam clamp or by the approach of the instrument to the apex might create disruptive behavior in some children.

The material used to obturate root canals in primary teeth must be resorbable, so that as the tooth resorbs, it offers no resistance or deflection to the eruption of the permanent tooth. *Permanent obturating materials, such as gutta-percha, are contraindicated in root canal therapy for primary teeth that have permanent successors.*

Pulpectomy

Pulpectomy and root canal filling procedures on primary teeth have been the subject of much controversy. Fear of damage to developing permanent tooth buds and a belief that the tortuous root canals of primary teeth could not be adequately negotiated, cleaned, shaped, and filled have led to the needless sacrifice of many pulpally involved primary teeth. Much has been written regarding potential damage to the developing permanent tooth bud from root canal fillings. The extraction of pulpally involved primary teeth and placement of space maintainers is an alternative to pulpectomy. However, there is no better space maintainer than the primary tooth. If a space maintainer is placed but adequate monitoring and preventive care are not provided, further problems often occur.

For example, with a "band and loop" design of space maintainer, loose bands and poor oral hygiene increase the risk of dental caries and gingival inflammation. Prolonged retention of the appliance may cause deflection of the erupting permanent tooth, and premature loss of the band can result in loss of space, particularly if the patient delays returning for treatment.

It has been reported that minor hypoplasia is increased in permanent successor teeth after root canal treatment of the primary precursors.[119] Coll[39] reported no such increased effect and concluded that defects result from the infection existing before the pulpectomy and not the procedure itself. It is noteworthy that these studies are retrospective, involving erupted permanent teeth; findings should be viewed with caution.

Economics has been advanced as an argument against endodontic treatment of primary teeth, but it is not a reasonable argument when compared with the cost of space maintainers, including the required follow-up treatment. In fact, endodontic treatment is probably the less expensive alternative when the entire treatment sequence is considered.

The success of endodontic treatment on primary teeth is judged by the same criteria used for permanent teeth. The treated primary tooth must remain firmly attached and function without pain or infection. Radiographic signs of furcal and periapical infection should be resolved with a normal periodontal attachment. The primary tooth should resorb normally and in no way interfere with the formation or eruption of the permanent tooth.

Success rates ranging from 75% to 100% have been reported.[10,118,147,269,305] The usual modalities of evaluating root canal treatment in primary teeth have been clinical and radiographic. More comprehensive evaluation in this area is greatly needed.

Early reports of endodontic treatment on primary teeth usually involved devitalization with arsenic in vital teeth and the use of creosote, formocresol, or paraformaldehyde pastes in nonvital teeth. The canals were filled with a variety of materials, usually consisting of zinc oxide and numerous additives.[59,93,131,263]

Rabinowitch[221] published the first well-documented scientific report on endodontic procedures on primary teeth in 1953. A 13-year study of 1363 cases of partially or totally

nonvital primary molars was reported. Only seven cases were failures; most patients were followed for 1 or 2 years clinically and with radiographs. Patients underwent multiple visits to achieve root canal fillings of ZOE and silver nitrate. Periapically involved teeth required an average of 7.7 visits to complete treatment, and teeth with no periapical involvement required an average of 5.5 visits. Rabinowitch listed internal resorption and gross pathologic external resorption as contraindications to primary root canal fillings.

Another well-documented study reported a success rate of 95% in vital and infected teeth using a filling material of thymol, cresol, iodoform, and zinc oxide.[10] (See Bennett[19] for a review of the techniques of partial and total pulpectomy.)

In a well-controlled clinical study of primary root canal treatments using Oxpara paste as the filling material,[147] five preexisting factors were reported to render the prognosis less favorable:

1. Perforation of the furcation
2. Excessive external resorption of roots
3. Internal resorption
4. Extensive bone loss
5. Periodontal involvement of the furcation

When teeth with these factors were eliminated, a clinical success rate of 96% was achieved. When all symptoms of residual infection were resolved before filling of the canals, the success rate improved.

Contraindications to Primary Root Canal Fillings

Many primary teeth with pulpal involvement that has spread beyond the coronal pulp are candidates for root canal fillings, whether they are vital or nonvital. Box 24-6 lists the categories of teeth that are not good candidates for pulpectomy.

Internal resorption usually begins just inside the root canals near the furcation area. Because of the thinness of the roots of the primary teeth, once internal resorption has become visible on radiographs, a perforation of the root by the resorption invariably has occurred (see Fig. 24-8). The short furcal surface area of the primary teeth leads to rapid communication between the inflammatory process and the oral cavity through the periodontal attachment. The end result is loss of the periodontal attachment of the tooth and, ultimately, further resorption and loss of the tooth. Mechanical or carious perforations of the floor of the pulp chamber fail for the same reasons. It has been shown that root length is the most reliable criterion of root integrity; at least 4 mm of root length is necessary for the primary tooth to be treatable.[231]

BOX 24-6

Contraindications to Pulpectomy in the Primary Dentition

- Unrestorable tooth
- Internal resorption in the roots visible on radiographs
- Teeth with mechanical or carious perforations of the floor of the pulp chamber
- Excessive pathologic root resorption involving more than a third of the root
- Excessive pathologic loss of bone support, with loss of the normal periodontal attachment
- Presence of a dentigerous or follicular cyst
- Periapical or interradicular lesion involving the crypt of the developing permanent successor

Access Openings for Pulpectomy

Anterior Primary Teeth

Access openings for endodontic treatment on primary or permanent anterior teeth have traditionally been through the lingual surface. This continues to be the surface of choice, except for discolored maxillary primary incisors, for which a facial approach is recommended, followed by an acid-etched composite restoration to improve aesthetics (Fig. 24-15).[164]

Posterior Primary Teeth

Access openings into the root canals of posterior primary teeth are essentially the same as for the permanent teeth. Important differences between the primary and permanent teeth are the length of the crowns, the bulbous shape of the crowns, and the very thin dentinal walls of the pulpal floors and roots. The depth necessary to penetrate into the pulpal chamber is much less than that in the permanent teeth. Likewise, the distance from the occlusal surface to the pulpal floor of the pulp chamber is much less than in permanent teeth. In primary molars, care must be taken not to overinstrument the relatively thin pulpal floor, owing to the high risk of perforation (Fig. 24-16).

When the roof of the pulp chamber is breached and the pulp chamber identified, the entire roof should be removed. Because the crowns of the primary teeth are more bulbous, less extension toward the exterior of the tooth is necessary to uncover the openings of the root canals than in the permanent teeth.

Technique

As in permanent endodontic therapy, the main objective of the chemical and mechanical preparation of the primary tooth is debridement of the canals. Although an apical taper is desirable, it is not necessary to have an exact shape to the canals because obturation is achieved using a resorbable paste. Figure 24-17 provides a schematic overview of the procedure.

Canal Preparation

1. A preliminary working length is determined by measurement of a radiograph taken with a paralleling technique.
2. Local anesthesia is advisable.
3. Placement of a rubber dam is mandatory.
4. The working length is determined from a radiograph with an endodontic file in the canal. The use of apex locators is often unreliable in primary teeth because root resorption may create lateral openings into the periodontal tissues at any level,[231] causing unreliable readings.
5. To prevent overextension through the apical foramen, it is advisable to shorten the working length to 2 to 3 mm short of the radiographic length, especially in teeth showing signs of apical root resorption (see Fig. 24-17, *B*).
6. After establishment of the working length, the canal is cleaned and gently shaped (as described in Chapter 6). *Because of the thin root walls, sonic and ultrasonic cleaning devices should not be used to prepare the canals. Also, the use of Gates-Glidden (GG) or Peeso drills (Pulpdent, Watertown, Massachusetts) is contraindicated because of the danger of perforation or stripping of the roots.*
7. Flexible nickel-titanium (NiTi) instruments are recommended. Hand or rotary techniques are ideal for primary

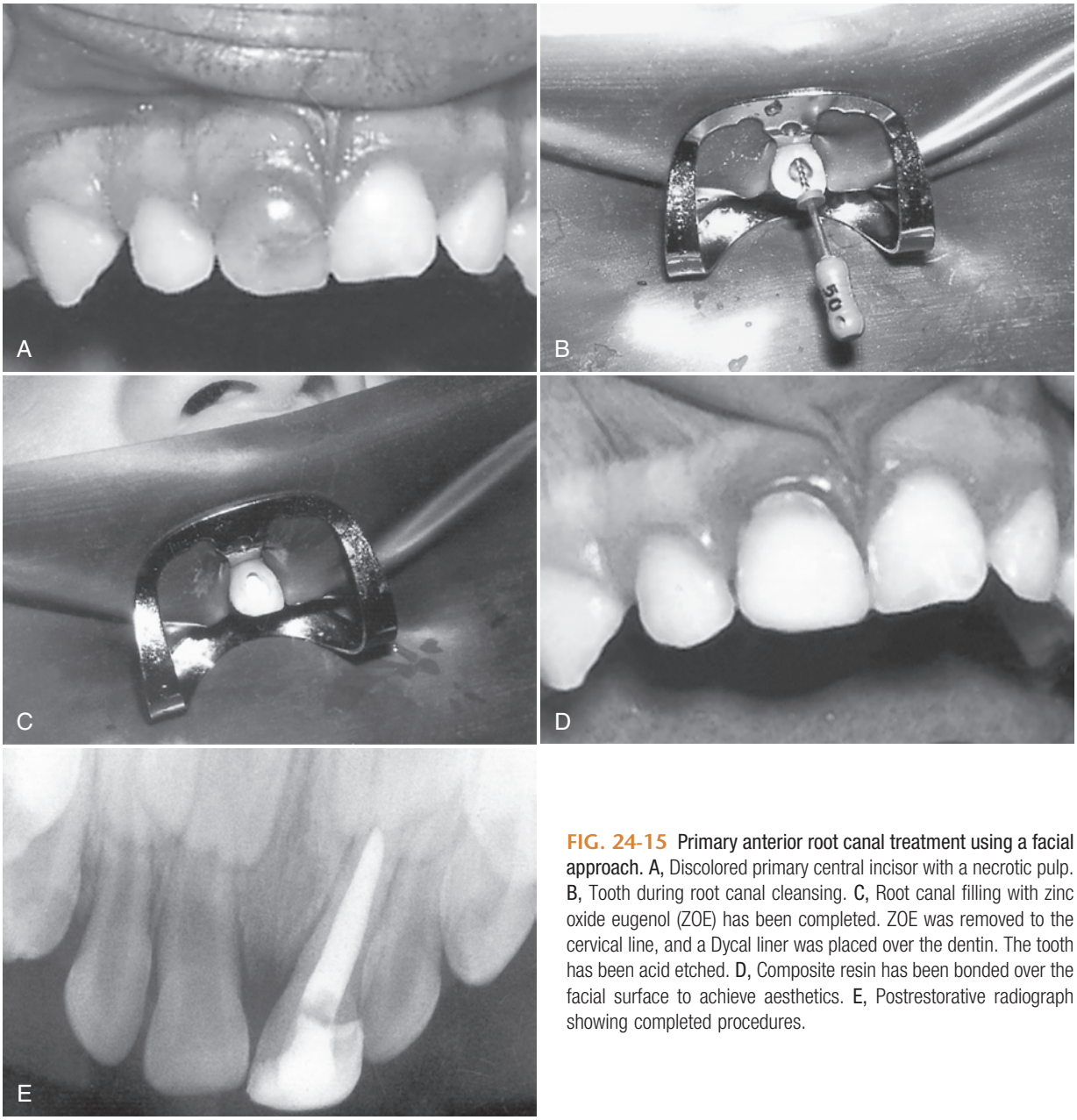

FIG. 24-15 Primary anterior root canal treatment using a facial approach. **A,** Discolored primary central incisor with a necrotic pulp. **B,** Tooth during root canal cleansing. **C,** Root canal filling with zinc oxide eugenol (ZOE) has been completed. ZOE was removed to the cervical line, and a Dycal liner was placed over the dentin. The tooth has been acid etched. **D,** Composite resin has been bonded over the facial surface to achieve aesthetics. **E,** Postrestorative radiograph showing completed procedures.

teeth. If stainless steel files are used, the instruments must be gently precurved to help negotiate the canals.

8. Care must be taken not to perforate the thin roots during cleaning and shaping procedures. The canals are enlarged several sizes past the first file that fits snugly in the canal, with a minimum size of 30 to 35.

Importance of Irrigation During Treatment

Because many of the pulpal ramifications cannot be reached mechanically, copious irrigation during cleaning and shaping must be maintained (see Chapter 6). Debridement of the primary root canal is more often accomplished by chemical means than by mechanical means.[124] This statement should not be misinterpreted as deemphasizing the importance of thorough debridement and disinfection of the canal. Initially, RC-Prep (Premier Dental Products, Norristown, Pennsylvania) may be used as the canals are negotiated. It helps to emulsify pulp tissue and is an effective lubricant. Once a working length has been established, sodium hypochlorite (NaOCl) solution is used to dissolve organic debris; this can play an important part in removal of tissue from inaccessible areas of the root canal system (see Fig. 24-17, *C*).

The Nonvital Discolored Primary Incisor

If a primary incisor is intrinsically discolored after loss of pulp vitality, aesthetics may demand an improvement of the tooth's color. Bleaching is not advised in the primary dentition. The anatomy of the maxillary primary incisors is such that access may be made successfully from the facial surface. The only variation to the opening is more extension to the incisal edge than with the normal lingual access to give as straight an approach as possible into the root canal.

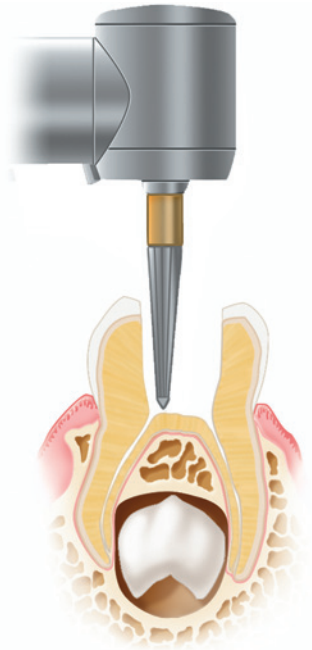

FIG. 24-16 Safe removal of the roof of a pulp chamber in a primary molar. A non–end cutting bur ensures that the relatively thin floor of the pulp chamber is not perforated inadvertently by rotary cutting instruments.

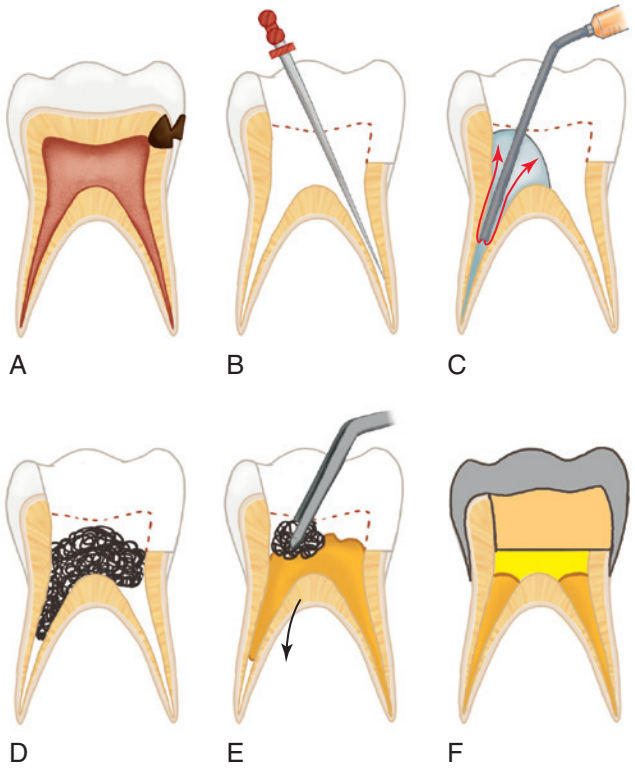

FIG. 24-17 Stages of pulpectomy and root canal filling in a mandibular second primary molar. A, Extensive approximal caries. Note the irreversible inflammation present in the coronal and radicular pulp. **B,** After caries removal and unroofing of the pulp chamber, the coronal pulp is amputated. Irreversibly inflamed tissue bleeds profusely. A premeasured hand file is placed approximately 2 mm from the radiographic apex; the canals are gently cleaned with minimal shaping. **C,** Irrigation with sodium hypochlorite or chlorhexidine digluconate solution should be performed during the cleaning phase. **D,** If the root canals are not to be obturated at the same visit, they may be dressed with nonsetting calcium hydroxide; or, the canals can be left empty and the tooth restored with a small cotton pledget and an interim intracoronal restoration. **E,** At the subsequent visit, root canals can be obturated with a resorbable root-filling material, such as zinc oxide eugenol (ZOE). This can be applied using various methods; here, the ZOE is tamped down the canal by the piston action of a cotton pledget held in tweezers. **F,** After root canal filling, the tooth is restored internally before definitive coverage with a preformed metal (stainless steel) crown.

The root canal is filled with ZOE (see the following section); then the ZOE is carefully removed to near the cervical line. A liner of calcium hydroxide cement, such as Dycal or Life, is placed over the ZOE to serve as a barrier between the composite resin and the root canal filling. The liner is extended over the darkly stained lingual dentin to serve as an opaquer. The access opening and entire facial surface are acid etched and restored with composite resin (see Fig. 24-15, *C* and *D*).

The Decision to Obturate

After canal debridement, the canals are again copiously flushed with NaOCl and then dried with sterile premeasured paper points. If the canals are dry and no exudate is present, obturation is performed in the same session. If the obturation cannot be done at the first appointment, a slurry paste of nonsetting $Ca(OH)_2$ can be injected into the canals and the tooth restored with a well-sealing temporary restoration.

At a subsequent appointment, the rubber dam is placed and the canals reentered. As long as the patient is free of all signs and symptoms of inflammation, the canals are irrigated with NaOCl to remove the intracanal dressing and dried before obturation. If signs or symptoms of inflammation are present, the canals are recleaned and remedicated, and the canal obturation delayed until a later time.

Root-Filling Materials for the Primary Root Canals

The ideal root canal filling material for primary teeth should meet the following criteria:

- Resorb at a similar rate as the primary root
- Be harmless to the periapical tissues and the permanent tooth germ
- Resorb readily if pressed beyond the apex
- Be antiseptic
- Fill the root canals easily
- Adhere to the walls of the root canal
- Not shrink
- Be easily removed if necessary
- Be radiopaque
- Not discolor the tooth[161]

No material currently available meets all these criteria. The filling materials most commonly used for primary pulp fillings are ZOE paste, iodoform paste, and $Ca(OH)_2$.

Zinc Oxide Eugenol Paste

Most reports in the U.S. literature have advocated the use of ZOE as the filler, whereas other parts of the world have used pastes containing iodoform.[118,143] The antibacterial activity of ZOE has been shown to be greater than that of an iodoform-containing paste (e.g., KRI paste, Pharmachemic AG, Zurich,

Switzerland), whereas ZOE's cytotoxicity in direct and indirect contact with cells is equal to and less than (respectively) that of KRI paste. *The filling material of choice in the United States is ZOE without a catalyst*. The lack of a catalyst is necessary to allow adequate working time for filling the canals.

Iodoform Paste

Several authors have reported the use of KRI paste, which is a mixture of iodoform, camphor, parachlorophenol, and menthol.[176,230] It resorbs rapidly and has no undesirable effects on successor teeth when used as a pulp canal medicament in abscessed primary teeth. Furthermore, if KRI paste extrudes into the periapical tissue, it is rapidly replaced with normal tissue.[118] Sometimes the material is also resorbed inside the root canal. A paste developed by Maisto has been used clinically for many years, and good results have been reported with its use.[171,274] This paste has the same components as the KRI paste, with the addition of zinc oxide, thymol, and lanolin.

Calcium Hydroxide

Several clinical and histopathologic investigations of $Ca(OH)_2$ and iodoform mixture (Vitapex, Neo Dental Chemical Products, Tokyo) have been published.[16,78,199] These authors found that this material is easy to apply, absorbs at a slightly faster rate than that of the roots, has no toxic effects on the permanent successor, and is radiopaque. For these reasons, Machida[161] considers the calcium hydroxide–iodoform mixture a nearly ideal primary tooth root canal filling material. Another preparation with similar composition, Endoflas, is available in the United States (Sanlor Laboratories, Cali, Colombia, South America). The results of root canal treatments using Endoflas were similar to those observed with KRI paste[85] and ZOE.[269]

Obturation of the primary root canal is usually performed without a local anesthetic. This is preferable, if possible, so the patient's response can be used to indicate proximity to the apical foramen. However, it sometimes is necessary to anesthetize the gingiva with a drop of anesthetic solution to place the rubber dam clamp without pain.

Obturation of the Root Canal

The chosen obturation technique depends upon the material used and the accessibility of the canal to relevant instruments.

If ZOE is used, it is mixed to a thick consistency and carried into the pulp chamber with a plastic instrument or on a Lentulo spiral. The material may be packed into the canals with pluggers or a Lentulo spiral. A cotton pellet held in cotton pliers and acting as a piston within the pulp chamber is quite effective in forcing the ZOE into the canals (see Fig. 24-17, *E*). The endodontic pressure syringe is also effective for placing the ZOE in root canals.[20,98] However, in a study of apical seal and quality of filling evaluated on radiographs, no statistically significant differences were reported between the Lentulo spiral, pressure syringe, or plugger.[50]

When the root canal is filled with a resorbable paste, such as KRI, Maisto, or Endoflas, a Lentulo spiral mounted in a low-speed handpiece can be used to introduce the material into the canal. When the canal is completely filled, the material is compressed with a cotton pellet. Excessive material is rapidly resorbed.

Vitapex is packed in a convenient sterile syringe, and the paste is injected into the canal with a disposable plastic needle. This technique is particularly easy to use for primary incisors but less practical for the narrow canals of primary molars.[203]

Regardless of the method used to fill the canals, care should be taken to prevent extrusion of the material into the periapical tissues. It is reported that a significantly greater failure rate occurs with overfilling of ZOE than with filling just to the apex or slightly underfilling.[39,118] The adequacy of the obturation is checked by radiographs (see Fig. 24-15, *E*; also Fig. 24-18, *A-C* and Fig. 24-19, A-E).

In the event a small amount of the ZOE is inadvertently forced through the apical foramen, it is left alone (because the material is absorbable). It has been reported that defects on successor teeth have no relationship to length of the ZOE filling.[39]

When the canals have been satisfactorily obturated, a fast-setting temporary cement is placed in the pulp chamber to seal over the root canal filling. The tooth may then be restored permanently. In primary molars, it is advisable to place a preformed (stainless steel) crown as the permanent restoration to ensure good coronal seal and prevent possible fracture of the tooth (see Fig. 24-17, *F*).

If a primary tooth requires pulpectomy and the permanent successor tooth is absent, the primary root canals are filled with gutta-percha and sealer in an attempt to retain the primary tooth long term (Fig. 24-20).

Follow-Up After Pulpectomy in the Primary Dentition

As previously stated, the rate of success after primary pulpectomy is high. However, these teeth should be periodically recalled to check for success of the treatment and intercept any problem associated with a failure. It has been reported that pulpectomized primary teeth may show delayed exfoliation.[39,265] One study[39] described a 20% incidence of crossbites or palatal eruption of permanent incisors after pulpectomy on primary incisors. In the posterior teeth, extraction was required in 22% of cases because of ectopic eruption of the premolars or difficulty in exfoliation of the treated primary molar.[39] After normal physiologic resorption of the roots reaches the pulp chamber, the large amount of ZOE present may impair the resorptive process and lead to prolonged retention of the crown. Treatment usually consists of simply removing the crown and allowing the permanent tooth to complete its eruption.

Retention of ZOE in the tissues is a common sequela to primary pulpectomy. One long-term study reported that after loss of the tooth, 50% of cases had retained ZOE. Teeth filled short of the apices had significantly less retained filling material, and in time, most showed complete absorption or reducing amounts. Retention of filling material was not related to success and caused no pathosis.[238] Therefore, no attempt is made to remove retained material from the tissues (see Fig. 24-18, *A*, and Fig. 24-19, *C*).

While resorbing normally without interference from the eruption of the permanent tooth, the primary tooth should remain asymptomatic, firm in the alveolus, and free of pathosis. Traditionally, root canal treatments were considered successful when no pathologic resorption associated with bone rarefaction was present.[91,118] If evidence of pathosis is detected, extraction and conventional space maintenance are recommended.

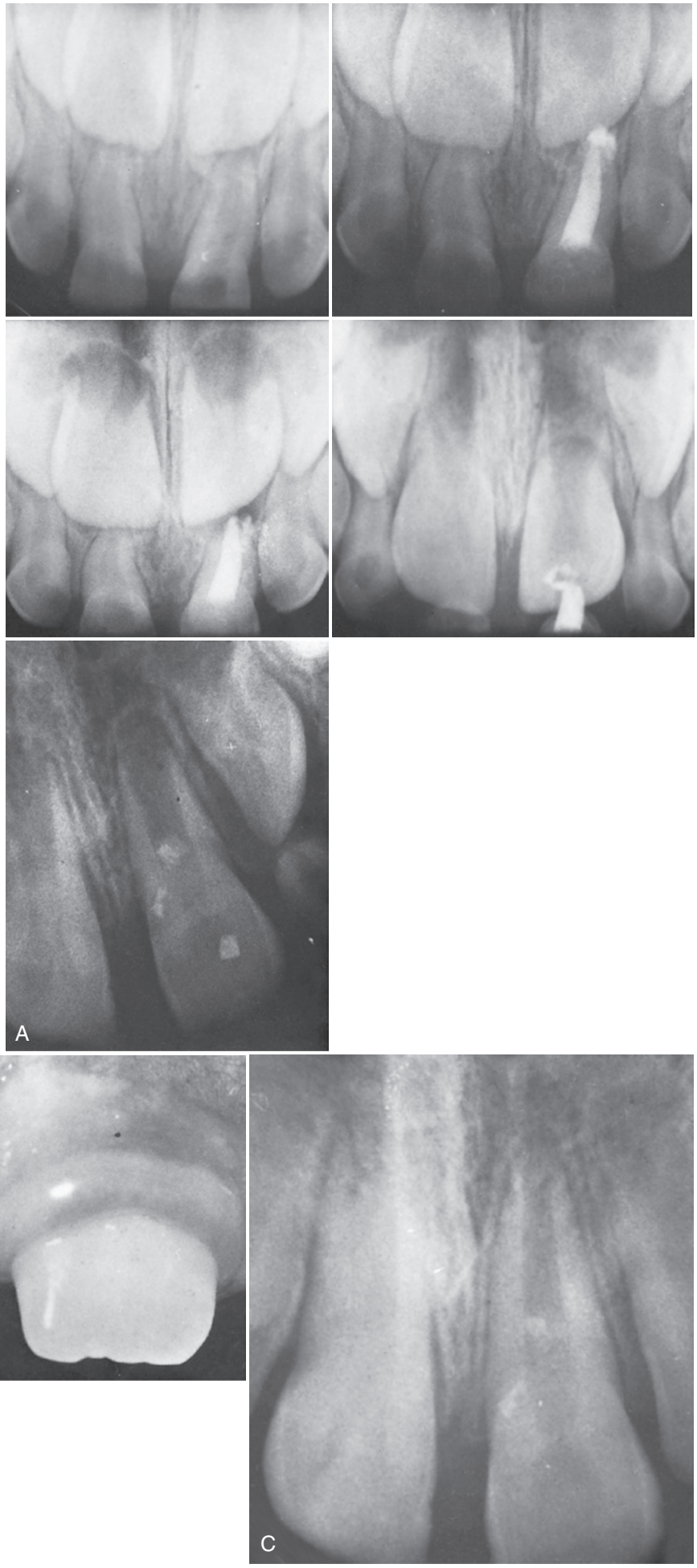

FIG. 24-18 **Pulpectomy and root canal filling with zinc oxide eugenol (ZOE) paste in a primary maxillary central incisor.** **A,** The root canal has been slightly overfilled, with extrusion of ZOE paste apically. **B,** The same patient showing newly erupted permanent incisors. Note that no enamel defects are present on the crown, despite overfill of the root canal of the predecessor. **C,** Radiograph almost 5 years after pulpectomy and root canal filling of predecessor. Note the normal apical development and almost total absorption of ZOE remnants.

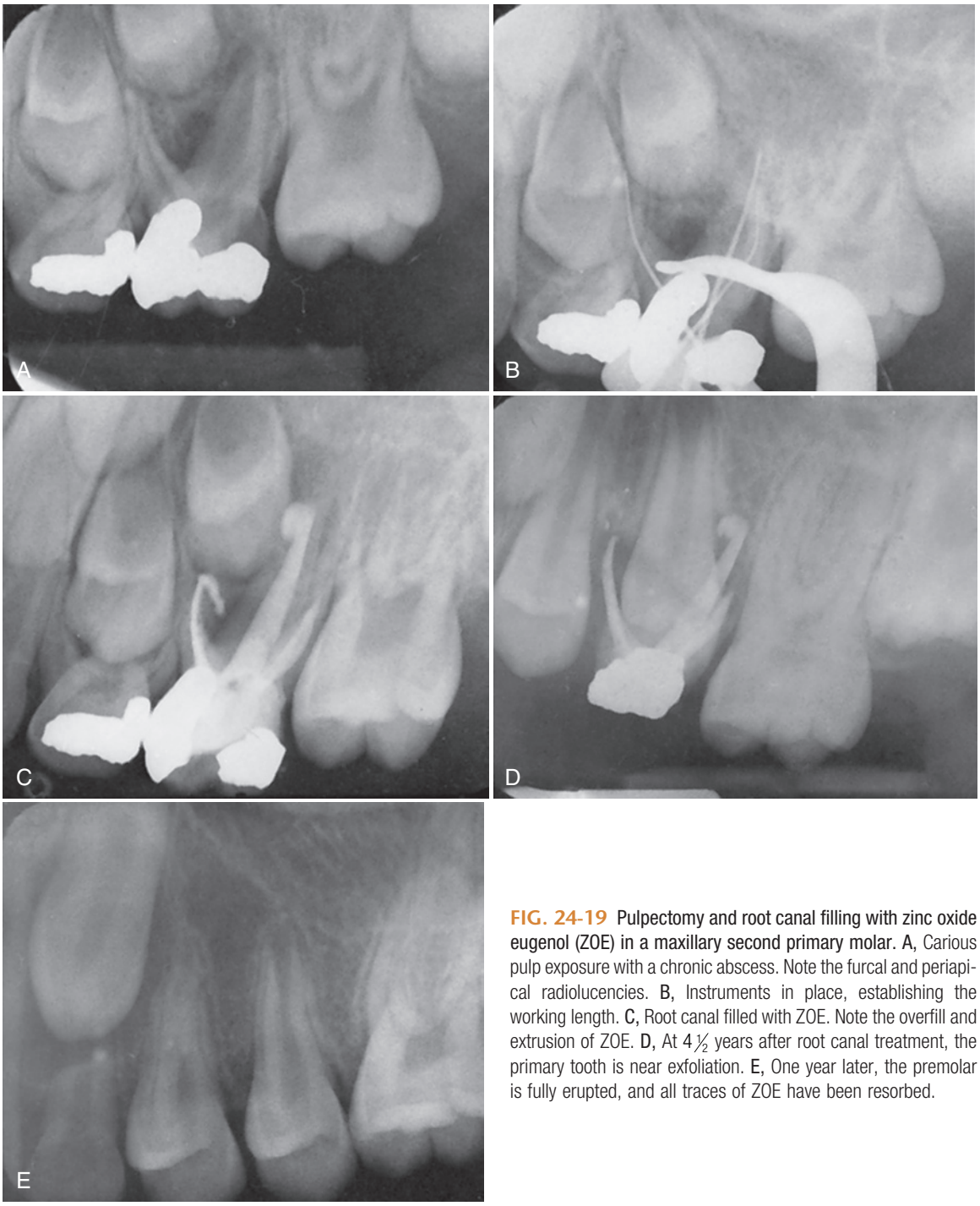

FIG. 24-19 **Pulpectomy and root canal filling with zinc oxide eugenol (ZOE) in a maxillary second primary molar. A,** Carious pulp exposure with a chronic abscess. Note the furcal and periapical radiolucencies. **B,** Instruments in place, establishing the working length. **C,** Root canal filled with ZOE. Note the overfill and extrusion of ZOE. **D,** At 4½ years after root canal treatment, the primary tooth is near exfoliation. **E,** One year later, the premolar is fully erupted, and all traces of ZOE have been resorbed.

Payne et al.[212] claim that most clinicians are prepared to accept pulp-treated primary teeth that have a limited degree of radiolucency or pathologic root resorption in the absence of clinical signs and symptoms. This is contingent on the assurance that the parent will contact the clinician if an acute problem develops, and the patient will return for review in 6 months. These criteria seem to be more suitable for pediatric dental practices and have been adopted clinically by Fuks et al.,[85] who consider such teeth to be "successfully treated."

PULPAL THERAPY FOR THE YOUNG PERMANENT DENTITION

It could be argued that mature permanent teeth can survive for a lifetime without the support of a vital pulp. For the immature permanent tooth, the future is less secure. Premature loss of a functioning pulp results in a fragile tooth with a compromised crown-to-root ratio, thin dentin walls, and a wide and often apically diverging root that presents significant endodontic and

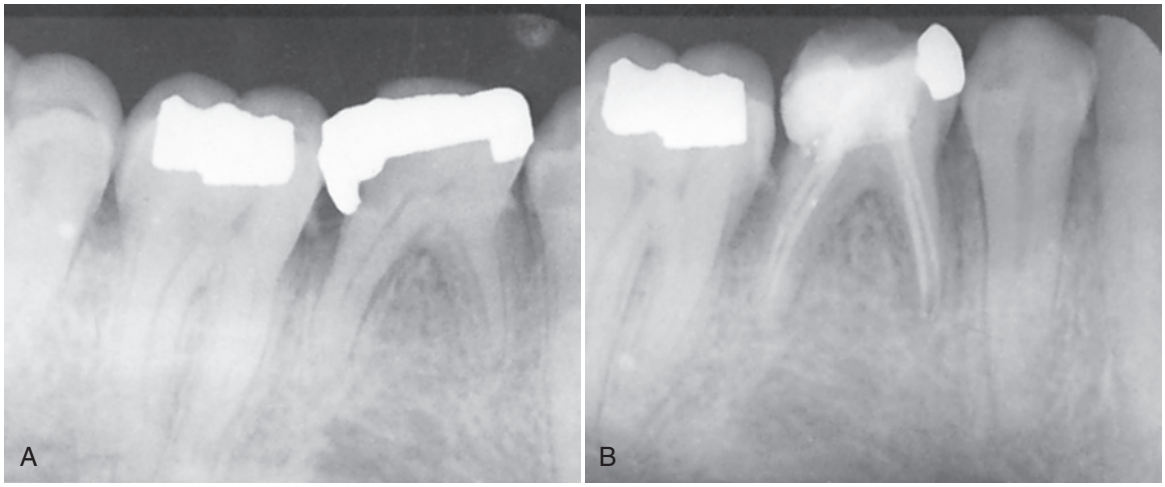

FIG. 24-20 Pulpectomy and root canal filling with gutta-percha in a retained mandibular primary second molar with no succedaneous permanent tooth. **A,** Carious exposure of the pulp. **B,** Because the permanent premolar is absent, the root canals were filled with gutta-percha and sealer rather than just zinc oxide eugenol.

FIG. 24-21 Premature loss of vital pulp functions in an immature maxillary permanent incisor. Development is arrested, leaving a fragile tooth with a compromised crown-to-root ratio, thin dentin walls, and a wide-open root end; these factors present endodontic and restorative challenges.

restorative challenges (Fig. 24-21). A central responsibility of all clinicians is therefore to safeguard pulp survival at least until dental development is complete.

The procedures described in this section have much in common with those for primary teeth and focus on preserving all or part of the pulp in a functional condition. In addition to aspects of pulp protection, indirect pulp therapy, direct pulp capping, and pulpotomy, attention is given to the root canal treatment and restoration of nonvital, immature permanent teeth. The emerging potential of pulp regeneration and bioroot engineering is also considered.

Indirect Pulp Therapy: Avoiding Pulp Exposure

The pulps of young permanent teeth are at risk of breakdown after traumatic injuries (considered fully in Chapter 20), dental caries, and restorative dentistry. Good evidence indicates that residual dentin thickness is a key determinant of pulp survival after cavity preparation,[183] and avoiding pulp exposure has been considered advantageous. The management of deep caries by partial or serial excavation has gained considerable support in recent years, reducing the risks of pulp exposure and harnessing the natural defenses of the pulp in laying down protective tertiary (reactionary) dentin.* In the case of serial excavation, the need for a secondary excavation has recently been brought into question,[166] although the need for a tightly sealing coronal restoration to ensure that any residual caries is inactivated has been acknowledged.[228] Researchers continue to investigate the role of antimicrobial treatments, including ozone fumigation,[216] photoactivated disinfection (PAD), and antimicrobial resins in sterilizing deep layers of affected dentin[286] and creating the conditions for arrest and remineralization. Considerable interest has also focused on the active up-regulation of reactionary dentinogenesis by applying bioactive agents, such as the transforming growth factor beta (TGF-β) family of molecules, to the depths of cavity preparations.[285] Therapeutic approaches present a hopeful future for pulp protection after deep caries, but significant challenges remain, not least in defining optimal agents and predictably delivering them to the pulp through the fluid-filled dentinal tubules of vital teeth.[219] More basic and clinical research is needed before such agents can be widely adopted in practice.

However, a substantial body of evidence supports the view that complete excavation is not necessary for the successful management of deep caries[200] and that indirect pulp therapy may be the most effective means of securing pulp health in asymptomatic teeth.

*References 22, 23, 52, 99, 106, and 165.

FIG. 24-22 Direct pulp capping in a symptomatic young permanent mandibular molar with an open apex. **A,** Extensive carious exposure in an immature molar with a history of spontaneous pain. **B,** After complete caries excavation and hemostasis, the pulp is overlaid with mineral trioxide aggregate and the tooth sealed with composite resin. **C,** Three-year recall shows completed root formation. Note the lack of mineralization in the pulp chamber and canals.

Managing Pulp Exposure

Even when treated with the greatest clinical judgment and restorative skill, pulps are sometimes exposed during deep caries excavation. A 2007 survey showed that 62% of clinicians would remove all caries when presented with a case in which one would expect pulp exposure, whereas only 18% would partially excavate caries, and 21% would initiate root canal treatment.[204] In a comparable study among Brazilian public health dentists, 71% favored direct, complete caries excavation over more conservative approaches, although dentists graduating after the year 2000 were more likely to embrace conservative approaches.[299] Direct pulp capping has generally not enjoyed predictable success in cariously exposed teeth. One study showed a 44.5% failure at 5 years and a disappointing 79.7% failure at 10 years.[17] A more recent systematic review points to success of 73% at 3 years for permanent teeth, but longer term outcomes appear variable and uncertain.[3]

Although much of the evidence is relatively short term and involves previously uninflamed pulps, calcium silicate cements, such as MTA, are developing a profile for promoting reparative tertiary dentin bridge formation after direct pulp capping.[148,193]

In a radically different approach to the management of deep caries in immature permanent teeth where pulp diagnosis may be uncertain and the stakes for pulp preservation are high, one of the previous authors of this chapter (Dr. Joe Camp) has advocated complete caries excavation and direct capping of the exposure in the immature teeth of children (Fig. 24-22). Teeth with percussion sensitivity, swelling, or other obvious signs of pulpal necrosis are not considered good candidates, and the pulp tissue exposed during caries excavation must appear vital with no signs of degeneration or suppuration. In this procedure, the tooth is anesthetized and isolated with a rubber dam. All decay is removed with round carbide burs and copious water spray. No further pulpal tissue is removed except that occurring with the caries removal. The often profuse bleeding that occurs is controlled by rinsing the pulp with NaOCl, which is not only antimicrobial but appears to have no adverse effects on pulpal healing, odontoblastic cell formation, or dentinal bridging.[2,42] The solution may be left in contact with the exposed pulp tissue for 10 to 15 minutes; it is refreshed with new solution every 3 to 4 minutes. Care must be taken in aspirating the excess NaOCl to prevent further hemorrhage; the aspirator tip is placed lateral to, and never directly above, the crown. Once hemorrhage has been controlled, the tooth structure is cleansed with cotton pellets moistened with NaOCl, again avoiding further pulp hemorrhage.

The exposed pulp is then covered with a 0.5- to 1-mm thickness of MTA, which is gently teased against the exposed

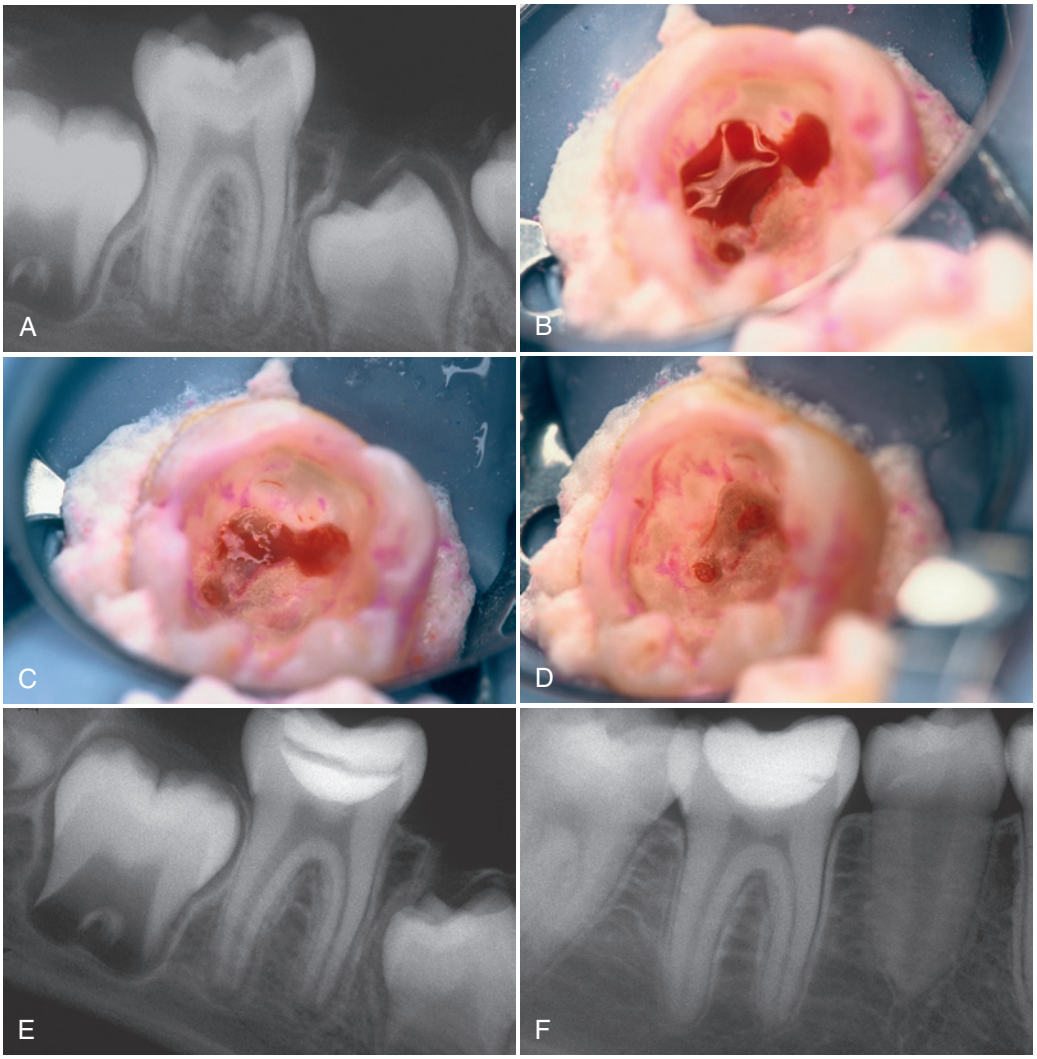

FIG. 24-23 **Radiographic and clinical sequence of mineral trioxide aggregate (MTA) direct pulp capping of a mandibular right molar in a 9-year-old female. A,** Pretreatment radiograph showing initial deep caries and immature apices. **B** to **D,** Five-minute application of 5.25% sodium hypochlorite hemostasis on two 1.5- to 2-mm exposures. **E,** Radiograph of molar with MTA, water-moistened cotton pellet, and unbonded Clearfil Photocore (Kuraray Medical, Okayama, Japan) provisional restoration after initial visit. **F,** Radiograph taken at the 5.5-year recall appointment shows permanent restoration and evidence of complete root formation. The tooth showed a normal response to cold testing. (From Bogen G, Kim JS, Bakland LK: Direct pulp capping with mineral trioxide aggregate: an observational study, *J Am Dent Assoc* 139:305, 2008.)

pulp tissue with a damp cotton pellet. Wisps of cotton are wet with sterile water and placed over the MTA so it is completely covered. The tooth is then provisionally sealed with a temporary cement, such as Cavit, to allow hardening of the MTA.

The patient is seen again within 12 to 48 hours, and the tooth is anesthetized and isolated with a rubber dam. After removal of the Cavit and cotton, the MTA is examined to ensure that it has set hard, and the tooth is restored with a bonded composite restoration. If the MTA has failed to harden, Camp advocated washing out the uncured material and repeating the procedure after removing pulp tissue to the canal orifice level. This approach has received further support from two studies. In a study by Farsi et al.,[69] 30 asymptomatic permanent molars were reviewed clinically and radiographically after caries excavation and direct pulp capping with MTA; 93% were successful at 24 months, with continued root development. In the second report, Bogen et al.[25] described a series of 53 teeth diagnosed at the outset with deep caries and reversible pulpitis but with no periapical involvement. Caries was completely eliminated with the help of a caries-detector dye and magnification (Fig. 24-23, *A* and *B*), often resulting in multiple, large pulp exposures. Hemostasis was secured by bathing the pulp with 5.25% to 6% NaOCl for 1 to 10 minutes (Fig. 24-23, *B* to *D*). One pulp that continued to bleed after this time was considered unsuitable for direct capping. After application of 1.5 to 3 mm of gray or white MTA (Fig. 24-23, *E*) and permanent bonded restorations at 5 to 10 days, the teeth were reviewed for 1 to 9 years (mean, 3.94 years). The recall rate was 92.5%, and 97.96% of the teeth were found to have favorable outcomes

on the basis of radiographic appearances, subjective symptoms, and cold testing (Fig. 24-23, *F*). Of the 15 teeth that were immature at the time of treatment, 100% went on to complete root formation.

At the time of writing, a conservative general consensus probably holds with partial or serial caries excavation, avoiding pulp exposure and attempting to create an environment that will up-regulate reactionary tertiary dentin responses.[22,228,280] However, evidence is growing that in correctly chosen cases with no indication of irreversible pulpitis or periapical change and when radical caries excavation is followed by confirmation of hemostasis, direct capping with calcium silicate cements, such as MTA, can achieve remarkable levels of success in teeth with incomplete apices.

Because the loss of vital pulp functions is so devastating in teeth with immature apices, it seems advisable to attempt conservative pulp-preserving procedures in deeply carious permanent teeth. If these procedures fail, apexification or regenerative techniques can always be considered.

PULPOTOMY

The pulpotomy procedure involves removing only part of the pulp, eliminating tissue that has inflammatory or degenerative changes and leaving intact the underlying healthy pulp tissue.[12] The healthy tissue is then covered with a wound dressing agent in an effort to promote healing at the amputation site and to promote the survival of the underlying pulp tissue. Traditionally, the term *pulpotomy* has implied removal of pulp tissue to the cervical line. However, the depth to which tissue is removed is determined by clinical judgment of how deeply the pulp is affected. Superficial amputation (partial pulpotomy) may allow better visualization of the working area but risks leaving damaged tissue that may go on to break down. In multirooted teeth, the procedure may be simplified by removing tissue to the orifices of the root canals (complete or pulp-chamber pulpotomy).

The Cvek Pulpotomy on Immature Permanent Teeth

Pulpotomy is an established technique for preserving vital pulp functions in immature teeth that have been subject to pulp-exposing trauma. A full account of the Cvek pulpotomy is provided in Chapter 20 (see Fig. 20-11). Briefly, studies have shown that inflammation is confined to the surface 2 to 3 mm of the pulp when it is traumatically exposed and left untreated for up to 168 hours.[47,108,109] In experimental animals, the results were the same whether the crowns were fractured or ground off.[47] Direct invasion of vital pulp tissue by bacteria did not occur, although the pulps were left exposed to saliva.

In a classic report, Cvek[45] described a pulpotomy technique in which only the superficial 2 to 3 mm of hyperplastic inflamed tissue was removed with a water-cooled, high-speed diamond bur[97] to place the wound in a healthy site. Hemostasis was then secured before capping with an appropriate material. If hemostasis could not be secured after several minutes of saline-moistened cotton pellet application, the preparation was checked carefully for residual superficial tags of bleeding tissue that had not been fully removed, or questions were asked about the condition of the underlying pulp. Persistent bleeding from an inflamed pulp usually indicates that the tissue should be resected at a deeper level to preserve a vital apical pulp stump (see the section on apexogenesis).

Hemostasis was obtained with saline, and a pulp cap of calcium hydroxide was overlaid with a sealing coronal restoration. Cvek reported success in an impressive 94% to 96% of cases (Fig. 24-24). More recent reports suggest that histologic tissue responses may be more favorable in response to calcium silicate cements, such as MTA, than to $Ca(OH)_2$.[133,191,193,215] Thus MTA is recommended as the pulp-capping agent of choice in cases that do not extend deeply into the roots, where subsequent retrieval may present a considerable challenge. Preference should be given to white MTA products in an effort to minimize the risk of unsightly tooth staining, although this possibility cannot be ruled out.[18]

The question, then, is whether such techniques can be applied to posterior teeth, especially those with symptomatic cariously affected pulps. The answer historically has been no, except as a short-term, pain-relieving exercise. Studies involving cavity preparations into teeth that left areas of impacted food, debris, and bacteria in contact with pulp tissue resulted in inflammation extending from 1 to 9 mm into the pulp, with abscess and pus formation.[47,108] The extent of inflammation in a cariously exposed pulp is difficult to judge. Pulpotomy may consequently be less predictable in preserving vital pulp functions after a carious pulp exposure compared to a traumatic pulp exposure.

Partial Pulpotomy on Asymptomatic Young Permanent Posterior Teeth

A technique called *partial pulpotomy for the management of vital carious pulp exposure on young permanent molars* has been reported by several researchers.[169,172,175,202] This approach has usually been reserved for teeth with little or no history of pain and in the absence of radiographic signs, percussion sensitivity, swelling, or mobility.

The procedure usually involves removal of 1 to 3 mm of pulp tissue suspected to be inflamed, beneath the exposure site, in order to reach underlying healthy tissue. After hemostasis, the exposure site traditionally has been covered with $Ca(OH)_2$ and the tooth sealed with ZOE and a permanent restoration. Mejàre and Cvek[175] followed 31 initially symptom-free teeth for a mean of 56 months (range, 24 to 140 months) and found a healing rate of 93.5%. Mass et al.[169] reported a 91.4% success rate in 35 cases followed for 12 to 48 months. Exposures exceeding 2 mm in diameter and those in which bleeding could not be controlled within 1 to 2 minutes were excluded from this study.

The use of MTA as the capping agent has met with success in the hands of the authors and others,[201,244] although long-term success has also been reported after the use of more traditional materials in properly selected cases.[170]

Pulpotomy on Symptomatic Young Permanent Teeth

Although some success has been reported,[175] vital pulp therapy in permanent teeth with a history of pain has generally been contraindicated. However, reports do support this pulp-preserving approach, even for symptomatic teeth (see Chapter 26 for more details).[244] In the study by Mejàre and Cvek,[175] of the six teeth with temporary pain, widened periodontal

FIG. 24-24 **Follow-up of a Cvek pulpotomy. A,** Pretreatment radiograph of a right maxillary central incisor after traumatic pulp exposure. **B,** Four months after Cvek pulpotomy and application of a calcium hydroxide dressing, formation of a dentin bridge can be seen. **C,** Three-year recall shows continued root formation. In this case, the dentin bridge has not thickened. There is no evidence of uncontrolled mineralization in the pulp space.

ligament space, and/or condensing osteitis that were managed by $Ca(OH)_2$ pulpotomy, 66.7% healed. A study by Calişkan[30] undertook a similar $Ca(OH)_2$ pulpotomy procedure on 26 permanent vital molars with carious pulp exposures and apical periodontitis. Observation between 16 and 72 months revealed that 24 (92.3%) were free of clinical symptoms, responded to sensibility testing, and showed evidence of hard tissue barrier formation, resolution of periapical involvement, and the absence of intraradicular pathosis radiographically.

Partial pulpotomy was also found to be successful in a randomized, controlled trial[220] that compared $Ca(OH)_2$ and MTA pulpotomies in the permanent molars (n = 64) of children. Mean follow-up was 34.8 months (range, 25.5 to 45.6 months), and success was comparably favorable in both the $Ca(OH)_2$ (91%) and MTA (93%) groups.

A full coronal pulpotomy may be more predictable in the symptomatic case in which the depth of pulp inflammation is difficult to predict. Follow-up of MTA pulpotomies in 15 children with immature molars revealed no clinical or radiographic failures at 12 months, although four pulps had undergone calcific metamorphosis.[61] In a 2006 clinical report, Witherspoon et al.[304] described a series of 23 irreversibly pulpitic anterior and posterior teeth in children and adolescents, treated by full coronal pulpotomy, hemostasis for 1 minute with 6% NaOCl, and wound dressing with MTA. Follow-up of 6 to 53 months (mean, 19.7 months) revealed that 79% had healed, 16% were healing, and only 5% had evidence of persistent disease. More research is needed, but the MTA pulpotomy seems to be gaining an evidence base as a reliable technique for preserving vital pulp functions in both asymptomatic and symptomatic teeth in children.

This body of evidence suggests that young teeth, with their rich vascular supply, are good candidates for conservative pulp-preserving treatments. Again, the stakes are high, and efforts to secure vital pulp functions are to be commended. Outcomes are probably more predictable in asymptomatic cases than symptomatic, and care should be taken preoperatively to distinguish between cases with symptoms of reversible and irreversible pulpitis. However, the correlation of clinical symptoms with histologic state is poor.[57]

Follow-Up After Pulp Capping and Pulpotomy

Patients undergoing pulp capping and pulpotomy procedures should be seen periodically for 2 to 4 years to determine

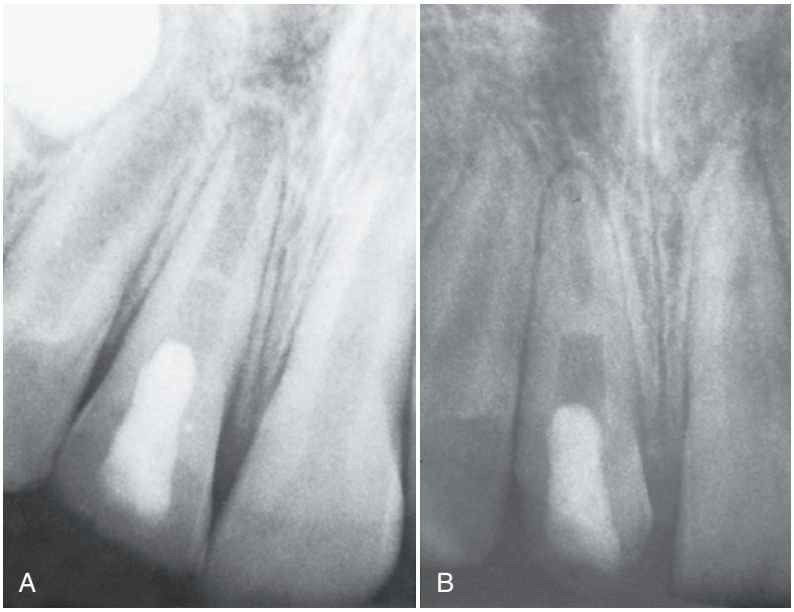

FIG. 24-25 Deep pulpotomy for apexogenesis. A, Immature maxillary central incisor 10 weeks after traumatic pulp exposure and a deep pulpotomy dressed with calcium hydroxide. Note the early formation of a dentin bridge. **B,** At three-year recall—the dentin bridge has thickened, and root formation is complete; the tooth remained asymptomatic and required no further treatment at that time. Note the absence of uncontrolled mineralization in the apical pulp space.

success. Although normal sensibility tests (e.g., electrical and thermal sensitivity tests) are reliable after pulp capping, they are not usually helpful in the pulpotomy-treated tooth. Because histologic success cannot be determined, clinical success is judged by the absence of any clinical or radiographic signs of pathosis and the presence of continued root development in teeth with incompletely formed roots.

Controversy exists as to whether the pulp should be reentered after the completion of root development in the pulpotomy-treated tooth. Langeland et al.[146] believe that pulp capping and pulpotomy procedures invariably lead to progressive calcification of the root canals. They advocated that, after successful root development, pulpectomy and root canal treatment should be performed before canals became obliterated, and difficult to manage endodontically if they did become infected. However, it has been the experience of the authors and others[48,84,109] that with good case selection, a gentle technique in removing infected tissue and dentin chips, and care in avoiding the compaction of pulp dressing into underlying pulp tissue, calcification of the pulp is an infrequent sequela of pulpotomy (see Fig. 24-24, *C*, and Fig. 24-25). The routine use of microscopy has also transformed the management of many cases that might in the past have been considered untreatable. The prophylactic root canal treatment of teeth after successful pulpotomy is therefore difficult to justify.

In a follow-up study of clinically successful pulpotomies,[45] researchers removed the pulps 1 to 5 years later for restorative reasons and found the tissue to be histologically normal.[48] They concluded that the changes observed do not present sufficient histologic evidence to support routine pulpectomy after pulpotomy in accidentally fractured teeth with pulp exposures. Thus, routine reentry to remove the pulp and place a root canal filling after completion of root development is contraindicated unless dictated by restorative considerations, such as the need for a retentive post. Nevertheless, calcific obliteration, internal resorption, and pulp necrosis are potential sequelae of pulp-capping and pulpotomy procedures, and the clinician should screen for these problems during recall appointments. Although unlikely, they are possible, and the patient should be clearly informed.

Formocresol Pulpotomy on Young Permanent Teeth

Because of the historical track record of formocresol pulpotomy in primary teeth, interest developed in this technique for the management of young permanent teeth. Reports have demonstrated the potential for continued apical development after formocresol pulpotomy in young permanent teeth,[73,184,240,294] although the high incidence of internal resorption[82,213] adds to broader concerns about the use of formocresol in pediatric endodontics.

In most circumstances, the formocresol pulpotomy has been performed as an emergency treatment, with reports showing that teeth may remain symptom free after 3 years.[282] However, alternatives to the formocresol pulpotomy are preferred, and renewed interest has been shown in the potential of pulpotomy with materials such as MTA as a short- and long-term treatment strategy for damaged pulps.[259]

APEXOGENESIS

Apexogenesis is a treatment designed to preserve vital pulp tissue in the apical part of a root canal so that formation of the root apex may be completed (Fig. 24-26).[107]

The clinical procedure is essentially a deep pulpotomy undertaken to preserve the formative capacity of the radicular pulp in immature teeth that have deep pulpal inflammation.

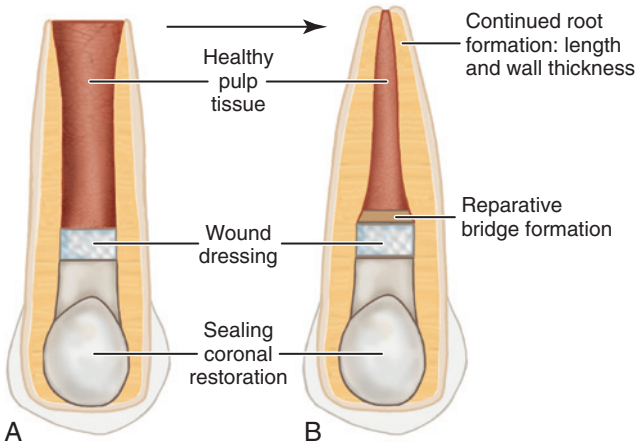

FIG. 24-26 Schematic representation of apexogenesis. *Apexogenesis* is a treatment designed to preserve at least the apical portion of pulp tissue in a healthy condition so that root formation can be completed. **A,** After a deep pulpotomy and hemostasis, the radicular pulp is dressed and a sealing coronal restoration is applied. **B,** Success is evidenced by continued root development (length and wall thickness) and formation of a calcific barrier in response to the wound dressing.

Examples include carious exposures and some trauma cases in which treatment of the exposed pulp is delayed and it becomes necessary to extend farther into the canal to reach healthy tissue.

Deep resection of pulp tissue is usually undertaken in single-rooted anterior teeth with a small endodontic spoon excavator or round, abrasive diamond bur (see Fig. 24-25). In posterior teeth, the use of endodontic files or reamers may be necessary if tissue is to be amputated within the canals. Extension of the amputation site into the root canals is undertaken only when there is little confidence that more superficial pulp capping or pulpotomy will be successful and when there is a desperate desire to preserve pulp functions in teeth with wide-open, blunderbuss apices (Fig. 24-27).

Bleeding is usually controlled with saline-soaked cotton pellets or NaOCl. If hemostasis cannot be secured by conventional means, this may indicate that even the deep pulp is inflamed and treatment will be compromised. Despite the risks, it is legitimate to attempt pulp-preserving treatment after controlling the hemorrhage with hemostatic chemicals such as aluminum chloride or ferric sulfate.

The pulp wound is then covered with a dressing material before the crown is securely restored. It is challenging to determine the status of pulp tissue deep in the root canal, and its capacity for survival is difficult to predict. Radiographic and clinical follow-up is mandatory, and if there is no evidence of

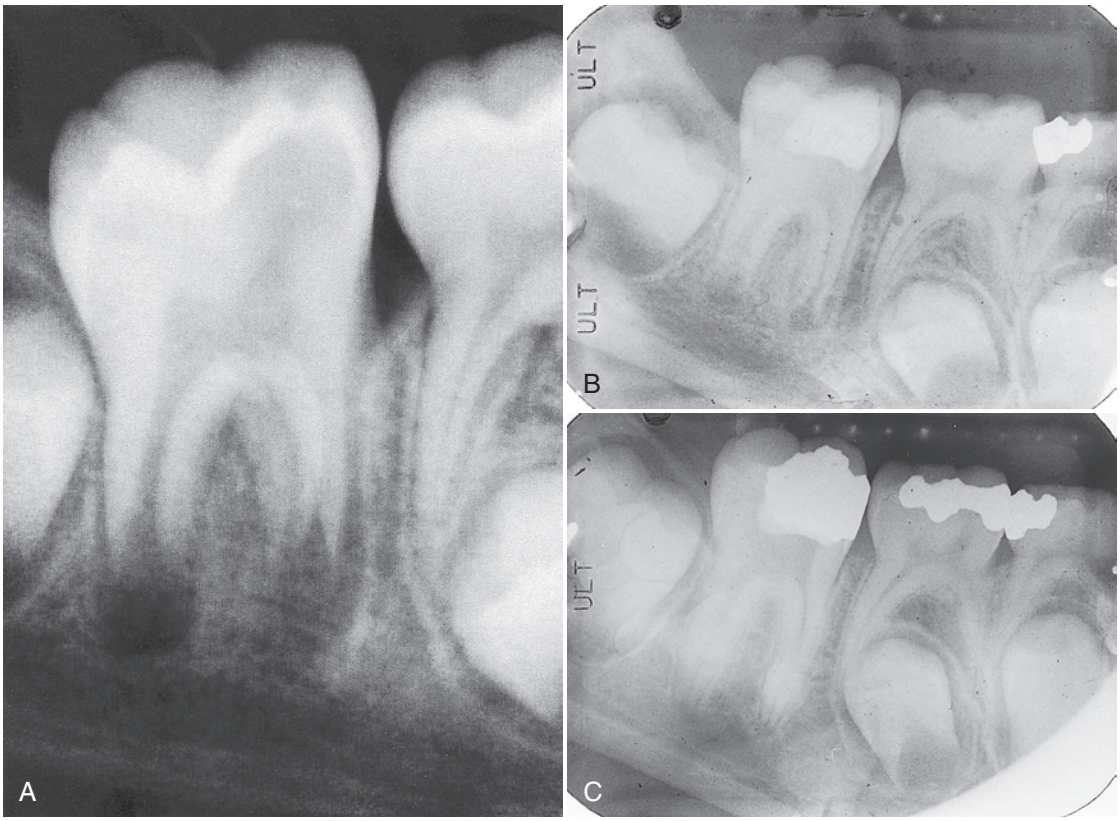

FIG. 24-27 Apexogenesis after deep calcium hydroxide pulpotomy on a mandibular permanent molar. **A,** Pretreatment radiograph showing extensive caries, incomplete root development, and possible periapical pathosis. The tooth was asymptomatic. **B,** One year after deep pulpotomy (which extended several millimeters into the canals), hemostasis, and restoration. Root formation is progressing, and the periapical tissues appear healthy. **C,** Two years later, root formation is complete.

continued root formation and calcific barrier formation in response to the dressing, apexification or a regenerative technique may be considered.

Because of the depth at which this procedure is performed, preference has usually been given to the use of $Ca(OH)_2$ rather than MTA because, in the event of failure, this may facilitate reentry to the root canal to perform apexification or pulp regeneration. Also, if apexogenesis is successful and root-end formation is complete, the tooth could be reentered if desired for conventional root canal treatment. It is unclear whether these concerns hold true in an era of microscopy and contemporary microinstrumentation.

Calcium hydroxide powder has usually been preferred over hard-setting products, carried into the canal with an amalgam carrier or gun system used for MTA application. Small increments of $Ca(OH)_2$ powder are carefully teased against the entire surface of the pulp stump with a rounded-end, plastic instrument, ideally with microscope control. Care must be taken not to pack the $Ca(OH)_2$ into the pulp tissue because this causes greater inflammation and increases the chances of failure. Even if pulpotomy is successful, there is an increased risk that the remaining pulp tissue will mineralize around impacted particles of $Ca(OH)_2$.[287]

Commercial nonsetting calcium hydroxide pastes may also be spun or injected to flow lightly over the pulp stump. Care must be taken to avoid trapping air bubbles when such products are applied, and it is recommended that they be overlaid with a hard-setting calcium hydroxide or glass ionomer cement[44] before meticulous cleaning of the cavity walls and restoration with a bonded composite resin restoration. Although little has been published on this topic, MTA probably represents a viable alternative to calcium hydroxide, provided the challenges in its retrieval are recognized.

APEXIFICATION

Apexification, or root-end closure, is the process in which a nonvital, immature, permanent tooth that has lost the capacity for further root development is induced to form a calcified barrier at the root terminus (Fig. 24-28). This barrier forms a matrix against which root canal filling or restorative material can be compacted with length control.

Unlike the pulp capping, pulpotomy, and apexogenesis procedures described previously, apexification at best results in closure of the root end and cannot be expected to cause further root development in terms of length or wall thickness. Apexification thus is regarded as a treatment of last resort in immature teeth that have lost pulp vitality. The growing body of recent evidence on pulp regeneration, even in infected, nonvital immature teeth, may also relegate this approach to the history archives in the years to come.[120]

For now, root-end closure techniques, both those involving the generation of a biologic calcific barrier and those involving artificial root-end closure with a material such as MTA, still have a place in practice and should be considered.

Before the introduction of conservative apical closure techniques, the usual approach to this problem was surgical. Although this could be successful, psychological and patient-management issues in patients who were usually young children offered many contraindications. Local dental considerations presented a further disincentive, including a reduction in the crown-to-root ratio if root-end reduction was required,

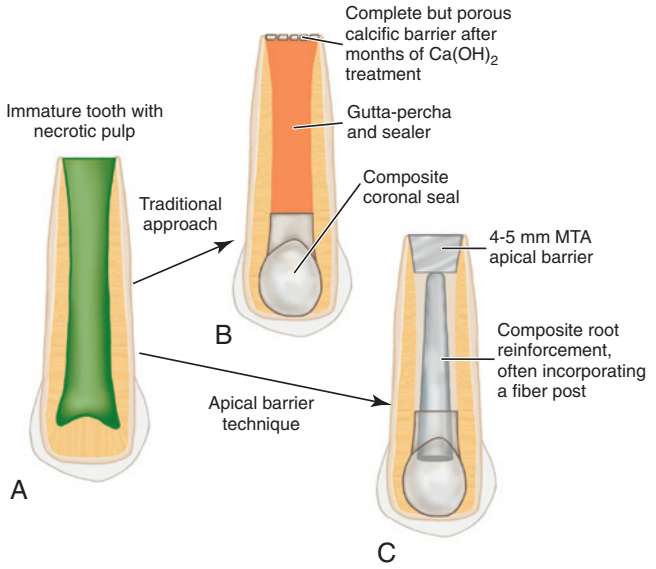

FIG. 24-28 Schematic representation of apexification. *Apexification* is a treatment performed to develop a hard barrier at an open root end. **A,** Immature permanent tooth with a nonvital pulp. **B,** Traditional approach: a "calcified barrier" forms at the root end after repeated dressing over many months with calcium hydroxide. The canal is subsequently filled with gutta-percha and sealer before coronal restoration. **C,** Artificial apical barrier technique: a 4- to 5-mm plug of mineral trioxide aggregate is placed at the root end. The canal space is subsequently restored with dual-curing composite resin, often accompanied by a fiber post to provide mechanical support.

and the intrinsic difficulties of sealing a fragile, incompletely formed apex with traditional materials. A more predictable and less traumatic approach was desirable.

Until recently, the most widely accepted technique has involved cleaning and filling the canal with a temporary paste, most commonly $Ca(OH)_2$, which was replaced at intervals over several months to stimulate the formation of an apical calcified barrier (see Fig. 24-28, *A* and *B,* and Fig. 24-29).[266]

Diagnosis of pulpal necrosis in a tooth with an incompletely formed apex is often difficult; the electronic pulp tester rarely provides meaningful data, and thermal tests often give equivocal or false results in young children and traumatized teeth. The presence of acute or chronic pain, percussion sensitivity, mobility, coronal discoloration, or a discharging sinus may be helpful guides, whereas radiographic diagnosis can be complicated by the normal radiolucencies appearing at the apices of developing teeth. Comparison of root formation with contralateral teeth should always be considered.

If any doubt persists, it is usually wise to adopt a watch and wait approach before entering the immature tooth endodontically. Only when convincing evidence indicates pulp breakdown should the tooth be entered, and in the interim, exposed dentin should be covered to reduce the risks of microbial entry to a potentially compromised pulp.

The extent of apical closure may be difficult to ascertain with plain radiographs. Three-dimensional imaging offers new opportunities to understand the complexity of root ends, which may have different conformations in mesiodistal and faciolingual dimensions (see Fig. 24-9). In practical terms, the precise limit of apical opening that can be filled by conventional means is unclear.

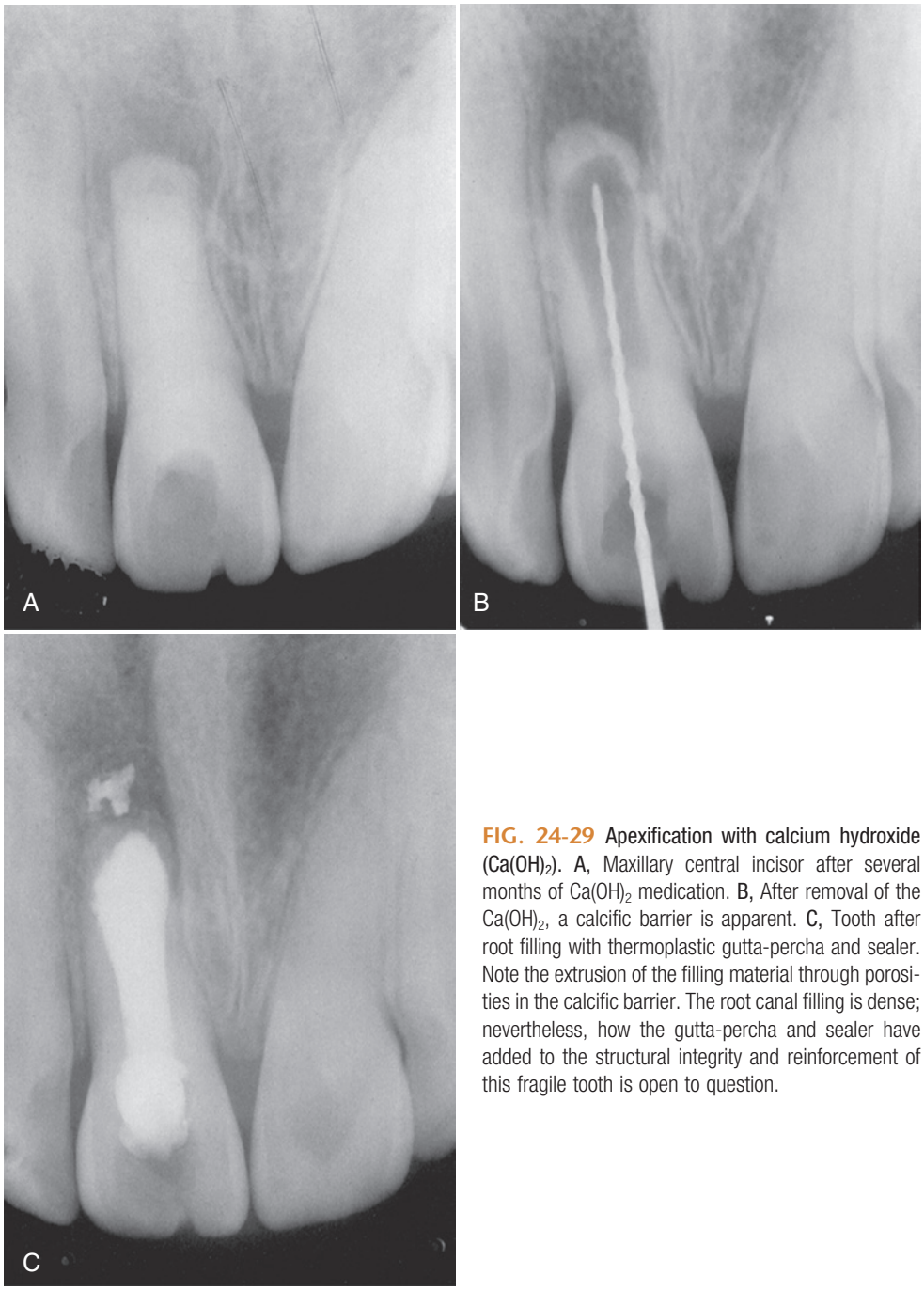

FIG. 24-29 **Apexification with calcium hydroxide ($Ca(OH)_2$).** **A,** Maxillary central incisor after several months of $Ca(OH)_2$ medication. **B,** After removal of the $Ca(OH)_2$, a calcific barrier is apparent. **C,** Tooth after root filling with thermoplastic gutta-percha and sealer. Note the extrusion of the filling material through porosities in the calcific barrier. The root canal filling is dense; nevertheless, how the gutta-percha and sealer have added to the structural integrity and reinforcement of this fragile tooth is open to question.

Many materials have been reported to stimulate apexification successfully. The use of nonsetting $Ca(OH)_2$ was first reported by Kaiser in 1964.[134] The technique was popularized by the work of Frank.[77] Since that time, $Ca(OH)_2$ alone or in combination with other drugs was the most widely accepted material for promoting apexification, until the development of MTA and the potential for rapid, artificial barrier formation.

For historical perspective, $Ca(OH)_2$ powder has been mixed with camphorated mono-chlorophenol (CMCP), metacresyl acetate, Cresanol (i.e., a mixture of CMCP and metacresyl acetate), physiologic saline, Ringer's solution, distilled water, and local anesthetic solution. All have been reported to stimulate apexification. Most reports in the U.S. literature[31,103,267] have advocated mixing $Ca(OH)_2$ with CMCP or Cresanol, whereas reports from other parts of the world[44,174] showed the same success using distilled water or physiologic saline as the vehicle. The addition of 1:8 barium sulfate to $Ca(OH)_2$ enhanced radiopacity with no apparent adverse effects on apexification.[298] Comparable outcomes have been noted in humans and animals with tricalcium phosphate,[41,140,233] collagen calcium phosphate,[198] osteogenic protein-1,[252] bone growth factors,[281] and a number of other materials.[298]

The most important factors in achieving apexification seem to be thorough debridement of the root canal to remove all necrotic pulp tissue and microbial infection, and sealing of the tooth to prevent the ingress of bacteria and substrate.[64]

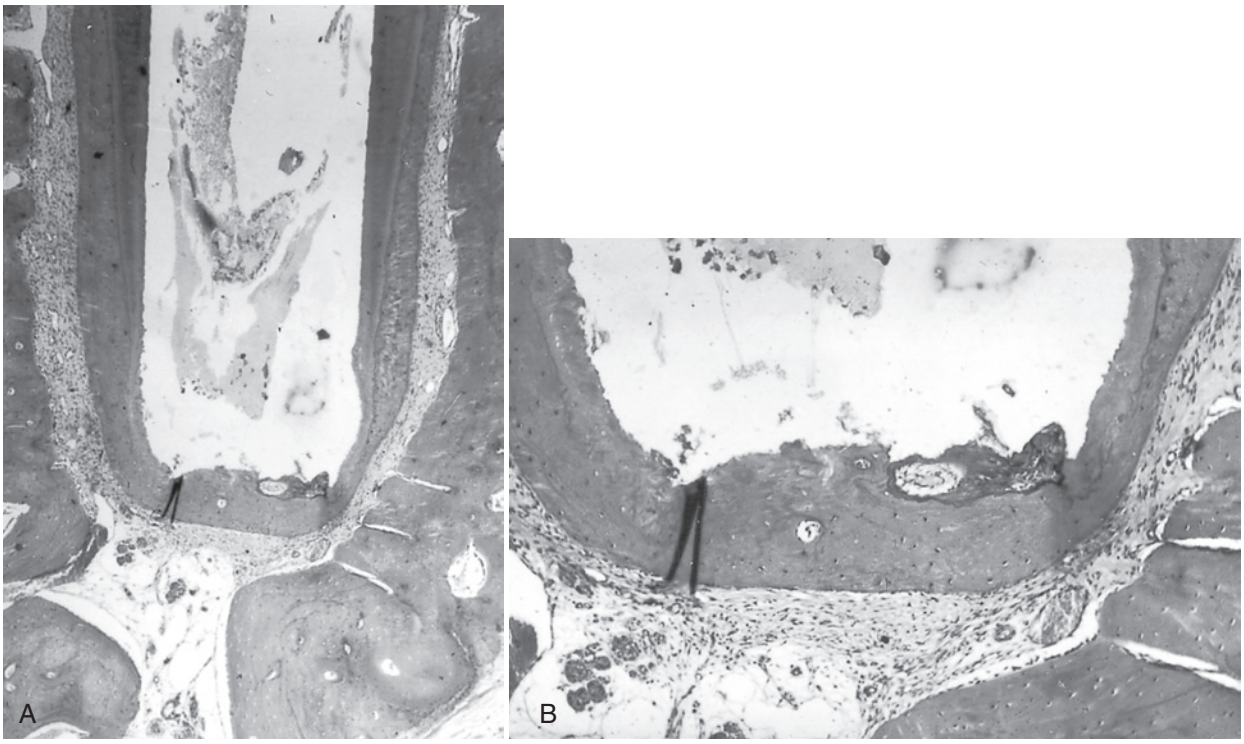

FIG. 24-30 Histologic section of a dog's tooth after calcium hydroxide apexification. A, Cementum-like mineralized tissue is closing the wide-open root end. Debris is apparent in the canal because of inadequate debridement before filling. B, Higher magnification shows cellular detail; the periodontal ligament is free of inflammation. Root canal filling material was lost in the histologic preparation. Note the presence of tissue communication through the apical barrier (stain, H&E).

Apexification does not occur when the apex of the tooth penetrates the cortical plate. To be successful, the apex must be completely within the confines of the medullary space.

Apexification Technique

In the apexification technique, the canal is cleaned and disinfected in line with the principles defined in Chapter 6. The use of a rubber dam is mandatory, and resourcefulness may be needed to isolate partially erupted or damaged teeth in children.

The access opening may require some extension to fully unroof pulp horns, but care should be taken not to heavily instrument the already thin and relatively fragile walls of the root.

The length of the canal is established by radiographs because the absence of an apical constriction may make electronic methods unreliable.[122,138] A constant drying point, determined with paper points, may provide helpful additional information on length.[14] Irrigation is central to the debridement of immature teeth, and with proper precautions, operators should not hesitate to benefit from the antimicrobial and tissue-solvent properties of NaOCl. Sonic, ultrasonic, and other vibratory devices capable of activating the irrigant within the canal may be advantageous,[291] and benefit may also come from the use of small brushes of the sort that are designed for interproximal tooth brushing or the application of etchants to post channels. After thorough debridement, the canal is dried and medicated with a fluid $Ca(OH)_2$ paste, carried into the canal with a Lentulo spiral or injected from a proprietary paste syringe. There is little evidence to commend any commercial $Ca(OH)_2$ paste over another in this application. The tooth is then sealed coronally, and the patient is recalled at 3-month intervals so that the clinician can wash out the $Ca(OH)_2$ paste and inspect clinically (with the aid of gutta-percha or paper points or by direct visual inspection through the microscope) and radiographically for the development of a calcified barrier (see Fig. 24-29, *B*). Treatment typically extends over 9 to 24 months, with obvious demands on patient and parent compliance. The reduced mechanical properties of root dentin and consequential risks of cervical root fracture after exposure to $Ca(OH)_2$ for 5 weeks or longer are also recognized.[46,307]

Histologic studies consistently report the absence of Hertwig's epithelial root sheath, and normal root formation should never be anticipated. Instead, differentiation of adjacent connective tissue cells into specialized cells appears to occur, and calcified tissue is deposited adjacent to the filling material. The calcified material that forms over the apical foramen was histologically identified as an osteoid (i.e., bonelike) or cementoid (i.e., cementum-like) material (Fig. 24-30).[31,103,266] In a primate study, bridging of the root end with osteodentin was reported after vital pulpectomy and canal medication with $Ca(OH)_2$/parachlorophenol paste. The material appeared to be distinct from but continuous with the cementum, dentin, and predentin at the root apex.[58] The closure of the apex may be partial or complete, but minute communications with the periapical tissues are consistently seen (see Fig. 24-28, *B*; Fig. 24-29, *C*; and Fig. 24-30). For this reason, apexification stimulated by

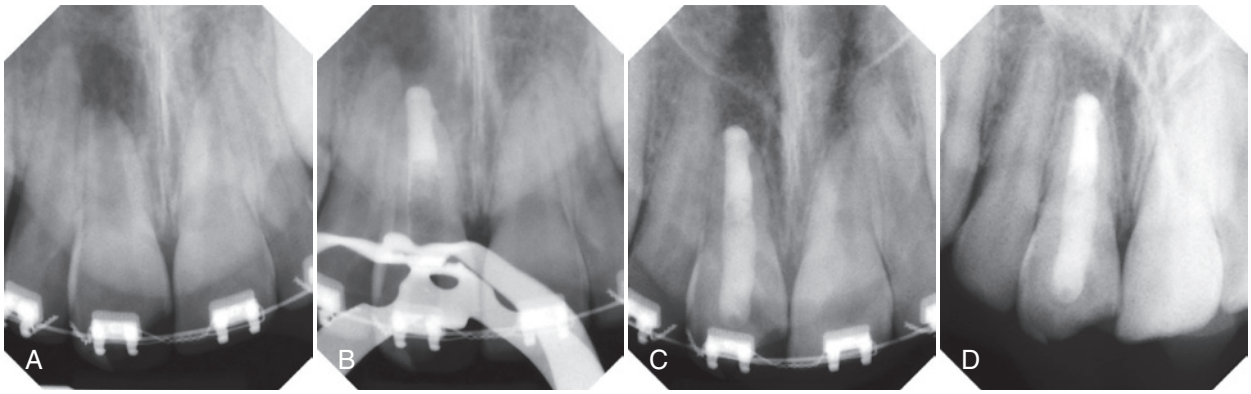

FIG. 24-31 **Mineral trioxide aggregate (MTA) apical barrier apexification procedure and restoration with bonded composite. A,** Maxillary right central incisor with a large periapical lesion. The patient is undergoing orthodontic treatment. **B,** After placement of MTA in the apical 4 mm of the canal. **C,** Six months later; the entire canal coronal to the MTA apical barrier is filled with bonded composite resin. Note that calcification has occurred periapically as orthodontic treatment has continued. **D,** Thirty months after treatment; the periapical lesion has healed.

pastes must always be followed by obturation of the canal with a permanent root canal filling, traditionally of thermoplastic gutta-percha and sealer, although MTA would be a good contemporary alternative.

Although the apexification technique with $Ca(OH)_2$ has enjoyed considerable tooth-preserving success, the many disadvantages of this protracted treatment have justified a search for alternatives, such as artificial barrier techniques, with their potential for more rapid treatment, and regeneration techniques, with their potential for continued tooth development.

Artificial Apical Barrier Techniques

(see Fig. 24-28, *C*)

Coviello and Brilliant[41] reported the use of tricalcium phosphate as an apical barrier in 1979. The material was packed into the apical 2 mm of the canal, against which gutta-percha was compacted. The treatment was completed in one appointment, and radiographic assessment confirmed successful apexification comparable to that achieved with $Ca(OH)_2$. Calcium hydroxide powder has also been used successfully as an apical barrier against which to pack gutta-percha.[248]

The use of MTA as an apical barrier was first reported in 1996,[281] and subsequent clinical investigations in animals[252] and humans[217,242,260] have established this as the standard, with biologic outcomes in terms of periapical healing and root-end closure at least comparable to those treated with $Ca(OH)_2$.[217] Simon et al.[260] treated 57 teeth in patients with a mean age of 18 years (standard deviation, 12 years). Of the 43 teeth with at least 12-month follow-up, healing had occurred in 81%. More recently, a series of 17 nonvital immature incisors in children with a mean age of 11.7 years was followed up for a mean of 12.5 months. This study reported 94.1% clinical success, with radiographic healing in 76.5% and a further 17.6% uncertain.[242] One study of 38 cases also suggested that the presence of a preoperative periapical lesion had no influence on the outcome.[195] This approach to the problem of root-end closure allows treatment to be completed on a short time scale, with advantages including improved patient compliance, reduced cost of clinical time, and the ability to securely restore the tooth at an earlier stage.[49] The risk of tooth fracture due to long-term $Ca(OH)_2$ medication is also eliminated.[307]

MTA Barrier Technique (see Fig. 24-28; also Figs. 24-31 and 24-32)

In the apexification technique, the canal is cleaned and disinfected as described for $Ca(OH)_2$ apexification. Lee et al.[151] have suggested that the tissue pH may affect the hydration reaction and final physical properties of MTA, and it has become standard practice to medicate canals for at least 1 week with $Ca(OH)_2$ to raise the acidic pH of the inflamed periapical tissues before permanent sealing.

When the tooth is free of signs and symptoms of infection, it is reisolated with a rubber dam and the $Ca(OH)_2$ is washed free, often with the help of ultrasonics and small brushes. After it is dried with large, sometimes inverted, premeasured paper points, the canal is filled incrementally with MTA, which is delivered to the canal with a dedicated MTA carrier or deposited in small pellets from an amalgam gun. The material can then be worked up the canal with premeasured pluggers, or inverted paper-points, set some 1 to 3 mm short of the root end, often with the help of sonic or ultrasonic energy to settle the material.[308] Apical matrices have not generally been considered necessary to limit the flow of MTA, although impressive-looking results may be obtained by packing the apical region with calcium sulfate before MTA application.[283] An apical plug 4- to 5-mm thick is usually considered optimal (see Fig. 24-31, *B*).[289] The adequacy of the apical plug is verified radiographically.

All excess MTA is removed from the canal walls by scrubbing with large, moistened paper points or brushes. Meticulous cleanup is important to allow optimal bonding of the subsequent composite resin restoration, which will extend deeply into the canal and offer internal reinforcement of the fragile root.

A very wet cotton pellet is placed in the canal to provide moisture for the setting reaction. The pellet should not be in contact with the MTA because fibers of the cotton can become impregnated into the material. Excess water in the access

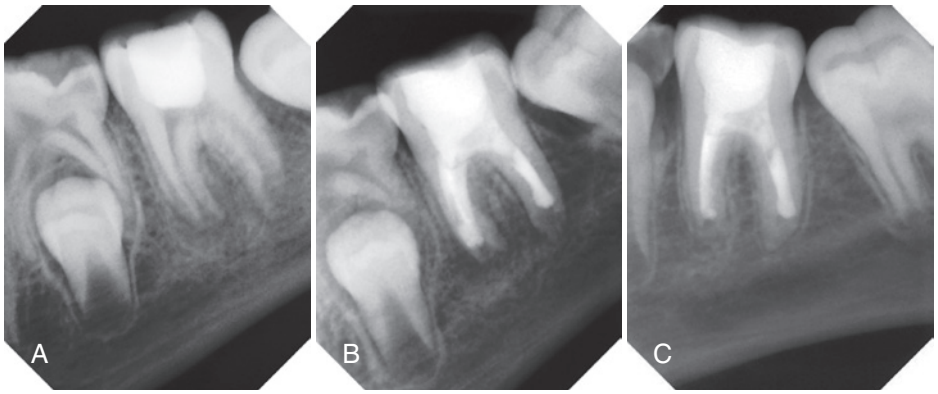

FIG. 24-32 Apexification with mineral trioxide aggregate (MTA) barriers in a mandibular permanent molar. **A,** Periapical lesion reveals open apices in this pulpless molar. **B,** Apical plugs of MTA and the remainder of the pulp space are restored with bonded composite resin. **C,** Two years later; closure of the root ends with cementum.

preparation is dried with cotton pellets, and the opening is sealed with a provisional restorative material, such as Cavit. At a subsequent appointment, the tooth is reisolated and the hard set of the MTA verified with an endodontic file or probe. If for some reason the MTA has not hardened, the canal can be recleansed and the procedure repeated before final bonded restoration.

Restoration After Apexification

Immature teeth, particularly those that are pulpless and have undergone apexification, are at high risk of fracture. It has been reported that, within 3 years of long-term $Ca(OH)_2$ medication and root filling with gutta-percha, 28% to 77% of immature teeth suffered a cervical root fracture.[46] The degree of dental development appeared to be a key variable. Clinically, it is the impression of the authors that rapid MTA plug techniques, in combination with the internal placement of bonded composite resin, appear to have virtually eliminated cervical root fractures. Many of the studies are laboratory based, using simulated rather than real immature teeth, but the use of contemporary dentinal bonding techniques has been shown to strengthen endodontically treated teeth to levels close to those of intact teeth.[100,110,136] A 2004 study[149] demonstrated significantly greater resistance to root fracture after placement of a 4-mm thick apical plug of MTA followed by an intracanal composite resin, compared with MTA followed by gutta-percha and sealer. Root reinforcement has also been reported to be improved by the cementation of a metal post within the channel created by removal of a light-transmitting composite curing post.[34] The potential of fiber-reinforced posts would also appear great,[26,251] though little clinical evidence has yet been published on the clinical treatment of immature teeth.

Alternative materials that have been suggested to bond and reinforce fragile roots include resin-modified glass ionomer cements[95] and, most recently, Resilon.[277] However, the evidence on Resilon remains contentious,[303] and the ideal of predictable bonding and tooth reinforcement throughout the length of the root canal system has not yet been achieved.[276]

Bonding of dual- or light-curing composite resin directly over the MTA plug, with no interposing layer of gutta-percha, has become an established clinical method (see Fig. 24-28, *C*, and Fig. 24-31, *C*). The clinician should take care to etch and bond the canal according to the manufacturer's instructions, with proper washout of etchant and avoiding gross pooling of unfilled resin. Complete resin infiltration of dentin cannot be guaranteed in the depths of a root canal, and the potential exists that host-derived metalloproteinases liberated by acid etching may degrade resin-dentin bonds with time. Flushing the preparation after etching with a synthetic protease inhibitor, such as 2% chlorhexidine, may help counter some of these adverse events.[33]

Clear light-transmitting posts (e.g., Luminex System, Dentatus USA, New York) have been developed to ensure complete bonding of deep increments of light-activated composite. This may be less of an issue with dual-curing materials.

In the Luminex technique, the clinician places a light-curing composite resin in the canal, building up 2-mm increments with care to avoid trapping air bubbles. The Luminex post is placed to the depth of the preparation, and the composite is cured by transmitting light through the post. After curing, the plastic post is trimmed to the cervical line, and the incisal opening is restored.

If a core is needed for crown placement, a Luminex post without serrations is used for curing the composite. Because the composite does not bond to the smooth post, it can be gently removed and a corresponding metal Dentatus post cemented into the space with a resin cement. A composite buildup for crown retention may then be completed.

A variety of commercial quartz and glass fiber post systems are also available for use in such applications, ensuring the delivery of light deep into the canal system and offering the potential for internal reinforcement (see Fig. 24-28, *C*).[245] The heads of fiber posts should always be covered with composite resin to prevent them from absorbing oral fluids and delaminating.

NEW HORIZONS FOR PULP REGENERATION

Until recently, few treatment options were available for the nonvital, permanent immature tooth. On confirming pulp necrosis, efforts could be made to preserve the tooth by apexification, or it could be extracted within a broader orthodontic treatment plan. This view has recently been challenged by a

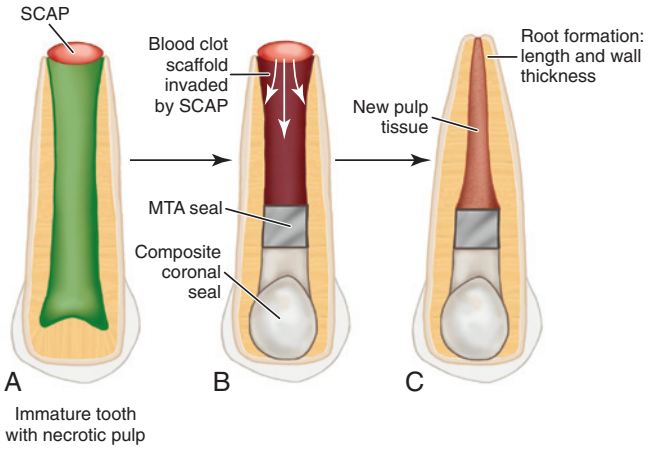

FIG. 24-33 Schematic representation of pulp regeneration. **A,** Immature, nonvital permanent tooth, showing the location of the apical papilla with its rich collection of stem cells. **B,** After the canal has been medicated, classically with a triantibiotic paste, it is overinstrumented to encourage bleeding up to the cervical level. A subsequent blood clot is overlaid with MTA and a sealing restoration, forming a scaffold for invasion by stem cells from the apical papilla (i.e., SCAP cells). **C,** Pulp regeneration is expected to allow continued root formation in a previously pulpless tooth.

number of ground-breaking reports in which nonvital immature teeth with clear evidence of pulp breakdown and periapical suppuration were encouraged to resume root formation (length and wall thickness), possibly by the regeneration of pulp tissue (see also Chapter 10).[15,37,128,132,279] Success is believed to depend on the activity of a newly identified population of stem cells, the so-called stem cells from apical papilla (SCAP) cells (Fig. 24-33, *A*), a hidden treasure with enormous potential for tissue regeneration and bioroot engineering.[121]

The potential of bioengineering is huge, and work continues to optimize scaffolds that may encourage revascularization of the pulp space and to explore the options of seeding cell populations into the properly sterilized pulp spaces of immature teeth.[182] Our call for action has never been louder as we seek effective, biologically based treatments for our pediatric patients.

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CHAPTER 25

Endodontic and Periodontal Interrelationships

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CHAPTER OUTLINE

Intercommunication between Pulpal and Periodontal Tissue
Influence of Pulpal Pathologic Condition on the Periodontium
Influence of Periodontal Inflammation on the Pulp
Theoretic Pathways of Osseous Lesion Formation
Primary Endodontic Lesions
Primary Endodontic Lesions with Secondary Periodontal Involvement
Primary Periodontal Lesions
Primary Periodontal Lesions with Secondary Endodontic Involvement
True Combined Lesions
Concomitant Pulpal and Periodontal Lesions
Differential Diagnosis
Lateral Periodontal Cysts
Treatment Alternatives
Forced Eruption or Extrusion
Summary

The interrelationships between pulpal and periodontal disease primarily occur by way of the intimate anatomic and vascular connections between the pulp and the periodontium; these interrelationships have been traditionally demonstrated using radiographic, histologic, and clinical criteria. Pulpal and periodontal problems are responsible for more than 50% of tooth loss.[16] Diagnosis is often challenging because these diseases have been primarily studied as separate entities, and each primary disease may mimic clinical characteristics of the other disease. Some studies suggest that these two diseases may have etiologic influences on the progression of the other disorder. Pulp tissue succumbs to degeneration by way of a multitude of insults, such as caries, restorative procedures, chemical and thermal insults, trauma, and periodontal disease. When products from pulp degeneration reach the supporting periodontium, rapid inflammatory responses can ensue that are characterized by bone loss, tooth mobility, and sometimes sinus tract formation. If this occurs in the apical region, a periradicular lesion forms. If this occurs with crestal extension of the inflammation, a retrograde periodontitis or reverse pocket is formed. However, the lesion formed has little anatomic similarity to a periodontally induced defect.

Periodontal disease, in contrast, is a slowly progressing disease that may have a gradually atrophic effect on the dental pulp. A periodontal lesion is used to denote an inflammatory process in the periodontal tissues resulting from dental plaque accumulation on the external tooth surfaces. Research has shown that periodontitis is characterized by the presence of localized inflammation or tissue infarction, a decrease in cells, resorption, fibrosis, and coagulation necrosis.* Dystrophic calcification may cause some degeneration in the pulp and further influence periodontal disease. In addition, periodontal treatments, such as deep root planing or curettage, use of localized medicaments, and gingival injury or wounding, may accelerate further pulpal inflammation and provoke the interrelated disease process.[72,78,79]

In recent years, periodontal disease has been shown to be related to (and possibly even the cause of) pulpal disease, and pulpal disease may cause periodontal lesions that behave differently from chronic destructive periodontitis. The effects of periodontal disease on the pulp and the potential for healing of certain periodontal lesions after endodontic therapy have been documented extensively.† This chapter discusses the intercommunication between pulpal and periodontal tissues; the effects of pulpal disease on the periodontium; periodontal disease and its effects on the pulp; and the classification, differential diagnosis, and management and prognosis of endodontic and periodontal problems.

INTERCOMMUNICATION BETWEEN PULPAL AND PERIODONTAL TISSUE

Several possible channels between the pulp and periodontium that lead to the interaction of the disease process in both

*References 48, 55, 67, 72, 76, and 78.
†References 7, 18, 45, 54, 69, and 79.

TABLE 25-1
Incidence of Furcation Canals

Investigators	Techniques	Incidence
Rubach and Mitchell (1965)[67]	Sectioned teeth	45%
Lowman et al. (1973)[54]	Dissecting microscope	Maxillary molars: 59% Mandibular molars: 55%
Burch and Hulen (1974)[13]	Radiopaque dye	Accessory furcal canals: 76%
Vertucci and Williams (1974)[83]	Hematoxylin dye	46%
Kirkham (1975)[43]	Radiopaque dye	23%
Gutmann (1978)[33]	Safranin dye	Maxillary molars: 28.4% Mandibular molars: 27.4%

tissues have been suggested. These include neural (i.e., reflex) pathways, lateral canals, dentinal tubules, palatogingival grooves, periodontal ligament, alveolar bone, apical foramina, and common vasculolymphatic drainage pathways. The most interconnected and evidenced relationship between the two tissues is by way of the vascular system, as illustrated anatomically by the presence of the apical foramen, lateral (i.e., accessory) canals, and dentinal tubules.* These communications, when they exist, may serve as paths for inflammatory reciprocity.†

The apical foramen is the most direct route of communication to the periodontium, but by no means is it the only location where pulpal and periodontal tissues communicate with each other. Lateral and accessory canals, mainly in the apical area and in the furcation of molars, also connect the dental pulp with the periodontal ligament. Table 25-1 lists the incidence of furcation canals. These have been suggested as a direct pathway between pulp and periodontium and typically contain connective tissue and vessels that connect the circulatory system of the pulp with that of the periodontium. Research has demonstrated that inflammation in the interradicular periodontal tissues may develop after induction of pulpal inflammation.[71,72] Serial sectioning of 74 teeth has revealed that 45% of accessory canals are present primarily in the apical region.[67] More significantly, lateral accessory canals in eight teeth were located more coronally on the roots. Among them, connection of the accessory canals with periodontal pockets was microscopically demonstrable in five of the specimens. Gutmann[33] introduced safranin dye into 102 molar teeth that were placed in a vacuum chamber and found that 28% of the teeth had furcation canals, although only 10% of the total group showed canals on the lateral root surface.

In addition to the apical foramen and lateral accessory canals, dentinal tubules have been suggested as another common pathway between the periodontium and pulpal tissue. Dentinal tubules contain cytoplasmic extensions or odontoblastic processes that extend from the odontoblasts at the pulpodentin interface to the dentinoenamel junction (DEJ) or the cementodentinal junction (CDJ). It has been reported that the pulp chamber can communicate with the external root surface by way of dentinal tubules, especially when the cementum is denuded.[33,86]

Palatogingival grooves are developmental anomalies of the maxillary incisor teeth; lateral incisors are more often affected than central incisors (4.4% versus 0.28%, respectively).[87] These grooves usually begin in the central fossa, cross the cingulum, and extend apically for varying distances. Generally, the incidence of palatogingival grooves ranges from 1.9% to 8.5%.[26,87] In their study, Everett and Kramer[26] reported that 0.5% of the teeth examined had a palatogingival groove extension to the root apex, thus contributing to an endodontic pathologic condition. Bilateral buccal radicular grooves have also been reported on maxillary incisors.[41]

Perforation of the root creates a communication between the root canal system and the periodontal ligament. This may occur as a result of overinstrumentation during endodontic procedures, internal or external root resorption, or caries invading through the floor of the pulp chamber. The prognosis for teeth with root perforation is usually determined by the location of the perforation, the time left unsealed, the ability to seal the perforation, the chance of building new attachments, and the accessibility of the remaining root canals. Teeth that have perforations in the middle or apical third of the root have the greatest chance of healing. The closer the perforation is to the gingival sulcus, particularly into the coronal third of the root or the furcation region, the greater the likelihood of apical migration of the gingival epithelium in initiation of a periodontal lesion.[4]

A vertical root fracture can produce a "halo" effect around the tooth radiographically (see also Chapter 2).[65] Deep periodontal pocketing and localized destruction of alveolar bone are often related to longstanding root fractures. The fractured root can mimic a radiographic profile of occlusal trauma, with localized loss of lamina dura, altered trabecular pattern, and a widened periodontal ligament. The fracture site provides a portal of entry for irritants from the root canal system to the surrounding periodontal ligament. Vertical root fractures have contributed to the progression of periodontal destruction in the presence of apparently successful endodontic tooth therapy and overall periodontal site stability.[66]

INFLUENCE OF PULPAL PATHOLOGIC CONDITION ON THE PERIODONTIUM

Pulpal pathosis as a cause of periodontal disease has received much attention during the past decade. Pulpal degeneration results in necrotic debris, bacterial by-products, and other toxic irritants that can move toward the apical foramen, causing periodontal tissue destruction apically and potentially migrating toward the gingival margin. Investigators termed this *retrograde periodontitis* to differentiate the process from *marginal periodontitis,* in which the disease proceeds physically from the gingival margin toward the root apex. When pulpal disease progresses beyond the confines of the tooth, inflammation extends and affects the adjacent periodontal attachment apparatus.[75] This inflammatory process often results in dysfunction of the periodontal ligament and resorption of alveolar bone, cementum, and even dentin.

*References 7, 18, 45, 54, 69, and 79.
†References 13, 33, 45, 47, 54, and 67.

The endodontic infection has been regarded as a local modifying risk factor for periodontitis progression if left untreated.[23] It is believed that an unresolved periapical infection could sustain endodontic pathogen growth, and infectious products would egress into the periodontium by way of the apex and lateral or accessory canals, as well as encourage osteoclastic activity. These may aggravate periodontal pocket formation and bone loss and impair wound healing to accelerate further periodontal disease development and progression. In addition, the high concentrations of the medicaments used for root canal therapy (e.g., calcium hydroxide, corticosteroids, and antibiotics) can irritate the periodontal attachment apparatus.[10,11] The nature and extent of periodontal destruction depend on several factors, including the virulence of the irritating stimuli present in the root canal system (e.g., microbiota, medications, foreign body reactions); the duration of the disease; and host defense mechanisms.[16] The rare development of endodontic-periodontal lesions argues that periradicular pathoses in general have little effect on localized inflammatory bone resorption, given that few combined lesions are seen in cases of apical periodontitis.

The ability of the periodontium to regenerate lost attachment apparatus on pulpless teeth has been questioned, especially if these teeth contain a root canal filling and have been denuded of cementum. One investigator suggested that endodontically treated teeth may not respond as well as untreated teeth to periodontal procedures. He found 60% osseous regeneration of periodontal defects in teeth not treated endodontically, compared with 33% defect fill in endodontically treated teeth.[68] However, in a monkey study, others reported that all tissue of the periodontium had the potential for regeneration after periodontal surgery, regardless of the status of the pulp (vital, filled, medicated, or open).[20] Another study reported that pulpal status has little influence on initial cementogenesis and that substances leaching from certain root canal filling materials do not alter deposition of new cementum.[63]

Although endodontic infections have been highly correlated with deeper periodontal pockets and furcation involvement in mandibular molars, the causal relationship between the two pathoses has not yet been established.[39] It has been suggested that endodontic treatment should occur before treatment of furcation lesions (i.e., bone regeneration) to ensure successful results. Extensive evidence is lacking to prove this hypothesis, but there is general agreement that with the proper endodontic treatment, periodontal disease of pulpal origin should heal. The question of whether endodontic infections play a significant role in affecting the health of the periodontium remains to be further elucidated.[59]

INFLUENCE OF PERIODONTAL INFLAMMATION ON THE PULP

Clinically, it is not uncommon to observe an advanced periodontitis spreading to the apical foramen, with associated pulp necrosis. It is also recognized that infection from a periodontal pocket may spread to the pulp through accessory canals, which occur most often in the furcation and closer to the apex of teeth.[58] Investigators proved that pulpitis and pulp necrosis can occur as a result of periodontal inflammation involving accessory and apical canals.[67] Bacterial products and toxins may also gain access to the pulp by way of exposed dentinal tubules. The pulpal reaction is influenced not only by the stages of periodontal disease, but also by the type of periodontal treatment, such as scaling, root planing, and administration of medication.[32] Inflammatory lesions of varying severity and necrotic pulp tissue are usually found in teeth with large canals or in cases in which periodontal breakdown has extended to the apex.[32] Whyman[86] stated that during periodontal therapy, the blood vessels supplying the pulp by way of accessory canals may be damaged. Another animal study found that 70% of root specimens examined showed no pathologic changes despite the fact that 30% to 40% of the periodontal attachment was lost.[8] The remaining 30% of roots displayed only small inflammatory cell infiltrates or formation of reparative dentin, or both, in areas where pulp was adjacent to root exposed through periodontal destruction. These tissue changes were frequently associated with root surface resorption, which suggests that dentinal tubules must be uncovered before irritation can be transmitted.

These observations suggest that the presence of an intact cementum layer is important for the protection of the pulp from toxic elements produced by the plaque microbiota, so periodontal disease and periodontal treatments should be regarded as potential causes of pulpitis and pulpal necrosis. It has also been reported that the pulps of teeth with longstanding periodontal disease develop fibrosis and various forms of mineralization. Canals associated with periodontally involved teeth were reported to be narrower than canals of teeth that were not periodontally involved. This result is thought to be a reparative process rather than an inflammatory response.[7,49]

Although consensus supports the theory that a degenerating or an inflamed pulp can affect the periodontium, not all researchers agree about the effect of periodontal disease on the pulp. Specifically, inflammatory alterations and localized pulp necrosis have been observed adjacent to lateral canals in roots exposed by periodontal disease.[67,71,72] Additional studies have failed to confirm a direct correlation between periodontal disease and pulp tissue changes.[19,57,81] When pathologic changes occur in the pulp as a result of periodontal disease, the pulp usually does not degenerate so long as the main canal is not involved.[48] It seems plausible to assume that periodontal disease rarely jeopardizes the vital function of the pulp. Generally, if the blood supply through the apical foramen remains intact, the pulp is usually capable of withstanding physiologic insults induced by periodontal disease.

THEORETIC PATHWAYS OF OSSEOUS LESION FORMATION

For the clinician, the close relationship between pulpal and periodontal disease is reasonably established on clinical and radiographic levels. Because interpretations vary as to which came first (the proverbial "chicken or egg" controversy), clinical data gathering in endodontic and periodontal problems is often complex, requiring a medical history review, pulp vitality testing, pocket and furcation probing, tooth mobility determinations, and critical examination of radiographs. When formulating a differential diagnosis, the clinician should first consider both the periodontal and pulpal status of the affected tooth. If an interrelationship in disease entities exists, appropriate treatment must be rendered to remove true causative factors and enhance the prognosis for tooth retention.[74] Figure 25-1 shows interrelationships between pulpal and periodontal disease.

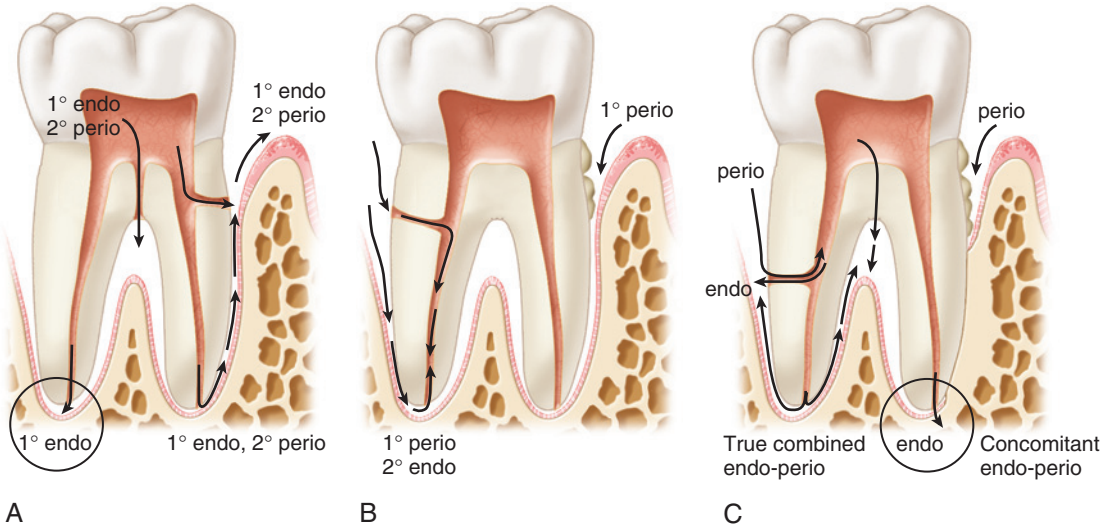

FIG. 25-1 Endodontic and periodontal pathways. **A,** Endodontic lesions. The pathway of inflammation is through the apical foramen, furcation canals, and lateral accessory canals to the periodontium. This results in a primary endodontic lesion, sometimes progressing to secondary periodontal involvement. **B,** Periodontal lesions. This is the progression of periodontitis by way of the lateral canal and apex to induce a secondary endodontic lesion. **C,** True combined endodontic and periodontal lesion and concomitant endodontic and periodontal lesions.

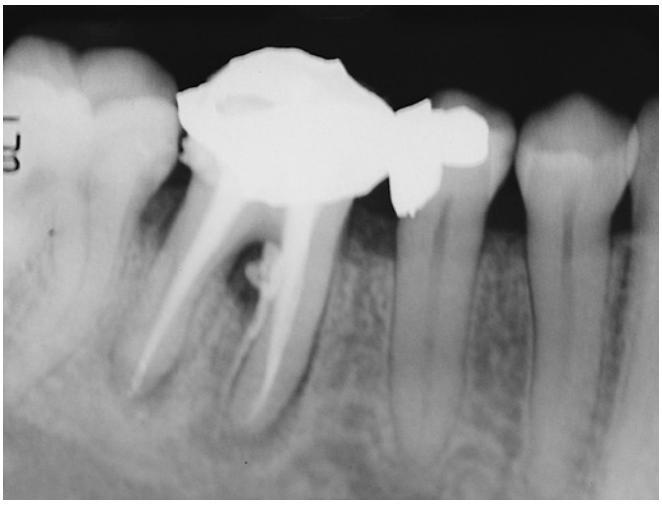

FIG. 25-2 Primary endodontic lesion. Mandibular molar showing endodontic filling material extending into the furcation and along the lateral root surface because of inadequate canal preparation. Furcation could be probed.

Primary Endodontic Lesions

Disease processes of the dental pulp frequently involve inflammatory changes. Caries, restorative procedures, and traumatic injuries are the most common causes. Typically, endodontic lesions resorb bone apically and laterally and destroy the attachment apparatus adjacent to a nonvital tooth. Inflammatory processes in the periodontium that occur as a result of root canal infection not only may be localized at the apex, but also may appear along the lateral aspects of the root (Fig. 25-2) and in furcation areas of two- and three-rooted teeth (Fig. 25-3).

The emergence of these processes may be associated with clinical signs of inflammation: pain, tenderness to pressure and percussion, increased tooth mobility, and swelling of the marginal gingiva, simulating a periodontal abscess. The suppurative process may cause a sinus tract along the periodontal ligament space or through patent channels (including the apical foramen and lateral accessory canals). This usually results in a narrow opening of the sinus tract into the gingival sulcus and pocket that can be easily traced with a gutta-percha cone or a periodontal probe. Such a tract can readily be probed down to the tooth apex, where no increased probing depth would otherwise exist around the tooth. In multirooted teeth, a periodontal ligament sinus tract can drain off into the furcation area and resemble a Grade III through-and-through furcation defect resulting from periodontal disease.

Clinically, endodontic testing procedures should reveal a necrotic pulp or, in multirooted teeth, at least an abnormal response, indicating that the pulp is degenerating. Because the primary lesion is an endodontic problem that has merely manifested itself through the periodontal ligament, complete resolution is usually anticipated after nonsurgical endodontic therapy, without any periodontal treatment.

Primary Endodontic Lesions with Secondary Periodontal Involvement

When a lesion of endodontic origin is not treated, pathosis usually continues, leading to destruction of the periapical alveolar bone and progression into the interradicular area, causing breakdown of surrounding hard and soft tissues (Fig. 25-4). As drainage persists through the gingival sulcus, accumulation of plaque and calculus in the purulent pocket results in periodontal disease and further apical migration of the attachment. When this occurs, not only does the diagnosis become more difficult, but the prognosis and treatment may be altered. Diagnostically, these lesions have a necrotic root canal and plaque or calculus accumulation, demonstrable by a probe and radiograph. Radiographs may show generalized periodontal disease with angular defects at the initial site of the endodontic involvement.

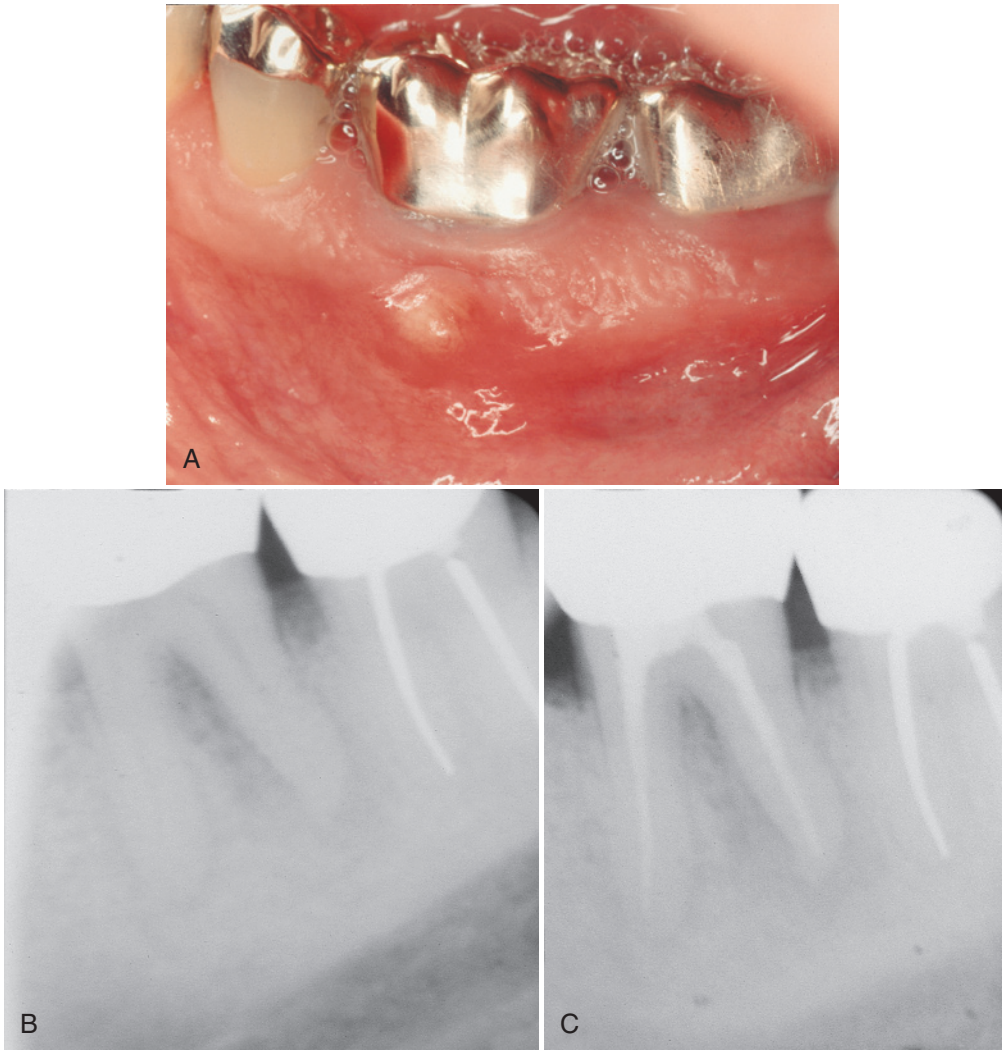

FIG. 25-3 **Primary endodontic lesion on mandibular first molar. A,** An abscess was noted on the buccal surface of the tooth. **B,** Apical radiolucency was evident on the distal root. **C,** Six months after root canal treatment, there is evidence of osseous healing at both the apex and furcation area.

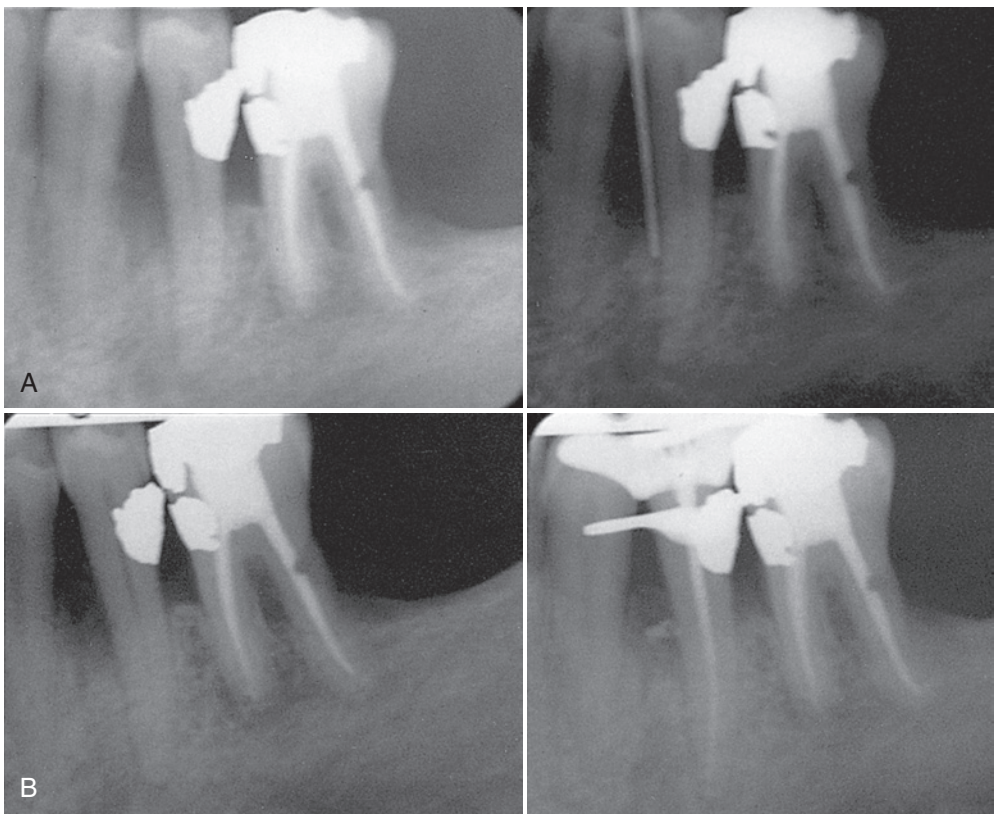

FIG. 25-4 **Primary endodontic lesion with secondary periodontal involvement. A,** The mesial aspect of the mandibular second premolar had deep periodontal pocketing (a periodontal probe in place demonstrated 6 mm of probing depth), even after periodontal therapy. **B,** Pulp tests were performed on the premolar; the tooth was nonresponsive. After endodontic treatment, the periodontal pocket resolved (filling of a lateral accessory canal after obturation is demonstrated).

Resolution of the primary endodontic and secondary periodontal lesion relies on treatment of both conditions. When only endodontic therapy is provided, only part of the lesion can be expected to heal. If endodontic therapy is adequate, the prognosis depends on the severity of periodontal involvement and efficacy of periodontal therapy.

Primary Periodontal Lesions

Periodontal disease has a progressive nature. It begins in the sulcus and migrates to the apex as deposits of plaque and calculus produce inflammation, causing loss of surrounding alveolar bone and supporting periodontal soft tissues. This leads to a loss of clinical attachment and formation of a periodontal abscess during the acute phase of destruction.[73] The progression of periodontal disease to the formation of osseous defects and subsequent radiographic appearance along lateral aspects of roots and in furcation areas is well known. These defects may or may not be in association with trauma from occlusion, which can often be the cause of an isolated periodontal problem. Osseous lesions of periodontal origin are usually associated with tooth mobility, and the affected teeth respond positively to pulp testing. In addition, careful periodontal examination usually reveals broad-based pocket formation and an accumulation of plaque and calculus. The bony lesion is usually more widespread and generalized than are lesions of endodontic origin (Fig. 25-5).

The prognosis for teeth affected by periodontitis worsens as the disease process and periodontal destruction progress. Treatment depends on the extent of the periodontitis and on the patient's ability to comply with potential long-term treatment and maintenance therapy. Because this is purely a periodontal problem, the prognosis depends exclusively on the outcome of periodontal therapy.

Primary Periodontal Lesions with Secondary Endodontic Involvement

As stated earlier, periodontal disease can have an effect on the pulp through dentinal tubules, lateral canals, or both. Primary periodontal lesions with secondary endodontic involvement differ from the primary endodontic lesion with secondary periodontal involvement only by the temporal sequence of the disease processes. The tooth with primary periodontal and secondary endodontic disease exhibits deep pocketing, with a history of extensive periodontal disease and, possibly, past treatment. When the pulp becomes involved, the patient often reports accentuated pain and clinical signs of pulpal disease. This situation exists when the apical progression of periodontal disease is sufficient to open and expose the pulp to the oral environment by way of lateral canals or dentinal tubules. On radiographs, these lesions may be indistinguishable from primary endodontic lesions with secondary periodontal involvement. The prognosis depends on continuing periodontal treatment subsequent to endodontic therapy.

True Combined Lesions

Pulpal and periodontal disease may occur independently or concomitantly in and around the same tooth. Once the endodontic and periodontal lesions coalesce, they may be clinically indistinguishable (Fig. 25-6). The prognosis of multirooted teeth with combined pulpal and periodontal lesions depends largely on the extent of the destruction caused by the periodontal disease component. A necrotic pulp or a failing endodontic treatment, plaque, calculus, and periodontitis will be present in varying degrees.

Concomitant Pulpal and Periodontal Lesions

An additional classification has been proposed for lesions that may commonly be seen clinically and reflect the presence of two separate and distinct entities.[6] This is referred to as the *concomitant pulpal and periodontal lesion* (Fig. 25-7). In essence, both disease states exist, but with different causative factors and with no clinical evidence that either disease state has influenced the other. This situation often goes undiagnosed, and treatment is rendered to only one of the diseased tissues in the hope that the other responds favorably. In actuality, the two disease processes must be treated concomitantly, and the prognosis depends on the removal of the individual etiologic

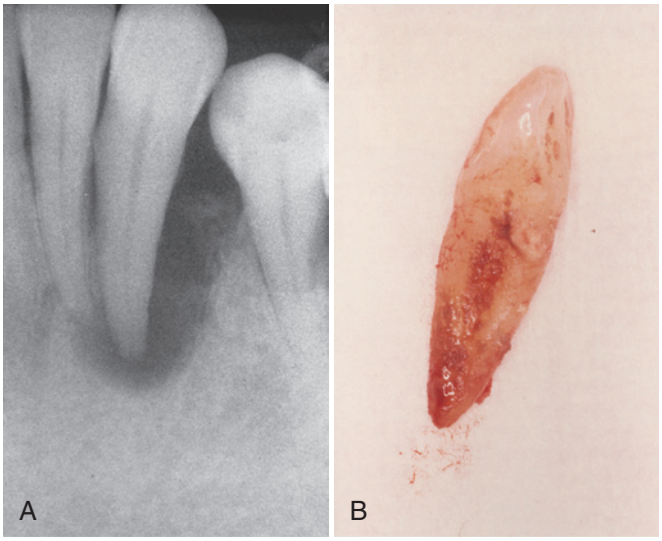

FIG. 25-5 **Primary periodontal lesion. A,** Mandibular cuspid exhibits extensive periodontal destruction; the tooth responded normally to pulp tests. **B,** Extracted tooth depicts extensive calculus accumulation and root concavity.

FIG. 25-6 True combined pulpal and periodontal lesions on the mandibular second premolar and first molar. Periodontal probing depths were to the apices in both teeth.

FIG. 25-7 Concomitant pulpal and periodontal lesion on the maxillary second premolar. An endodontic lesion was noted at the apex, with a noncommunicating periodontal pocket on the distal side.

factors and the prevention of any further factors that may affect the respective disease processes.

With the application of research findings and clinical experience, pathways of osseous lesion formation may be theorized. These concepts are intended as a guide for the evaluation and understanding of the clinician's successes and failures in treating teeth with osseous lesions. By understanding the nature of their formation, the clinician can better anticipate the healing potential after treatment.

DIFFERENTIAL DIAGNOSIS

During the course of treatment, clinicians are frequently presented with the dilemma of accurately assessing the contribution of endodontic and periodontal lesions. These lesions may be very separate from each other and present no extraordinary therapeutic consideration. In a few other situations, there is no obvious demarcation between the two lesions, which appear as one, both on radiographs and clinically. In the diagnosis of radiographic osseous lesions, the clinician must resist the temptation to label everything a "combined lesion." Table 25-2 summarizes the differential diagnosis between pulpal and periodontal lesions and highlights a number of common characteristics of these lesions.

Root fractures, especially vertical root fractures, present particular problems in diagnosis (Fig. 25-8). Symptoms and signs associated with vertical root fractures show a varying character and are frequently difficult to distinguish from those associated with periodontal and endodontic lesions. Breakdown can manifest itself on radiographs in a number of different ways. This may be anything from nothing detectable on radiographs to an area of rapid vertical bone loss, given that there is no way to predict when a patient may present for treatment. It is imperative for the clinician to take more than one radiograph at different angles, especially when no clear diagnosis emerges. In these cases, a minor alteration in the angulation may reveal a tooth fracture or periodontal furcation involvement. The diagnosis of vertical root fractures is often difficult because the fracture is usually not detectable by clinical inspection and radiographic examination unless there is a clear separation of the root fragments. Cemental tears or detachment of cementum from a root surface by means of trauma or aging has been reported.[14,51] The lesion often results in periodontal destruction and endodontic involvement (Fig. 25-9).[52,53]

Sometimes the definitive diagnosis of vertical root fractures has to be confirmed by exploratory surgical exposure of the root for direct visual examination.[84] Vertical root fractures have been associated with root-filled teeth where excessive lateral forces were applied during compaction or possibly with stress induced by post placement in root-filled teeth.[60] Clinical survey of fractured teeth also reveals that fractures are more common in teeth with extensive restorations, in older patients, and in mandibular posterior teeth.[30] The prognosis for teeth with vertical root fractures that involve the gingival sulcus and periodontal pocket usually is hopeless because of continuous bacterial invasion of the fracture space from the oral environment. Single-rooted teeth are generally extracted. In multirooted teeth, a treatment alternative is hemisection or resection of the fractured root. (See Chapter 21 for further discussion of vertical root fractures.)

Developmental grooves, primarily found in maxillary central and lateral incisors, are also capable of initiating localized periodontal destruction along the root surface.[26,41,50] It is

TABLE 25-2
Differential Diagnosis Between Pulpal and Periodontal Disease

	Pulpal	Periodontal
Clinical		
Etiology	Pulp infection	Periodontal infection
Vitality	Nonvital	Vital
Restorative	Deep or extensive	Not related
Plaque/calculus	Not related	Primary cause
Inflammation	Acute	Chronic
Pockets	Single, narrow	Multiple, wide coronally
pH value	Often acid	Usually alkaline
Trauma	Primary or secondary	Contributing factor
Microbial	Few	Complex
Radiographic		
Pattern	Localized	Generalized
Bone loss	Wider apically	Wider coronally
Periapical	Radiolucent	Not often related
Vertical bone loss	No	Yes
Histopathologic		
Junctional epithelium	No apical migration	Apical migration
Granulation tissues	Apical (minimal)	Coronal (larger)
Gingival	Normal	Some recession
Therapy		
Treatment	Root canal therapy	Periodontal treatment

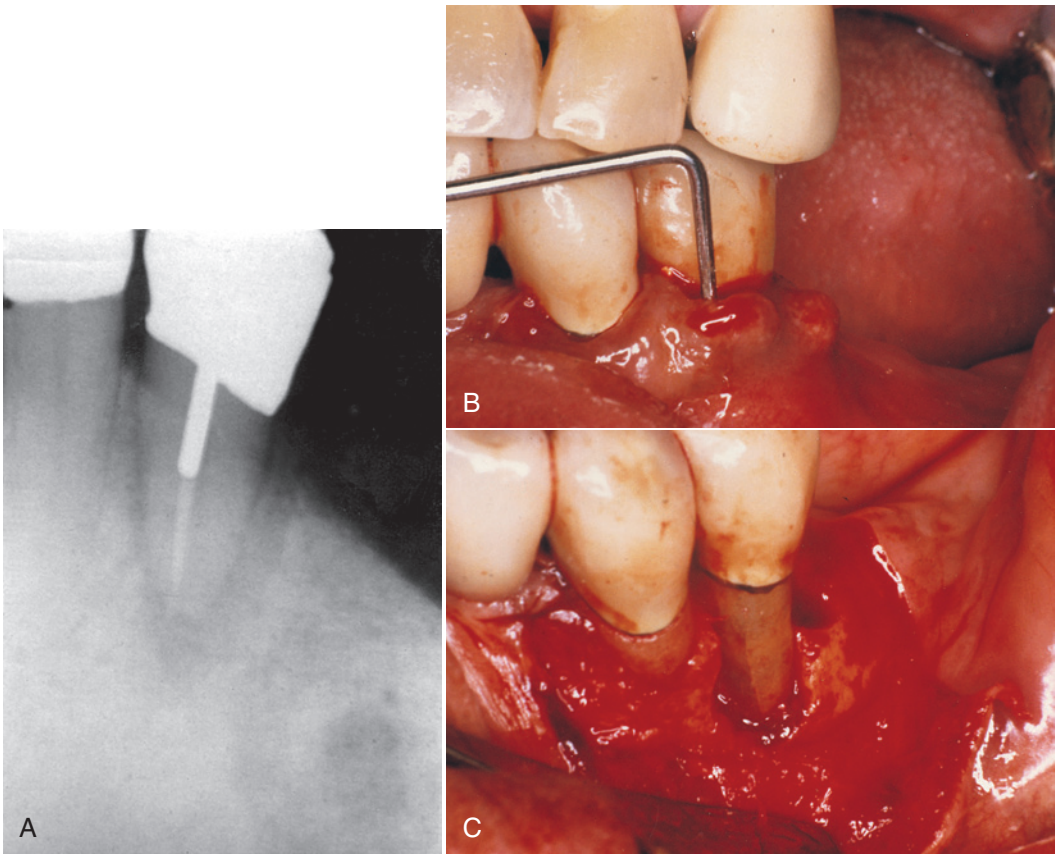

FIG. 25-8 **Vertical root fracture.** **A,** A radiograph revealed a widened periodontal ligament with a J-shaped radiolucency around the apex. **B,** A periodontal probe indicated more than 12 mm of probing depth. **C,** Exploratory surgery confirmed a vertical root fracture.

thought that they may be a genetic attempt to form an accessory root, but once plaque and calculus invade the epithelial attachment, the groove becomes a pathway for microbes and their noxious allies (toxins), with ample substrate from food debris to create a self-supporting periodontal lesion. Bone demineralization follows the path of the groove. Palatogingival grooves are often associated with poor periodontal health because of patients' inability to keep these areas clean, and a poor prognosis is usually assigned regardless of proper conventional therapy.[30,87] It is easy to identify these grooves if the clinician is aware of their existence. Clinically, these grooves may be asymptomatic, or symptomatic periodontal problems (either acute or chronic) may occur. It is thought that the pulp of these teeth may become secondarily involved and demonstrate symptoms of pulpal disease (Fig. 25-10). These lesions are often confused with the enamel projections in the furcation area of mandibular molars.[57] The prevalence of cervical enamel projections (Fig. 25-11) ranges from 18% to 45%.[36,57] Depending on the apical extent of these cervical grooves, some authors[36,57] also find a high association with pathologic furcation involvement (up to 82.5%[36]).

Lateral Periodontal Cysts

The clinical presentation of a lateral periodontal cyst is often without symptoms (Fig. 25-12). It may present as a gingival swelling on the facial aspect that may show pain and tenderness on palpation. Radiographic features are a well-circumscribed round or ovoid radiolucent area that usually has a sclerotic margin. Most lateral periodontal cysts are less than 1 cm in diameter and lie somewhere between the apex and cervical margin of a tooth. Three possible etiologies are reported in the literature: (1) reduced enamel epithelium, (2) remnants of dental lamina, or (3) cell rests of Malassez. Histologic evaluation reveals that these cysts are lined by epithelium that closely resembles reduced enamel epithelium lining. The lesion occurs predominantly in the fifth to seventh decades of life, with a predilection for males.[3] The lesion is usually slow growing. Treatment consists of careful excision to help prevent recurrence. The most common location is the mandibular cuspid-bicuspid area, although numerous cases have been reported in the anterior maxilla.

Altini and Shear[3] reported finding unicystic and multicystic (including botryoid) varieties. They found that lateral periodontal cysts were lined predominantly or exclusively by thin, reduced enamel, epithelium-like tissue that contained many clear cells, and epithelial thickenings referred to as *plaques*. Glycogen was present in the epithelium of two thirds of the cases, although not exclusively in the clear cells, many of which were not positive for glycogen. Some of their botryoid variety differed histologically, being lined predominantly by nonkeratinizing stratified squamous epithelium with crowded and pyknotic nuclei and no clear cells. One case contained melanin, and another showed epithelial crypt formation and superficial palisaded low columnar cells, as seen in the glandular odontogenic cyst. This raised the question of whether the latter may form part of the clinicopathologic spectrum of

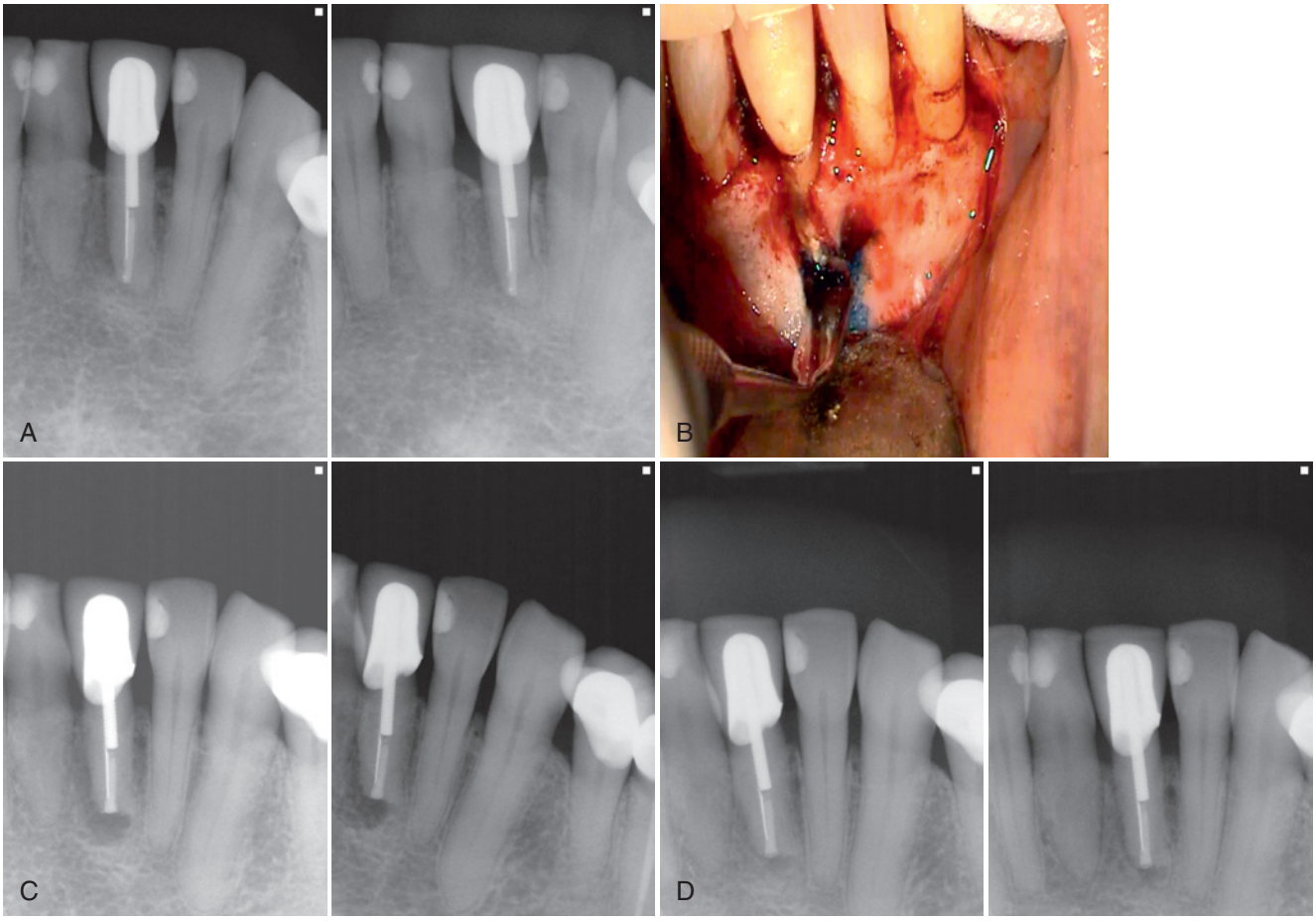

FIG. 25-9 **Cemental tear. A,** Radiographs revealed an endodontically treated tooth with post, separated instrument, and apical osseous breakdown; the tooth was very tender to both percussion and palpation. **B,** An "eggshell" piece of cementum on the buccal surface of the root before root-end resection; the cementum was removed with curettage. **C,** Postoperative radiographs after root-end resection, root-end preparation, and root-end filling. **D,** Six-month follow-up radiographs demonstrate periapical healing; the tooth was asymptomatic.

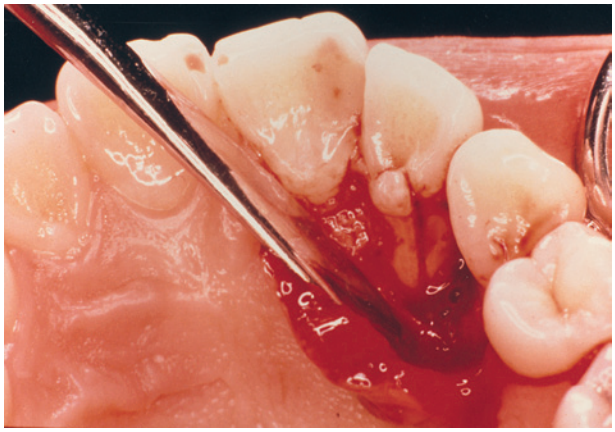

FIG. 25-10 Palatogingival groove on a maxillary lateral incisor with a periodontal defect.

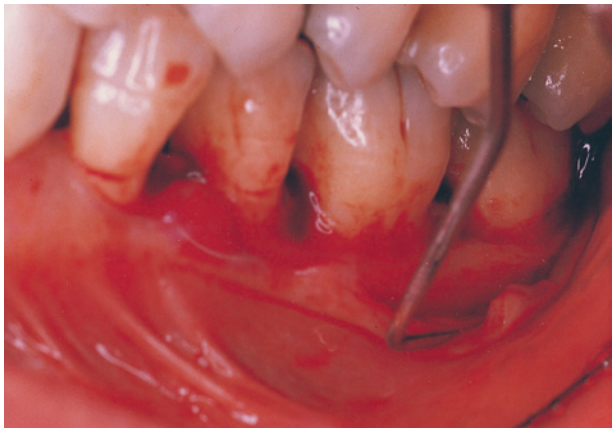

FIG. 25-11 Cervical enamel projection on a mandibular molar.

a lateral periodontal cyst. The histogenesis of lateral periodontal cysts is uncertain, but these cysts favor origin from reduced enamel epithelium. Some cases either do not fit a characteristic endodontic or periodontal lesion or do not respond to treatment as expected. A biopsy and histologic analysis are often recommended. Systemic diseases such as scleroderma, metastatic carcinoma, and osteosarcoma can mimic endodontic and periodontal disease visible on a radiograph. *The conscientious clinician must always be alert for lesions of nonendodontic or nonperiodontal origin and look for other causes.*

FIG. 25-12 Lateral periodontal cyst. **A,** Clinical photograph showing mild gingivitis around tooth #24; there is a 5-mm probing defect on the mesial aspect of tooth #24. **B,** A radiograph depicts the beginning lesion 2 years earlier. **C,** A radiograph shows the extent of the lesion at the time of referral to the periodontist. **D,** Initial flap reflection; the lesion is present interproximally between teeth #24 and #25.

TREATMENT ALTERNATIVES

When traditional endodontic and periodontal treatments prove insufficient to stabilize an affected tooth, the clinician must consider treatment alternatives. Generally, a localized periodontal defect associated with an endodontically untreatable tooth or an iatrogenic tooth problem is reason to explore other treatment options. Alternate treatments often consist of resection or regenerative approaches. Resection techniques focus on eliminating the diseased roots or teeth; regenerative efforts are aimed at restoring lost biologic structures. Resection methods involve removal of affected roots or extraction of involved teeth. When a tooth requires extraction, one of the first options for restoring occlusal function should include placement of dental implants with hybrid prostheses. Bone replacement grafts using guided tissue and bone regeneration techniques are ways to reestablish biologic structures that were lost during this disease process. Fixed partial dentures are still a viable option for some patients and certainly should be considered if abutment teeth already have restorations or endodontic treatment.

Root resection is the removal of a root, with accompanying odontoplasty before or preferably after endodontic treatment.[27] Formerly it was used when root canal therapy was considered too difficult, but now its indications are restricted to multirooted teeth in which one or more roots cannot be saved. The indications for root resection often include (but are not limited to) root fracture, perforation, root caries, dehiscence, fenestration, external root resorption involving one root, incomplete endodontic treatment of a particular root, severe periodontitis affecting only one root, and severe grade II or grade III furcation involvement. Root resection is a technique-sensitive procedure (Fig. 25-13) requiring a careful diagnostic process for selection of teeth that would likely be successful candidates, followed by meticulous interdisciplinary treatment. Factors such as occlusal forces, tooth restorability, and the value of the

FIG. 25-12, cont'd E, The lesion was enucleated and submitted for histologic evaluation; the diagnosis was a lateral periodontal cyst. F, Three-month posttreatment clinical photograph; tooth #24 was nonvital and symptomatic and was referred for root canal treatment. G, One-year posttreatment radiograph showing completed root canal treatment and osseous defect still present; continuous recall recommended. H, One-year posttreatment clinical photograph; probing depths were within normal limits; continuous recall was recommended.

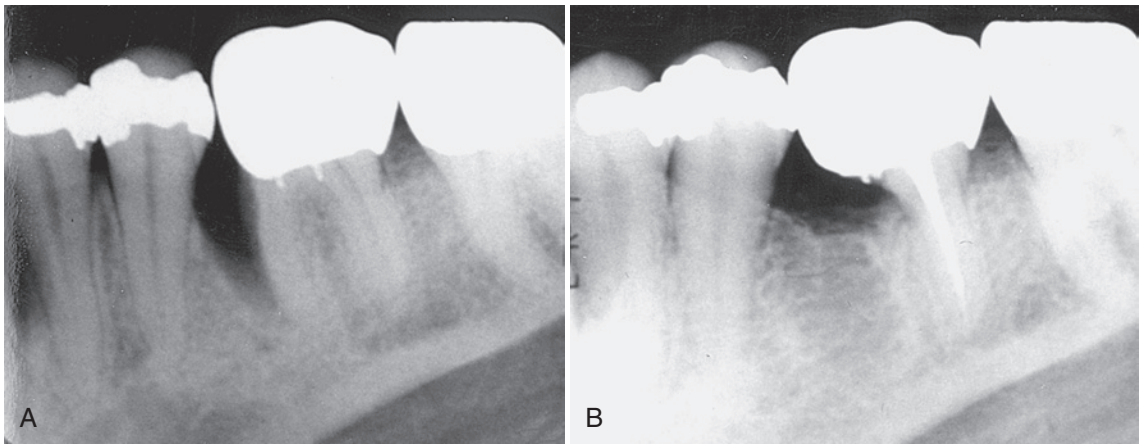

FIG. 25-13 Root resection to correct endodontic and periodontal defect. A, Pretreatment radiograph depicts severe periodontal involvement on the mesial. Endodontic treatment was performed before surgical removal of the mesial root. B, Five-year follow-up shows the remaining tooth well maintained.

remaining roots must be examined before treatment. A carefully constructed treatment plan is crucial to the success of this resection procedure.[31] Proper reshaping of the occlusal table and restoration of the clinical crown are essential, and the root surface must be recontoured to remove the root stump, thus preventing formation of a potential food trap.[42]

The effectiveness of this approach remains controversial, owing to the disparity of results reported in several long-term studies.* Retrospective longitudinal studies have observed the fate of sectioned teeth for time frames ranging from 3 to 12 years and have reported success rates ranging from 62% to 100%, with a low incidence (i.e., 10%) of periodontal breakdown. However, as most long-term studies will point out, the major cause of failure of resection procedures resides in failure of the endodontic and restorative components. Unique anatomic features, such as root length, curvature, shape, size, position of adjacent teeth, and bone density, may influence the end result. For example, root fusion makes resection all but impossible. The removal of roots purely to eliminate a resorptive or traumatic perforation defect, fractured root, or endodontically inoperable root usually results in definitive treatment. If, however, localized or generalized periodontal disease is present, conditions favorable for healing must be created, and concomitant periodontal therapeutic procedures can be implemented to restore the health of the periodontium.[31] The final restoration of root-resected teeth depends significantly on the nature of the resection, the amount of remaining tooth structure, the periodontal status, and the patient's occlusion. The prosthetic aspects of tooth restoration must be carefully assessed and integrated into the anticipated surgical procedure to ensure proper positioning of tooth margins relative to the osseous crest and also to manage the anticipated changes in occlusal relationships and masticatory forces.[85] Hemisection is the surgical separation of a multirooted tooth, usually a mandibular molar with severe furcation involvement, through the furcation in such a way that a root and associated portion of the crown may be removed or retained. A fixed partial denture often is placed in the area to restore the lost portion of the occlusal table (Fig. 25-14).

Controversy has also existed regarding the benefits of and need for endodontic therapy before root resection. Instances develop in which exploratory surgery is necessary; should the periodontal problem be more extensive than determined presurgically, removal of a root should be carried out at that time. In these instances, removal of the involved root without endodontic treatment would be acceptable, but root canal therapy should be performed as soon as possible after root removal.[29,77,80] After resection of a vital root, the pulpal opening in the crown may be sealed and restored with a permanent amalgam restoration or sedative base material (e.g., Dycal) as a temporary solution. Investigators evaluated vital-root resection in maxillary molar teeth for 9 years.[27] Amputated pulps were covered with Dycal base and amalgam. At 1 year, 38% of molar teeth remained vital, but at 5 years only 13% maintained vitality. These findings imply that the long-term prognosis for vital-root resection is poor, so *endodontic therapy should be done before or immediately after resection*. However, Haskell[35] reported that vitality of resected teeth can be maintained even after 16 years. Nonetheless, it is generally agreed that whenever possible, endodontics should be performed in advance (before root resection). If this is not possible, the endodontic treatment should be performed as soon as possible after vital-root amputation. Otherwise, pulpal complications may occur, such as internal resorption, pulpal inflammation, and necrosis.[2,79]

The concepts of guided tissue regeneration (GTR) and guided bone regeneration (GBR) have been used to promote bone healing after endodontic surgery.[28,62] Figure 25-15 illustrates an endodontic defect successfully treated with the GTR approach. Theoretically, the GTR barrier prevents contact of connective tissue with the osseous walls of the defect, protecting the underlying blood clot and stabilizing the wound.[34] An investigator treated large periradicular lesions with GTR barrier membranes and demonstrated that periradicular healing occurred more rapidly at membrane sites than at control sites.[61,62] The quality and quantity of the regenerated bone were superior with adjunctive use of the membrane than without such use. Similar findings were published in a case report with histologic examination of biopsy material obtained at barrier removal.[64] In addition, during examination of clinical cases, the closer the lesion is to the gingival margin, the greater the fluid and bacterial contamination from the sulcus (and the greater the risk of mechanical trauma). When GTR is considered, the combined endodontic and periodontal lesion probably has the least favorable prognosis compared with cases having only a periodontal lesion.[61]

Many varieties of GTR membranes are available for clinical use, but it is logical to use bioresorbable collagen and polymer membranes in endodontic surgeries because there is often no need for a second surgery to retrieve the membrane. Studies reveal similar results achieved with nonresorbable and resorbable membranes.[17] Long-term studies need to be completed to evaluate the success of this approach.

For more than 40 years, bone grafts have been used to treat osseous defects associated with periodontal disease.[70] Because the "apicoectomy" (i.e., root-end resection) defect has a surrounding bony wall, the need for adjunctive bone grafting during this procedure is questionable (unless the lesion is exceptionally large in diameter). With the introduction of the GTR concept, a combination of bone replacement graft and GTR membrane (Fig. 25-16) has shown promising results.[1,21,40,64,82] Further studies are required to explore the true benefit of this combination treatment approach in root-end resection procedures.

Forced Eruption or Extrusion

Teeth that have fractured, undergone extensive decay, or experienced internal or external root resorption or lateral perforation may be candidates for a forced eruption procedure, especially straight, tapering, single-rooted teeth (Fig. 25-17). To obtain good access for endodontic or restorative procedures, along with reduced probing depths, it may be necessary to perform extensive resection surgery to gain crown lengthening. Forced eruption may offer a better solution than surgical crown lengthening, which may produce poor aesthetics in some cases. A review of the literature reveals that most successfully treated cases are extrusions of less than 4 mm.[5,9,46] The infrequently used treatment method of forced eruption serves as an alternative to the sacrifice of the natural root system. Forced eruption can preserve the natural root system and related periodontal architecture, resulting in years of additional service for the patient. It also can maintain adjacent tooth structure while

*References 4, 10, 12, 15, 24, 27, and 44.

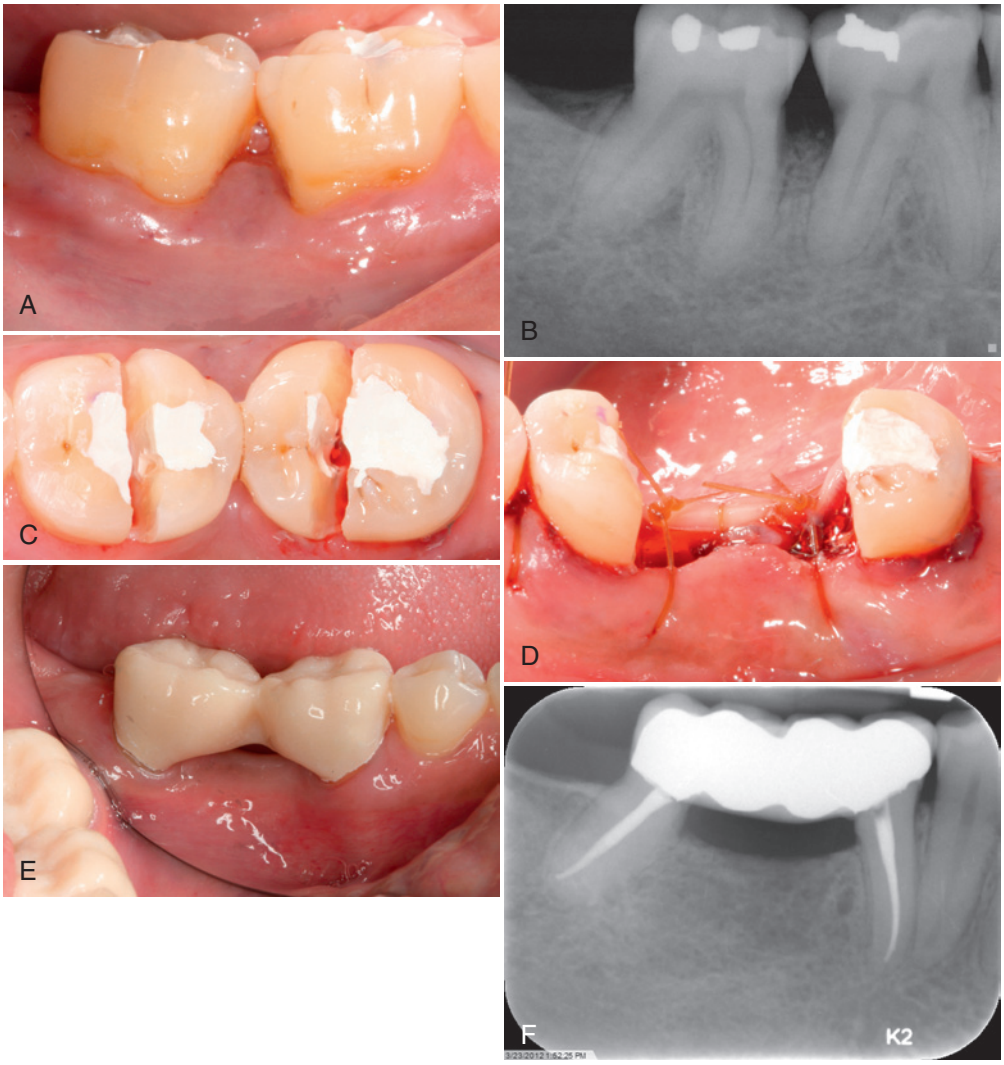

FIG. 25-14 Hemisection. **A,** Initial presentation of mandibular molars with severe attachment loss on the mesial aspect of tooth #31 and the distal aspect of tooth #30. **B,** Radiograph exhibiting interproximal periodontal defects. **C,** Occlusal view of hemisection after endodontic treatment. **D,** Suturing after removal of mesial half of tooth #31 and distal half of tooth #30. **E,** Eighteen-month follow-up with fixed restoration in place. **F,** Postoperative radiograph at 18 months showing normal bony and periodontal architecture.

retaining the option for future implant reconstruction if needed. Given the reported success of forced eruption, the technique should be used more often by dentists.[22,25] The increased use of implants in clinical treatments has stimulated clinicians' interest in augmenting bone in patients who have deficient alveolar ridges that might preclude ideal implant placement.

A nonsurgical technique for using the available bone for implant site development and fixture placement is orthodontic extrusion or forced eruption. The concept of extruding a tooth coronally by orthodontic forces and the clinical alterations in the soft tissue architecture of the periodontium demonstrated during orthodontic extrusive movement of periodontally compromised teeth have demonstrated probing depth reduction in some cases.[56] An investigator reported that tooth movement in the presence of inflammation might cause deepening of osseous defects. Often extrusions are accompanied by an immature-appearing tissue that is red compared with adjacent tissue. This results from the eversion of junctional epithelium, which is nonkeratinized in the sulcus; the vasculature is more readily visible until the newly exposed tissue has a chance to keratinize, in about 28 days.[37,38] Most extruded teeth should be retained in place twice as long as it took to force-erupt them to minimize relapse before attempting definitive restorative procedures.

SUMMARY

Endodontic and periodontal lesions result from the close interrelationship of pulp tissue and the periodontium. The major pathways of communication between the two types of tissue are the apical foramina, lateral and accessory canals, and dentinal tubules. The differential diagnosis of endodontic and periodontal lesions is not always straightforward and requires

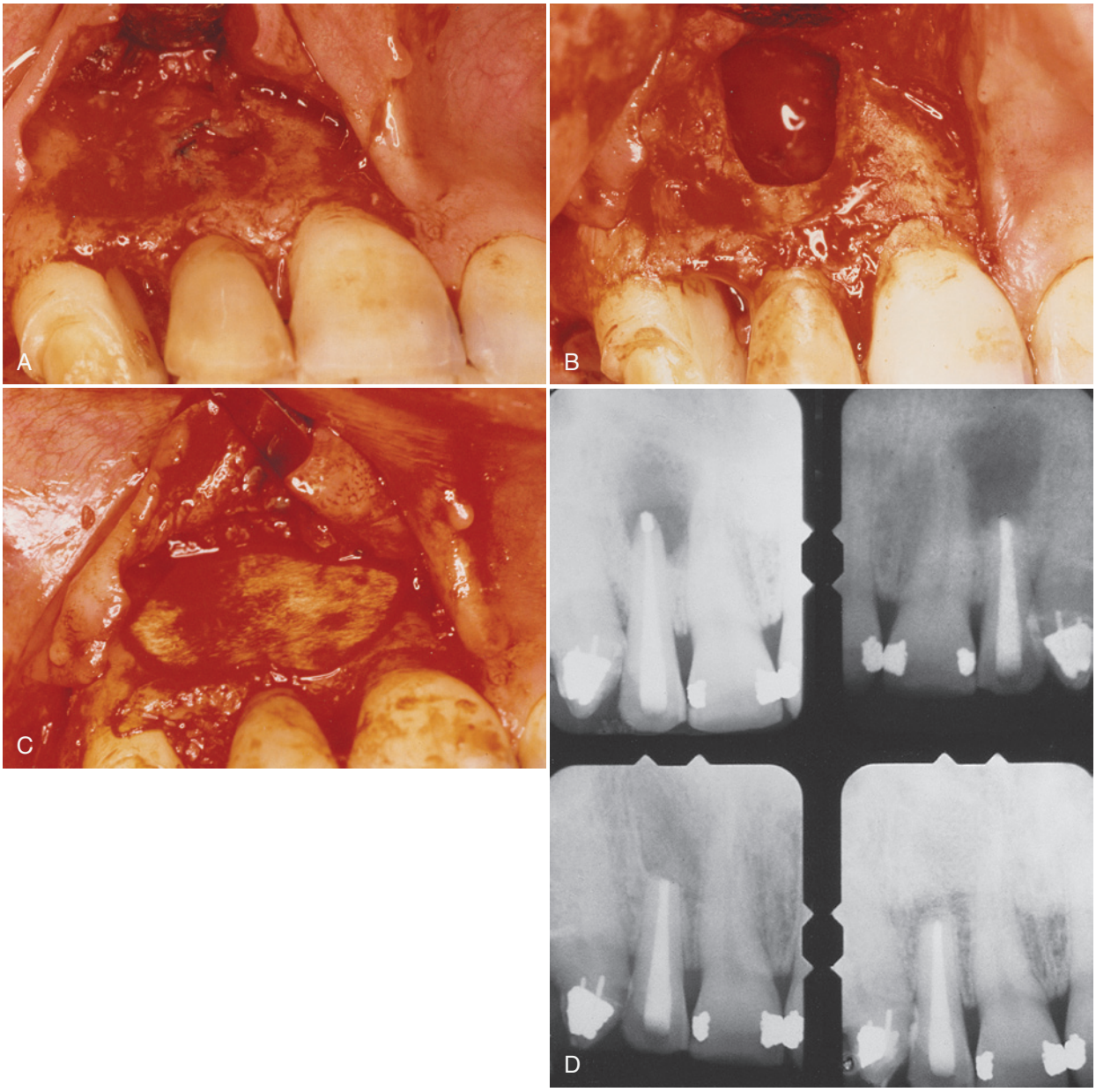

FIG. 25-15 **Periradicular endodontic surgery using the combination of bone replacement grafts and collagen guided tissue regeneration (GTR) barrier membranes.** **A,** A buccal flap was reflected (note extensive apical lesion). **B,** An osseous window was created with a bur. **C,** The defect was repaired with demineralized freeze-dried bone allograft (DFDBA) and covered with a collagen membrane. **D,** Radiographs show condition before surgery, at the time of surgery, and at 6 months and 2 years after surgery. At 2 years, the radiograph reveals complete bone regeneration.

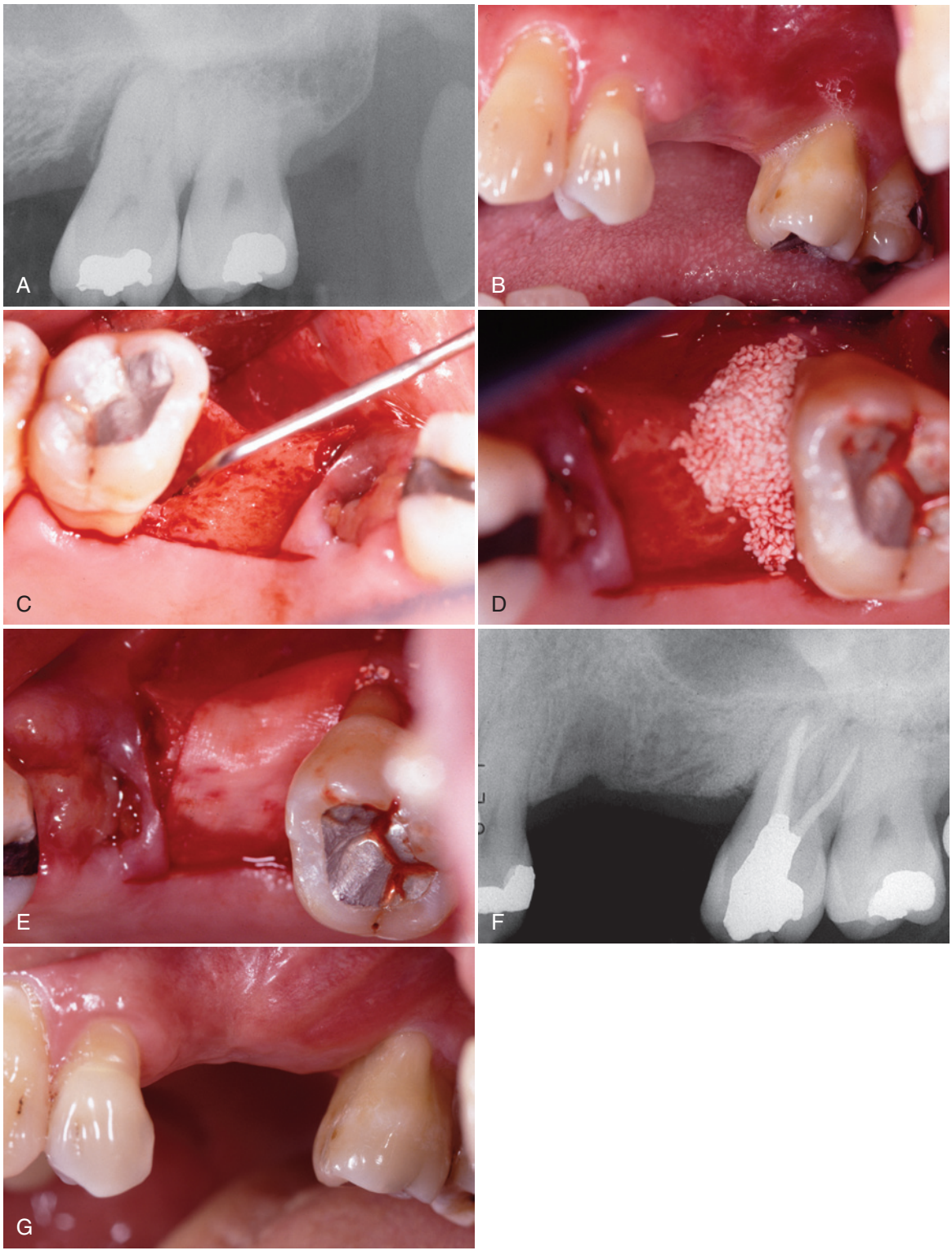

FIG. 25-16 **Periodontal surgery using guided bone regeneration.** **A**, A pretreatment radiograph demonstrates a significant osseous defect on the mesial aspect of tooth #14. **B**, Pretreatment clinical photograph of teeth #12 to #16. **C**, A full-thickness mucoperiosteal flap reflected with a periodontal probe 7 mm into osseous defect. **D**, Osseous defect filled with BioOss (cancellous bovine bone). Root canal treatment (RCT) was started for the patient before the surgical appointment; RCT was completed 6 weeks after osseous graft placement. **E**, Ossix membrane (collagen) placed over the BioOss graft. **F**, One-year posttreatment radiograph with evidence of osseous repair. **G**, One-year posttreatment clinical photograph of area.

FIG. 25-17 Clinical case of forced eruption. A, Initial presentation of tooth #29 (buccal view). **B,** Bonded wire to help extrude tooth #29. **C,** Lingual view of tooth #29 after 3 weeks of extrusion. **D,** Lingual view of cemented porcelain crown on tooth #29; 4 months after extrusion and after 1 month of crown retention.

clinical data accumulation from a number of diagnostic tests to obtain a correct diagnosis. When examining and treating the combined or individual lesion in endodontics and periodontics, the clinician must bear in mind that successful treatment depends on a correct diagnosis. Lesions with combined causes require both endodontic and periodontal therapy, and endodontic therapy should usually be completed first. Root resection and regenerative techniques offer alternative approaches, enhancing the clinician's ability to deal with these complex clinical problems.

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CHAPTER 26

Effects of Age and Systemic Health on Endodontics

CARL W. NEWTON | JEFFREY M. COIL

CHAPTER OUTLINE

An Aging Society
Medical History
Chief Complaint
Dental History
Subjective Symptoms
Objective Signs
Diagnosis and Treatment Plan
Consultation and Consent
Treatment
Isolation
Access
Preparation
Obturation
Success and Failure
Endodontic Surgery
Restoration

Dental service requirements are determined by four demographic and epidemiologic factors:

1. The population at risk
2. The incidence and prevalence of dental diseases
3. The accepted standards of care
4. The perceived need and expectations toward dental health by the public[26]

These factors are changing as a result of the aging of the population.

AN AGING SOCIETY

According to the U.S. Census Bureau, America's population aged 65 years or older grew by 82% between 1965 and 1995.[104] Between 1980 and 1995 this same population grew by 28%, and according to the most current demographic information, the number was 40.4 million in 2010, an increase of 15.3% since 2000. That represents 13.1% of the total U.S., population.[107] The *oldest old* are defined as those who are at least 85 years of age. This group is the fastest growing segment of America's senior citizen population. The number of people 85 years or older has more than doubled since 1965 and has grown by 40% since 1980.

The 75 million people born in the United States between 1946 and 1964 constitute the baby boom generation. In 1994, baby boomers represented nearly one third of the U.S. population. These people began to enter the 65 years or older category in 2011. As the baby boomers continue to age, the United States will see an unparalleled increase in the absolute number of older people.[16] One in eight Americans was age 65 or older in 1994; in a little more than 30 years, about one in five is expected to be in this group (Fig. 26-1).[107]

Information from the U.S. Census Bureau and the National Institute on Aging indicates that America's older population is unevenly distributed among geographic locations. The most populous states have the largest number of older adults; Florida and the Midwestern states currently have the highest proportions of older adults. In 1993, Florida was the only state where older adults accounted for greater than 16% of the population; however, it is predicted that 32 states will fall into this category by 2020.[73]

The increased longevity of the dentition, made possible by expanding fields of advanced restorative procedures and periodontology, implies that dentistry will see a substantial growth in the number of older patients.[24] Until 1983, individuals aged 65 years or older made an average of 1.5 dental visits annually, a lower utilization rate than for any other age group.[106] Between 1983 and 1986, a 29% increase in visits by those age 65 or older was noted by the National Health Survey.[105] According to this survey, older adults currently make more visits per year, on average, than those in the "all ages combined" category. The percentage of office visits, services provided, and patient expenditures attributed to patients age 65 or older exceeded the percentage of the population in that age group.[64] In the future, clinicians will routinely serve a growing number of older people, who will account for one third to two thirds of their workload.[62,63,65]

The growth of the population age 65 or older has affected every aspect of U.S. society, presenting both challenges and opportunities to policymakers, families, businesses, and health

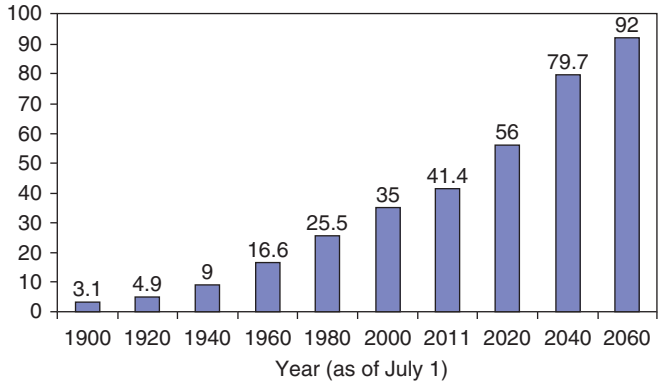

FIG. 26-1 Projected growth of U.S. adults age 65+ years to the year 2060. (From U.S. Department of Health and Human Services, Administration on Aging: A profile of older Americans: 2012. Available at: http://www.aoa.gov/. Accessed June 2014.)

care providers.[73] Age, ability to cooperate with dental treatment, and type of residence are important considerations in identifying preventive and treatment strategies.[69] Future dental services for older patients (including root canal procedures) are expected to be of two general types:

1. Services for relatively healthy older adults who are functionally independent
2. Services for older patients with complex conditions and problems who are functionally dependent[70]

The second group will require care from clinicians who have advanced training in geriatric dentistry. This age group is being targeted in dental education programs and advanced training through improved curriculums, research, and publications on aging. The National Institute on Aging has stated that all dental professionals should receive education in the treatment of older adults as part of their basic professional education.[21,70]

This chapter discusses the effect of aging on the diagnosis of pulpal and periapical disease and on successful root canal treatment. The quality of life for older patients can be significantly improved by saving teeth through endodontic treatment and can have significant value for overall dental, physical, and mental health.

With age, the simple pleasure of being able to eat what one wishes often becomes an issue, as does the increased need for proper diet and nourishment. Every tooth may be strategic, and old age is no time to be forced to replace sound teeth with removable appliances or dentures. Age is a factor in reduced stability and retention of removable partial dentures.[47] Consultation with older patients should help them overcome what may be a very limited knowledge of root canal treatment and lack of appreciation for regular dental care. Well-meaning friends and spouses who contend that their dentures are as good or better than their natural teeth may have had a lifetime of poor dental experiences or may have forgotten what it is like to enjoy natural dentition.

Negative social attitudes toward older adults tend to carry over into their care. Older patients are in danger of being dismissed as hopeless or not worth the effort. At times, clinicians shy away from providing care for seniors because of the perceived difficulty or cost of certain treatment procedures or because of complicated medical conditions. Clinicians sometimes consider older patients less able to pay for treatment because of their age and appearance. However, most older adults engage in normal activities and can recognize and afford the value of good dentistry.[9]

Most older patients have led active, productive lives; they are very interested in maintaining their dignity and do not consider themselves a bad investment. Tooth loss is often associated with the prospect of aging and loss of vitality.[31] As with any age group, older patients must be considered as individuals. This may prove difficult in the face of the tendency of many health care professionals to assign any person older than 65 years to the classification *geriatric* and to stereotype patients (e.g., confusion, dementia, poor treatment response). Each older adult patient comes with a unique psychological and social life history and a set of values, needs, and resources unlike those of any other patient the provider will see. Most seniors are more concerned with maintaining control of their lives than they are about being old.[82] Clinicians' perception of the effects of aging on the diagnosis and treatment of pulp and periradicular disease is also improving, along with the rate at which older patients need and seek specialty care.[40]

Because the primary function of teeth is mastication, it is presumed that loss of teeth leads to detrimental food changes and reduction in health.[49] However, this may not be the driving force when seniors are seeking treatment. Many times, social issues are the motivation for a senior to visit a clinician. After suggesting a mandibular anterior tooth extraction to an 89-year-old man, one clinician was told by the patient, "I can't, Doctor. I'm taking a cruise next week with my older brother and our girlfriends, I can't look like that." Most people older than age 60 who still have their own natural anterior teeth would not wish for a change in appearance if they needed prosthodontic reconstruction.[43]

Clinicians should not presume that they know what is best for senior patients or what they can afford without talking with the patient. The needs, expectations, desires, and demands of older people may exceed those of any age group, and the gratitude shown by older patients is among the most satisfying of professional experiences.

The desire for root canal treatment among aging patients has increased considerably in recent years. Older patients are aware that treatment can be performed comfortably and that age is *not* a factor in predicting success.*

In addition, obtaining informed consent requires that root canal treatment be offered as a favorable alternative to the trauma of extraction and the cost of prosthetic replacement. The distribution of specialists, the many technologic advances, and the improved endodontic training of all clinicians have broadened the availability of most endodontic procedures to everyone, regardless of age. Expanded dental insurance benefits for retirees and a heightened awareness of the benefits of saving teeth have encouraged many older patients to seek endodontia rather than extraction.[110] People with private dental insurance are more likely to visit a clinician. There is a trend toward less insurance in most demographic groups except the elderly, who also enjoyed benefits during their working years.[112] Most dental coverage in the United States is obtained through plans offered by employers; however, basic Medicare does not include such benefits, so seniors' spending on dental care is more sensitive to income than spending by

*References 7, 35, 52, 74, 88, 98, and 100.

younger age groups. Unfortunately, oral health provisions in recent U.S health care reform does not benefit older patients.[27]

This chapter compares the typical geriatric patient's endodontic needs with those of the general population. (Pulp changes attributable to age are discussed in Chapters 12 and 13; their effects on clinical treatment are also discussed in this chapter.)

MEDICAL HISTORY

It is important to focus on the factors that truly indicate the risks undertaken in treating the older patient. Clinicians must recognize that the biologic or functional age of an individual is far more important than his or her chronologic age. A medical history should be taken before the patient is brought into the treatment room, and a standardized form (see Chapter 1, Fig. 1-2) should be used to identify any disease or therapy that would alter treatment or its outcome. In general, the dramatic changes to the cardiovascular, respiratory, and central nervous systems caused by aging result in most of the older patient's drug therapy needs. However, the clinician also should consider the decline in renal[14] and liver function in older patients when predicting the behavior and interaction of drugs that may be used in dental treatment (e.g., anesthetics, analgesics, antibiotics).

The review of the patient's medical history is the clinician's first opportunity to talk with the patient. The time and consideration taken at the outset set the tone for the entire treatment process. This first impression should reflect a warm, caring clinician who is highly trained and able to help patients with complex treatments. Some older patients may need assistance filling out forms, and they may not be fully aware of their conditions or history. Some patients may withhold their date of birth to conceal their age for reasons of vanity or even fear of ageism. Some may not list current medications because they think it would be unnecessary information for the clinician to know. Vision deficits caused by outdated glasses or cataracts can adversely affect a patient's ability to read the small print on many history forms. Consultation with the patient's family, guardian, or physician may be necessary to complete the history; however, oral health care professionals function as primary caregivers in today's health care environment, and they are ultimately responsible for involving the patient in decision making and treatment choices.[38]

Today's dental office can be a confusing environment of required paperwork. An updated history, including information on compliance with any prescribed treatment and sensitivity to medications, must be obtained at each visit and reviewed. The elderly usually take more drugs, prescription and nonprescription, than the general adult population, and drug interactions and adverse drug reactions are more likely to result from polypharmacy.[44] As polypharmacy continues to increase, the likelihood of adverse drug interactions in older dental patients also will increase.[45] It is very important that the clinician take a careful history and update it at every visit.[66] *The Physicians' Desk Reference* (PDR) should be consulted, and any precaution or side effect of a medication noted. The PDR is available online (*www.pdr.net/*). Several other websites (e.g., Epocrates *[www.epocrates.com]*) have been developed specifically to be consulted about drug interactions and dental treatment.

Although geriatric patients are usually knowledgeable about their medical history, some may not understand the implications of their medical conditions in relation to dentistry or may be reluctant to let the clinician into their confidence. Their perceptions of their illnesses may not be accurate, so any clue to a patient's condition should be investigated.

Symptoms of undiagnosed illnesses may present the clinician with a screening opportunity that can disclose a condition that might otherwise go untreated or may lead to an emergency. Management of medical emergencies in the dental office is best directed toward prevention rather than treatment. Squamous cell carcinoma and salivary gland tumors increase with age, especially past age 80, and the prognosis is less favorable.[19]

Osteonecrosis of the jaw (ONJ) is a painful condition secondary to bisphosphonate therapy. Although it occurs at a much higher rate in patients receiving intravenous treatment for bone diseases than it does in older patients receiving the oral treatment common for osteoporosis, forced eruption and tooth or root retention are recommended alternatives to the risk of extraction (Fig. 26-2).[90] In addition, when discussing treatment options of endodontic retreatment versus endodontic surgery, the clinician should steer patients at risk for developing ONJ away from the surgical option (Fig. 26-3). The American Association of Endodontists' position paper on the endodontic implications of ONJ is available online (*www.aae.org/guidelines*).

Few families are without at least one member whose life has been extended as a result of medical progress. A great number have had diseases or disabilities controlled with therapies that may alter the clinician's case selection. Root canal treatment is certainly far less traumatic in the extremes of age or health than is extraction and implant placement.

Chief Complaint

Most patients experiencing dental pain have a pulpal or periapical problem that requires root canal treatment or extraction. Dental needs are often manifested initially in the form of a complaint, which usually contains the information necessary to make a diagnosis. The diagnostic process is directed toward determining the vitality of the pulp, whether pulpal or periapical disease is present, and which tooth is the source (see Chapter 1).

Without leading, the clinician should allow the patient to explain the problem in his or her own way. This gives the examiner an opportunity to observe the patient's level of dental knowledge and ability to communicate. Visual and auditory impairments may become evident at this time. An effort should be made to maintain privacy if the clinician is required to speak louder because of a patient's hearing impairment.

Patiently encouraging the patient to talk about problems may lead into areas of only peripheral interest to the clinician, but it establishes a needed rapport and demonstrates sincere interest. A patient may show some feelings of distrust if there is a history of failed treatments or if well-meaning, denture-wearing friends or relatives have claimed normal function and extolled their freedom from the need for dental treatment. The effect of the "focal infection" theory is still evident when other aches and pains cannot be adequately explained and loss of teeth is accepted as inevitable. The best patients are those who have already had successful endodontic treatment. Older patients are more likely to have already had root canal treatment and have a more realistic perception about treatment comfort.

Most geriatric patients do not complain readily about signs or symptoms of pulpal and periapical disease and may consider them to be minor compared with other health concerns and

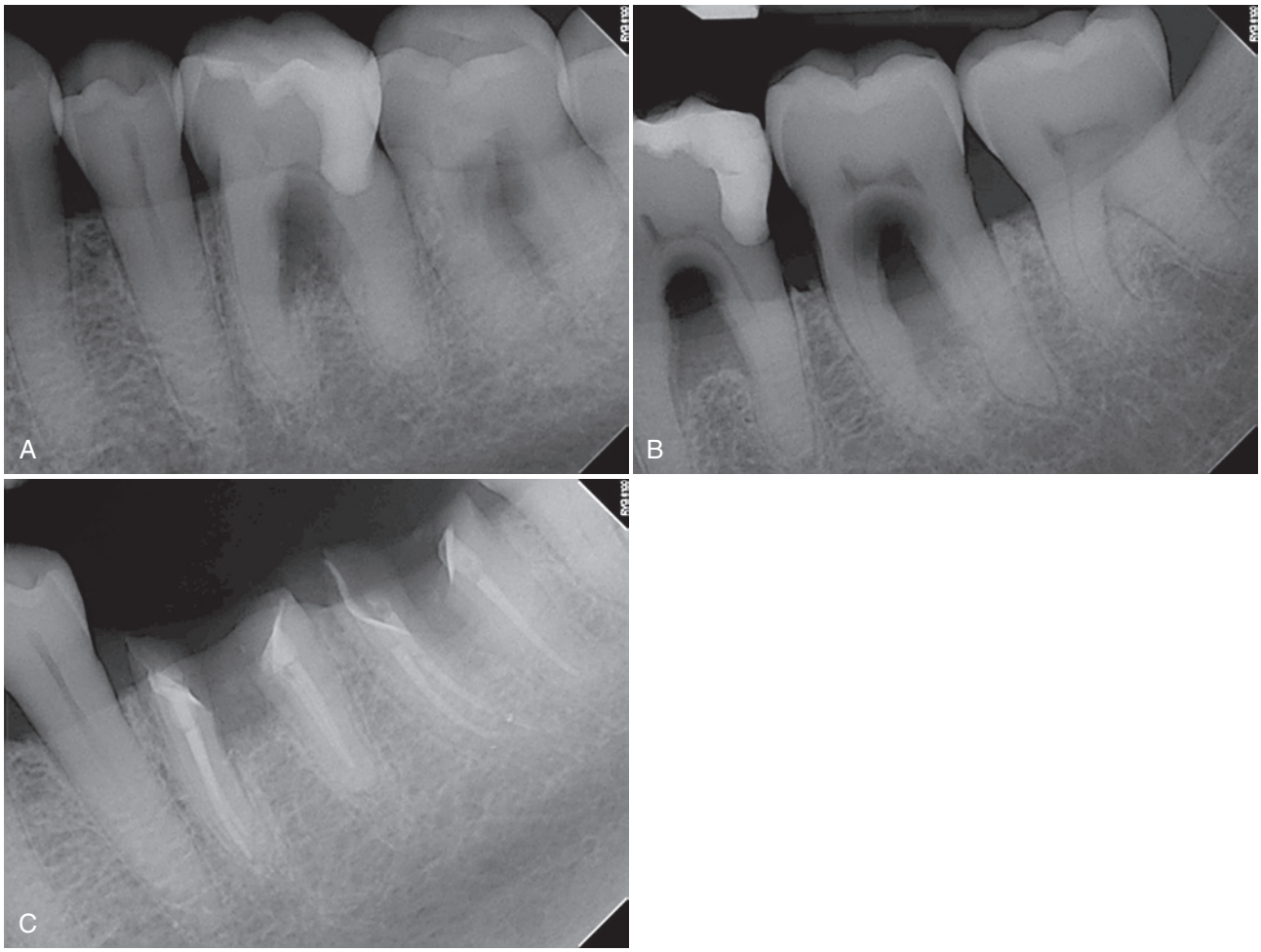

FIG. 26-2 **A** and **B**, This patient had undergone IV bisphosphonate chemotherapy and presented for consultation about these asymptomatic vital teeth. The root decay is a consequence of increased decay susceptibility due to gingival recession and xerostomia. **C**, After consultation with the patient's oncologist, it was agreed that nonsurgical root canal therapy (NSRCT) was the best option to avoid the complication of pain and infection from certain exposure during decay removal and restoration on teeth that would otherwise be extracted as unrestorable. (Courtesy Dr. Joe Spires.)

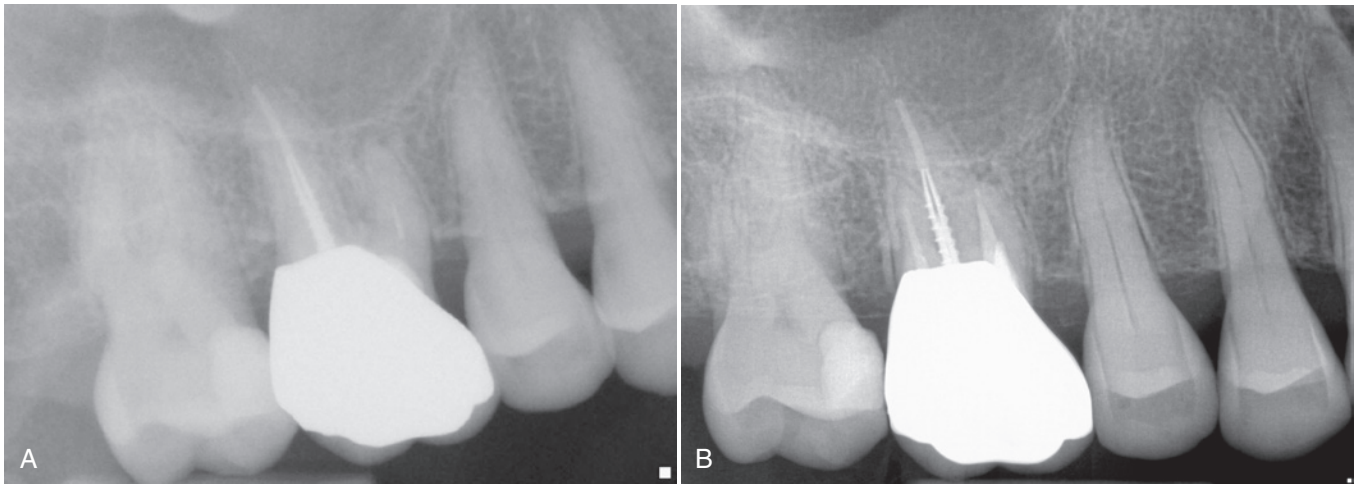

FIG. 26-3 **A**, A female patient at risk for osteonecrosis of the jaw had a symptomatic molar tooth with a broken instrument in the MB root. **B**, Nonsurgical root canal retreatment was performed on the MB root to avoid surgery.

discomfort. A disease process usually arises as an acute problem in children but assumes a more chronic or less dramatic form in the older adult. The *mere presence* of teeth indicates proper maintenance or resistance to disease. A lifetime of experiencing pain puts a different perspective on interpreting dental pain.

Pain associated with vital pulps (i.e., referred pain; pain caused by heat, cold, or sweets) seems to be reduced with age, and its severity seems to diminish over time. Heat sensitivity that occurs as the only symptom suggests a reduced pulp volume, such as that occurring in older pulps.[51] Pulpal healing capacity is also reduced, and necrosis may occur quickly after microbial invasion, again with reduced symptoms.

Although complaints are fewer, they are usually more conclusive evidence of disease. The complaint should isolate the problem sufficiently to allow the clinician to take a periapical radiograph before proceeding. Studying radiographs before an examination can prejudice rather than focus attention; accordingly, they should be reviewed *after* the clinical examination has been completed.

DENTAL HISTORY

The clinician should search patients' records and explore their memories to determine the history of involved teeth or surrounding areas. The history may be as obvious as a recent pulp exposure (Fig. 26-4) and restoration, or it may be as subtle as a routine crown preparation 15 or 20 years earlier (Fig. 26-5). Any history of pain before or after treatments may establish the beginning of a degenerative process. Subclinical injuries caused by repeated episodes of decay and its treatment may accumulate and approach a clinically significant threshold that can be later exceeded after additional routine procedures. Multiple restorations on the same tooth are common (Fig. 26-6).

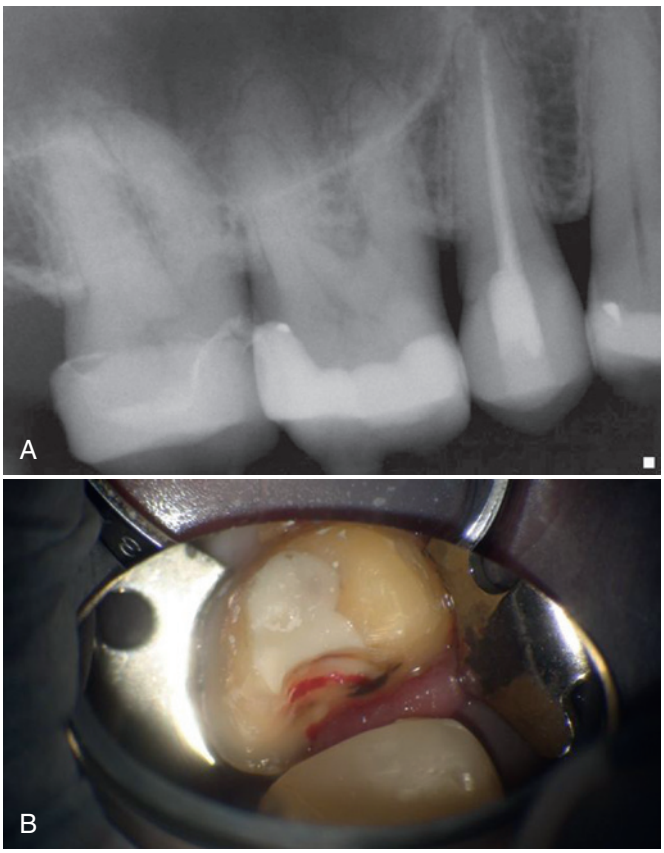

FIG. 26-4 **A,** Recent crown preparation of tooth #2 resulted in tooth ache pain. **B,** Removal of the temporary crown restoration revealed a pulp exposure along the mesial wall.

Recording information at the time of treatment may seem to be unnecessary "busy work," but it could prove to be helpful in identifying the source of a complaint or disease many years later. A patient's recall of dental treatments is usually limited to a few years, but the presence of certain materials or appliances, such as silver points, can sometimes date a procedure. Aging patients' dental histories are rarely complete and may indicate treatment by several clinicians at different locations. These patients are likely to have outlived at least one clinician and been forced to establish a relationship with a new, younger clinician. This new clinician may find dental needs that require an updated treatment plan.

Subjective Symptoms

The examiner can pursue responses to questions about the patient's complaint, the stimulus or irritant that causes pain, the nature of the pain, and its relationship to the stimulus or irritant. This information is most useful in determining whether the source of the pain is pulpal in origin, if the problem is reversible, and whether inflammation or infection has extended to the apical tissues. Thus the clinician can determine what types of tests are necessary to confirm findings or suspicions (see Chapter 1 for further information).

Diagnostic Procedures

It is important to remember that pulpal symptoms are usually chronic in older patients, and other sources of orofacial pain should be ruled out when pain is not soon localized. One example is herpes zoster, which commonly has a prodromal period of 2 to 4 days, when shooting pain, paresthesia, burning, and tenderness appear along the course of the affected nerve (Fig. 26-7). Although it rarely occurs in the maxillary or mandibular divisions of the trigeminal nerve, the viral eruptions may include peripheral nerve endings in the pulp and periodontal ligament (PDL), possibly leading to pulpitis, necrosis, or internal resorption and apical periodontitis.[41,42,80,84,92]

Much information obtained from the complaint, history, and description of subjective symptoms can be gathered in a screening interview by the clinician's assistant or over the phone by the receptionist. The potential need for treatment can be established, and the information obtained can provide a focus for the examination and for the determination of how much chair time will be required.

Objective Signs

The intraoral and extraoral clinical examination provides valuable firsthand information about disease and previous treatment. The overall oral condition should not be overlooked while the patient's chief complaint is evaluated, and all abnormal conditions should be recorded and investigated. Exposures to factors that contribute to oral cancers accumulate with age. Oral premalignant and malignant mucosal lesions require state-of-the-art diagnostic tools to ensure early diagnosis of these lesions.[87] Furthermore, many systemic diseases may initially manifest with prodromal oral signs or symptoms.[21]

Missing teeth contribute to reduced functional ability. The resultant loss of chewing efficiency leads to a higher

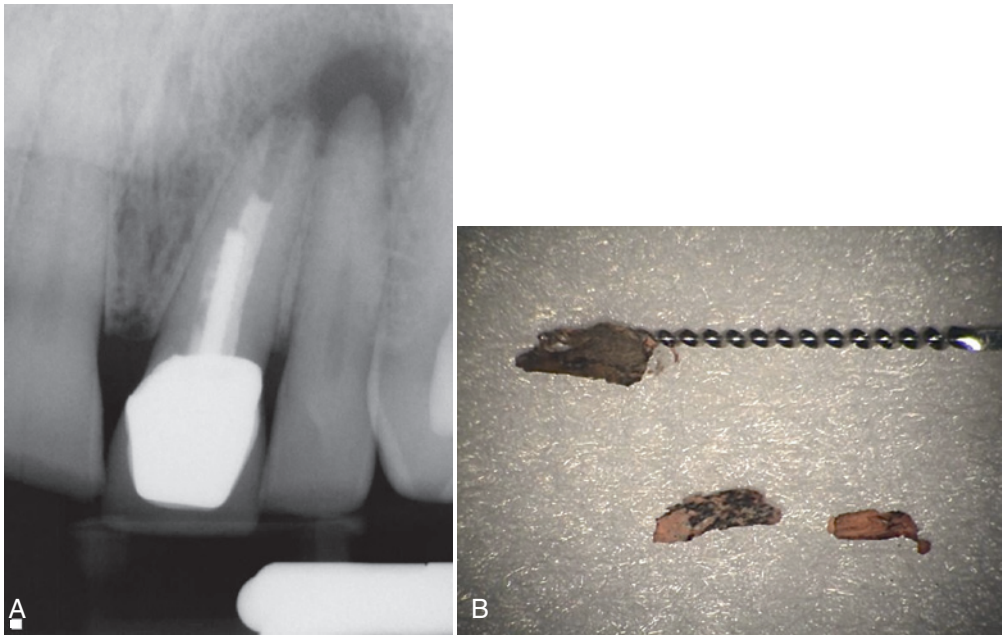

FIG. 26-5 A, This 70-year-old patient recalled having a crown and root canal procedure on this maxillary central incisor 20 years earlier. Teeth #9 and #10 were symptomatic, and radiographs revealed a periapical lesion associated with the root apices; previous root canal treatment of #9 showed an uncleaned and unfilled portion of the apical third of the root canal. B, Endodontic retreatment of #9 revealed that a cotton pellet had been left in the apical portion of the root canal. This contaminated pellet ultimately contributed to the symptomatic apical periodontitis.

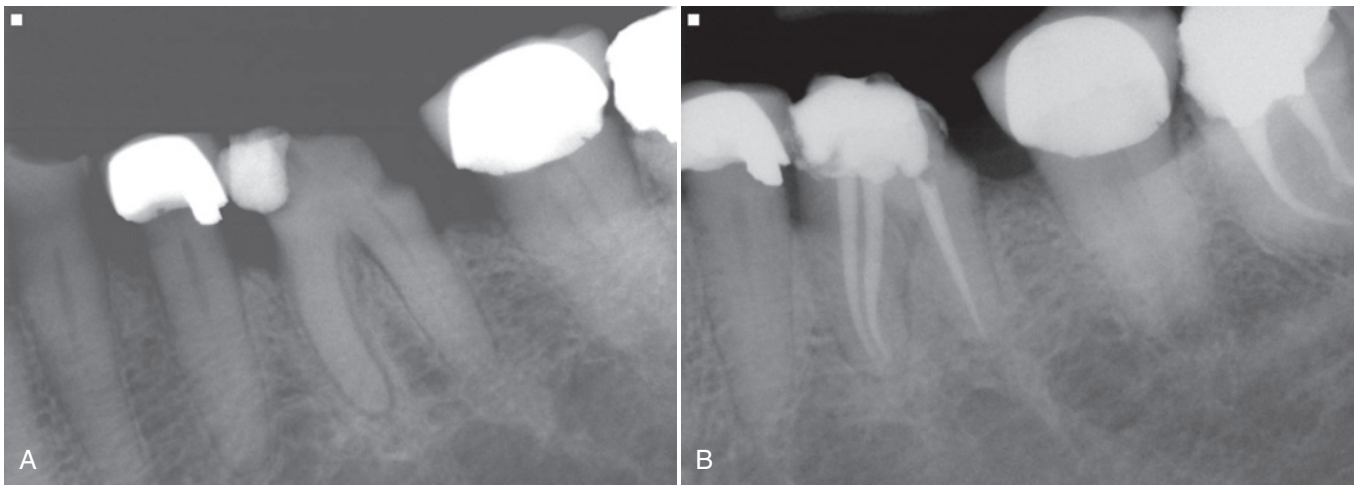

FIG. 26-6 A, Multiple restorations and a missing crown in this 71-year-old patient suggested repeated episodes of caries and restorative procedures, with pulpal effects that accumulated in the form of subclinical inflammation and calcification. B, Limited opening increased the difficulty of performing root canal treatment on calcified tooth #19.

carbohydrate diet of softer, more cariogenic foods. Increased sugar intake to compensate for loss of taste[34,68] and xerostomia[8] are also factors in the renewed susceptibility to decay. Saliva plays a significant role in the maintenance of oral and general health and may be of diagnostic value in evaluating overall health.[39] Aging per se has no significant clinical impact on salivary secretion. The most common cause of salivary hypofunction in the elderly is medication use, and it is most commonly associated with dental caries and oral fungal infections.[71,94]

Gingival recession, which creates sensitivity and is difficult to control, exposes cementum and dentin, which are less resistant to decay than enamel (Fig. 26-8). A clinical study of 600 patients over age 60 showed that 70% had root caries and 100% had some degree of gingival recession.[111] Caries and the use of removable prostheses can synergistically compromise tooth survival in older patients. Patients who wore prostheses and had multiple active carious teeth had the highest risk of losing teeth soon after the decay was treated.[17] The removal of root caries is irritating to the pulp and often results in pulp exposures, reparative dentin formation, or a deep restoration that could affect negotiation of the canal if root canal treatment is later needed (Fig. 26-9).[30] Asymptomatic pulp exposures on

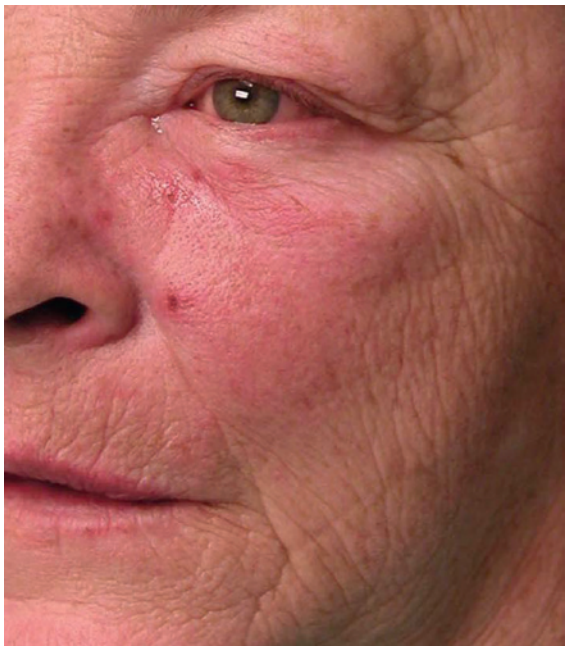

FIG. 26-7 Herpes zoster can cause dental pain and even pulpal involvement. Lesions eventually appeared as a diagnostic finding to confirm shingles.

FIG. 26-8 Missing teeth and the subsequent tilt, rotation, and supraeruption of adjacent and opposing teeth contributed to reduced functional ability and increased susceptibility to caries and periodontal disease.

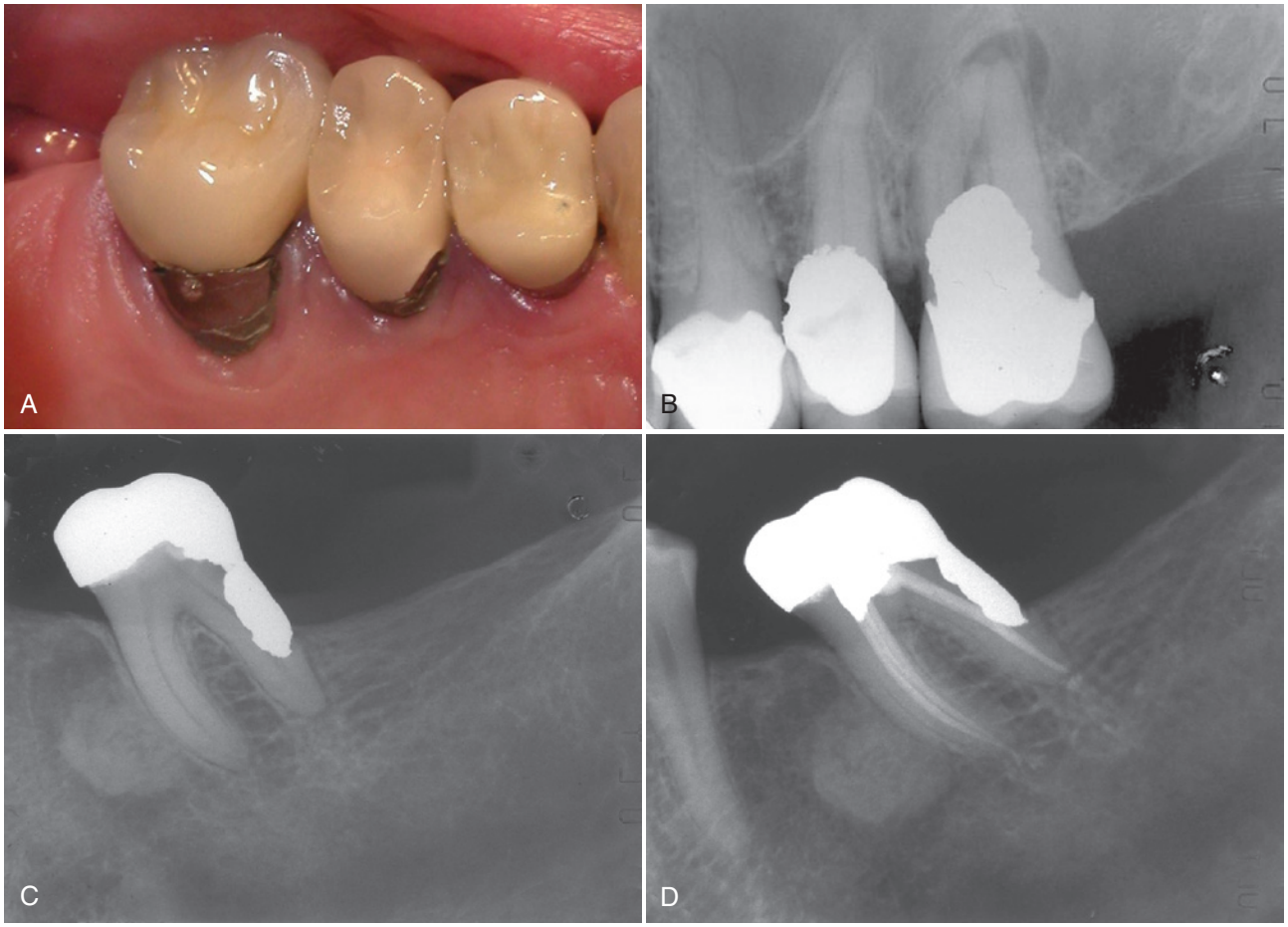

FIG. 26-9 Gingival recession exposed cementum and dentin, which are less resistant to caries. A and B, Restoration on the palatal root surface blocked access to the palatal canal. C and D, Root caries often results in pulp exposures that require endodontic treatment.

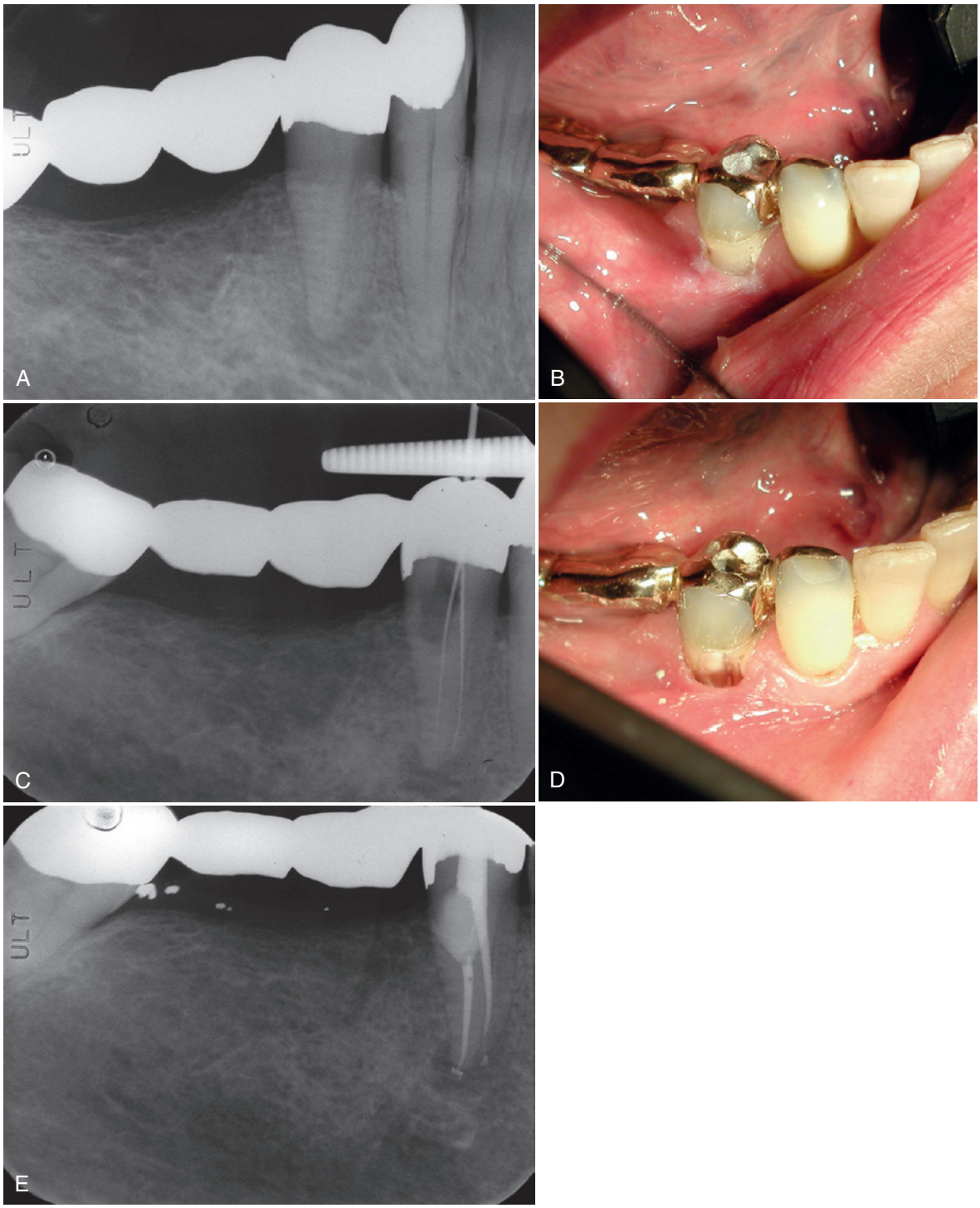

FIG. 26-10 A, Deep root caries that appeared to involve the pulp. B, Mesial abutment for a long fixed bridge. C, Canals were negotiated through the buccal access cavity. D, Buccal canal access. E, Treatment was completed, and the access cavity was permanently restored.

one root surface of a multirooted tooth can result in the uncommon clinical situation of the presence of both vital and nonvital pulp tissue in the same tooth (Fig. 26-10).

Interproximal root caries is difficult to restore, and restoration failure as a result of continued decay is common (Fig. 26-11). Although the microbiology of diseases is not substantially different in different age groups, the altered host response during aging may modify the progression of these diseases.[102]

Attrition (see Fig. 26-11), abrasion, and erosion (Fig. 26-12) also expose dentin through a slower process that allows the pulp to respond with dentinal sclerosis and reparative dentin.[95] Secondary dentin formation occurs throughout life and may eventually result in almost complete pulp obliteration. In maxillary anterior teeth, the secondary dentin is formed on the lingual wall of the pulp chamber[78]; in molar teeth, the greatest deposition occurs on the floor of the chamber.[13] Although this

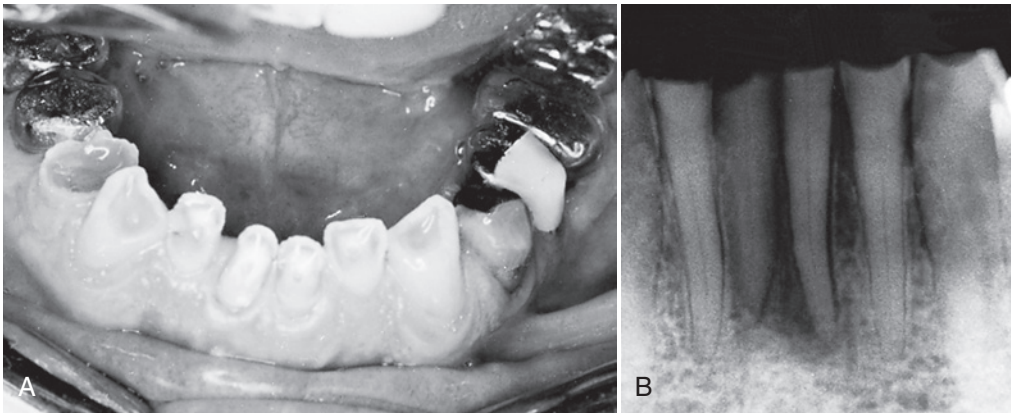

FIG. 26-11 A, Attrition exposes dentin through a slow process that allows the pulp to respond with reparative dentin, but pulp exposure eventually becomes clinically evident (B).

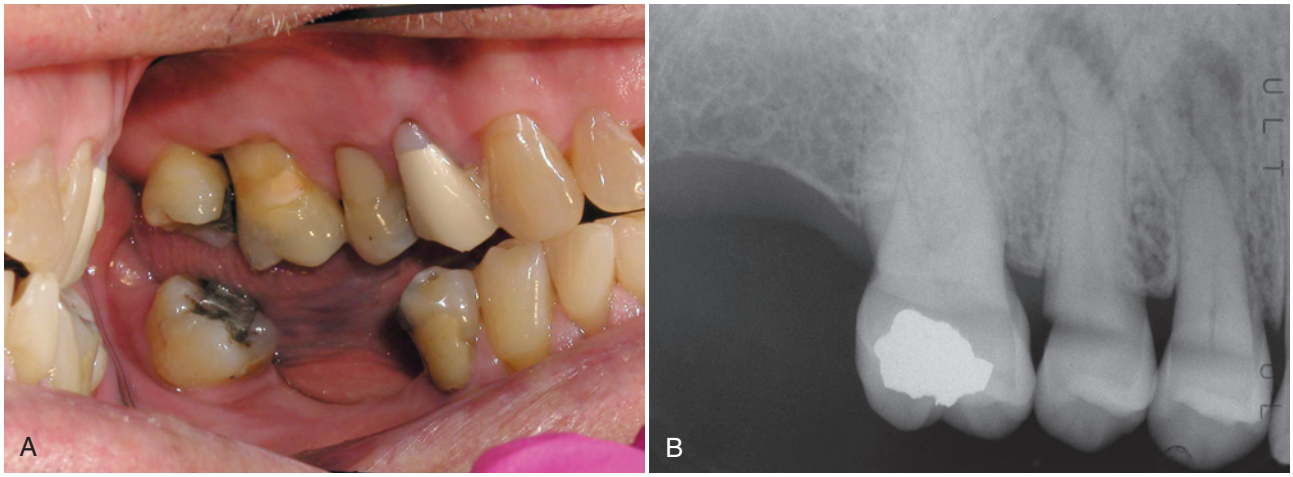

FIG. 26-12 Gingival recession also exposes cementum and dentin, which are less resistant to abrasion and erosion. This can result in exposure of the pulp or require restorative procedures that could cause pulp irritation.

pulp may appear to recede, small pulpal remnants may remain or leave a less calcific, more permeable, tract that may lead to pulp exposure.

In general, canal and chamber volume is inversely proportional to age: as age increases, canal size decreases (Fig. 26-13). Reparative dentin resulting from restorative procedures, trauma, attrition, and recurrent caries also contributes to diminution of canal and chamber size. In addition, the cementodentinal junction (CDJ) moves farther from the radiographic apex with continued cementum deposition (Fig. 26-14).[57] The thickness of young apical cementum[85] is 100 to 200 µm, and it increases with age to two or three times that thickness.[116]

The calcification process associated with aging appears clinically to be of a more linear type than that which occurs in a younger tooth in response to caries, pulpotomy, or trauma (Fig. 26-15). Dentinal tubules become more occluded with advancing age, reducing tubular permeability.[67] Lateral and accessory canals can calcify, thus decreasing their clinical significance.

The compensating bite produced by missing and tilted teeth (or attrition) can cause temporomandibular joint (TMJ) dysfunction (less common in older adults) or loss of vertical dimension. Diminished eruptive forces occur with age, reducing the amount of mesial drift and supraeruption.[86] Any limitation in opening reduces available working time and the space needed for instrumentation.

The presence of multiple restorations indicates a history of repeated insults and an accumulation of irritants. Marginal leakage and microbial contamination of cavity walls are a major cause of pulpal injury.[15] Violating principles of cavity design combined with the loss of resiliency that results from a reduced organic component to the dentin can increase susceptibility to cracks and cuspal fractures. In any further restorative procedures on such teeth, the clinician should consider the effect on the pulp and the effect on gaining access to and negotiating canals through such restorations if root canal therapy is indicated later.

Many cracks or craze lines (see Fig. 26-12) may be evident as a result of staining, but they do not indicate dentin penetration or pulp exposure.[18,50] Pulp exposures caused by cracks are less likely to present acute problems in older patients and often penetrate the sulcus to create a periodontal defect in addition to a periapical one. If incomplete cracks are not detected early, the prognosis for cracked teeth with necrosis is questionable (Fig. 26-16).[10]

Periodontal disease may be the principal problem for dentate seniors.[25] The relationship between pulpal and

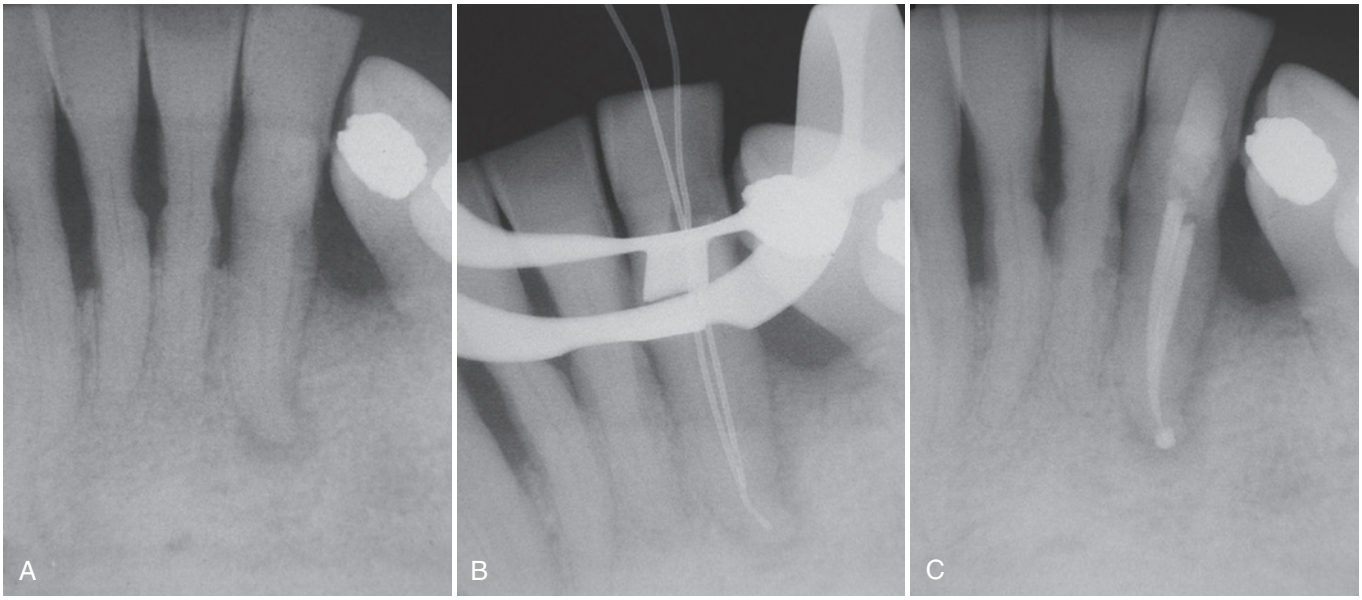

FIG. 26-13 A, In general, canal chamber volume is inversely proportional to age. As age increases, canal and chamber volume decreases, as seen in these lower incisor teeth in a 90-year-old patient. B, After both canals in tooth #23 were instrumented, final disinfection was performed. Two 30-gauge side-vented irrigation needles are seen here inserted to working length. C, Treatment was completed for these calcified canals, and obturation was performed using GuttaCore.

FIG. 26-14 Radiographic image of tooth #15 in an older individual shows increased cementum deposition with age.

FIG. 26-15 This radiograph illustrates pulp volume changes in mandibular premolars. Left to right, A normal pulp; reparative dentin adjacent to a shallow restoration; more extensive reparative dentin adjacent to a more extensive restoration; and the overall reduced volume that occurs during aging.

periodontal disease can be expected to be more significant with age. Retention of teeth alone demonstrates some resistance to periodontal disease. The increase in disease prevalence is largely attributable to an increase in the proportional size of the population who have retained their teeth.[61] The periodontal tissues must be considered a pathway for sinus tracts (Fig. 26-17). Narrow, bony-walled pockets associated with nonvital pulps are usually sinus tracts, but they can be resistant to root canal therapy alone when, with time, they become chronic periodontal pockets. Patients with diabetes have increased periodontal disease in endodontically treated teeth and have a reduced likelihood of success of endodontic treatment in cases with preoperative periradicular lesions.[33]

Periodontal treatment can produce root sensitivity, disease, and pulp death.[60] In developing a successful treatment plan, it is important that the clinician determine the effects of periodontal disease and its treatment on the pulp (Fig. 26-18). Age was not a factor in response to periodontal therapy for advanced periodontal tissue breakdown.[59] The mere increase in incidence and severity of periodontal disease with age increases the need for combined therapy. The chronic nature of pulp disease demonstrated with sinus tracts can often be manifested in a periodontal pocket. Root canal treatment is commonly indicated before root amputations are performed. With age, the size and number of apical and accessory foramina are actually reduced, which reduces these foramina's function as pathways

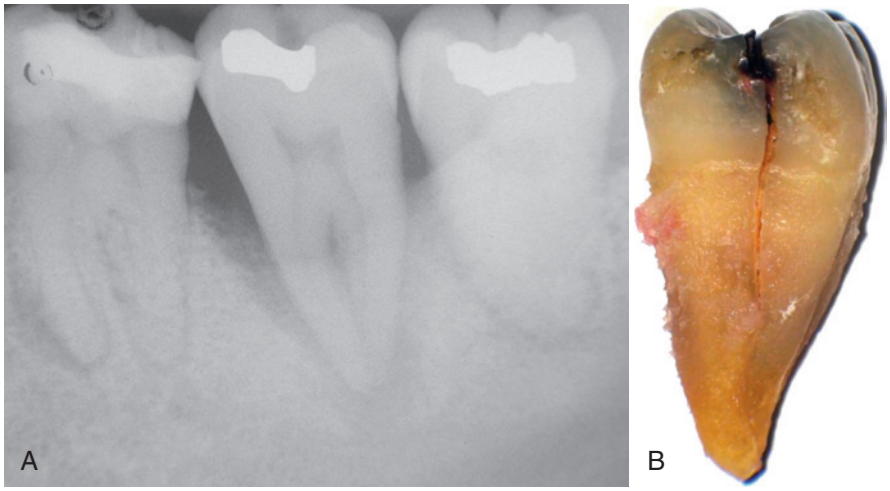

FIG. 26-16 A split tooth should always be considered when a nonvital pulp and chronic apical periodontitis are evident on a minimally restored or nonrestored tooth. The crack usually extends into the periodontium, and the prognosis generally is hopeless.

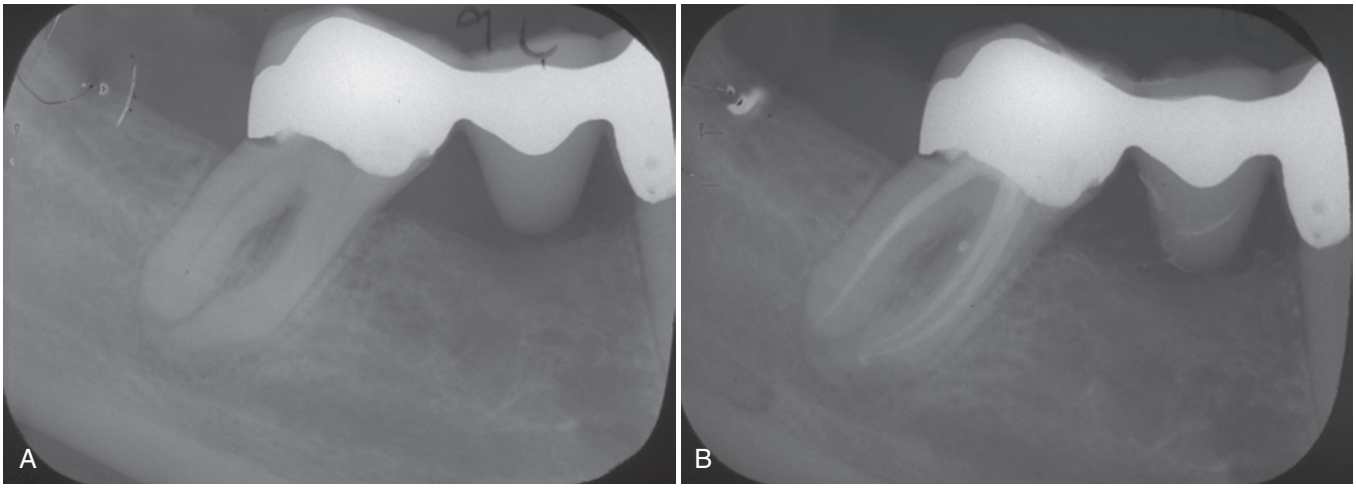

FIG. 26-17 A, A deep periodontal pocket is probed in the buccal furcation on this mandibular molar abutment. B, This could indicate pulpal or periodontal disease, or both. Root canal treatment resulted in reduction of the pocket depth, confirming the pulpal cause of this periradicular lesion.

of communication.[83] The permeability of dentinal tubules also is reduced.

Examination of sinus tracts should include tracing with gutta-percha cones to establish the tracts' origins (Fig. 26-19). Sinus tracts may have long clinical histories and usually indicate the presence of chronic periapical inflammation. Their disappearance after treatment is an excellent indicator of healing. The presence of a sinus tract reduces the risk of interappointment or postoperative pain, although drainage may follow canal debridement or filling.

Pulp Testing

Information collected from the patient's complaint, history, and examination may be adequate to establish pulp vitality and direct the clinician toward the techniques that are most useful in determining which tooth or teeth are the object of the complaint. During pulp testing, it is often very difficult to quantify the response to a stimulus applied to a tooth; age can also be a factor in sensitivity testing.[72] Slow and gentle testing should be done to determine pulp and periapical status and whether palliative or definitive therapy is indicated. Vitality responses must correlate with clinical and radiographic findings and must be interpreted as a supplement in developing clinical judgment. (Techniques for clinical pulp testing procedures are discussed in Chapter 1.)

Transilluminating[79] and staining have been advocated as means to detect cracks, but the presence of cracks is of little significance in the absence of complaints because most older teeth, especially molars, demonstrate some cracks. Vertically cracked teeth should always be considered a pathway for bacteria when pulpal or periapical disease is observed, especially where there is little or no cause for pulpal irritation observed clinically or radiographically. The high magnification available with microscopes during access opening and canal exploration permits visualization of the extent of cracks in determining the prognosis. Abbot[1] showed that 60% of all teeth requiring endodontic treatment demonstrated cracks after the complete removal of all restorations.

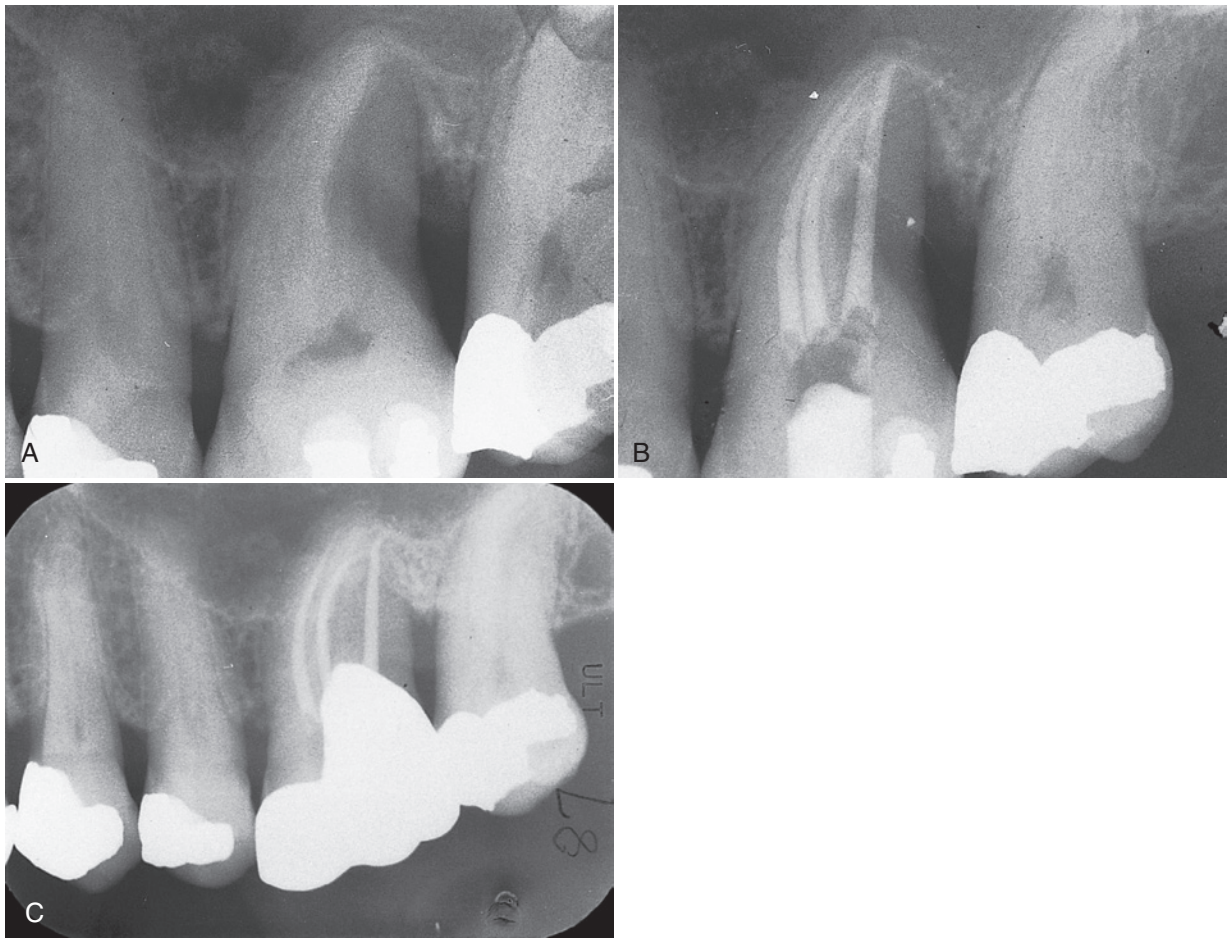

FIG. 26-18 A, Unexpected root amputation on vital pulps may occur during periodontal surgery. This requires root canal treatment on the remaining roots (B) and a crown with good marginal adaptation (C).

Cracks that are detected while the pulp is still vital can offer a reasonable prognosis if immediately restored with full cuspal coverage.[56] The chronic nature of any periapical pathologic condition caused by vertically cracked teeth indicates that it is longstanding, and the prognosis is questionable[10] (even when pocket depths appear normal). Periodontal pockets associated with cracks indicate a poor prognosis.[46]

The reduced neural and vascular components of aged pulps,[12] the overall reduced pulp volume, and the change in character of the ground substance[96] create an environment that responds differently to both stimuli and irritants than does that of younger pulps (Fig. 26-20). Arteriosclerosis, a common condition in older people, has not been shown to occur in the pulp.[55]

Fewer nerve branches are present in older pulps. This may be due to retrogressive changes resulting from mineralization of the nerve and nerve sheath (Fig. 26-21).[11] Consequently, the response to stimuli may be weaker than in the more highly innervated younger pulp.

No correlation exists between the degree of response to electric pulp testing and the degree of inflammation. The presence or absence of response is of limited value and must be correlated with other tests, examination findings, and radiographs.[72] Extensive restorations, pulp recession, and excessive calcifications are limitations in both performing and interpreting results of electric and thermal pulp testing. An alternative to the electric pulp test is assessment of pulp vitality by applying a thermal stimulus to the tooth surface. The electric pulp tester, CO_2 snow, and difluorodichloromethane were found to be more reliable than ethyl chloride or ice in producing a positive response.[37] Attachments that reduce the amount of surface contact necessary to conduct the electric stimulus are available (SybronEndo, Orange, California), and bridging the tip to a small area of tooth structure with an explorer has been suggested.[76] Use of even this small electric stimulus in patients with pacemakers[113] is not recommended; any such risk would outweigh the benefit. The same caution holds true for electrosurgical units.

A test cavity is generally less useful as the test of last resort because of reduced dentin innervation. Vital pulps can sometimes be exposed and even negotiated with a file with minimal pain (Fig. 26-22); then the root canal treatment becomes part of the diagnostic procedure. Test cavities should be used only when other findings are suggestive but not conclusive.

Diffuse pain of vague origin is also uncommon in older pulps and limits the need for selective anesthesia. Pulpal disease is progressive and produces diagnostic signs or symptoms in a relatively short time. Nonodontogenic sources should be considered when factors associated with pulpal disease are not readily identified or when acute pain does not localize within a short time.

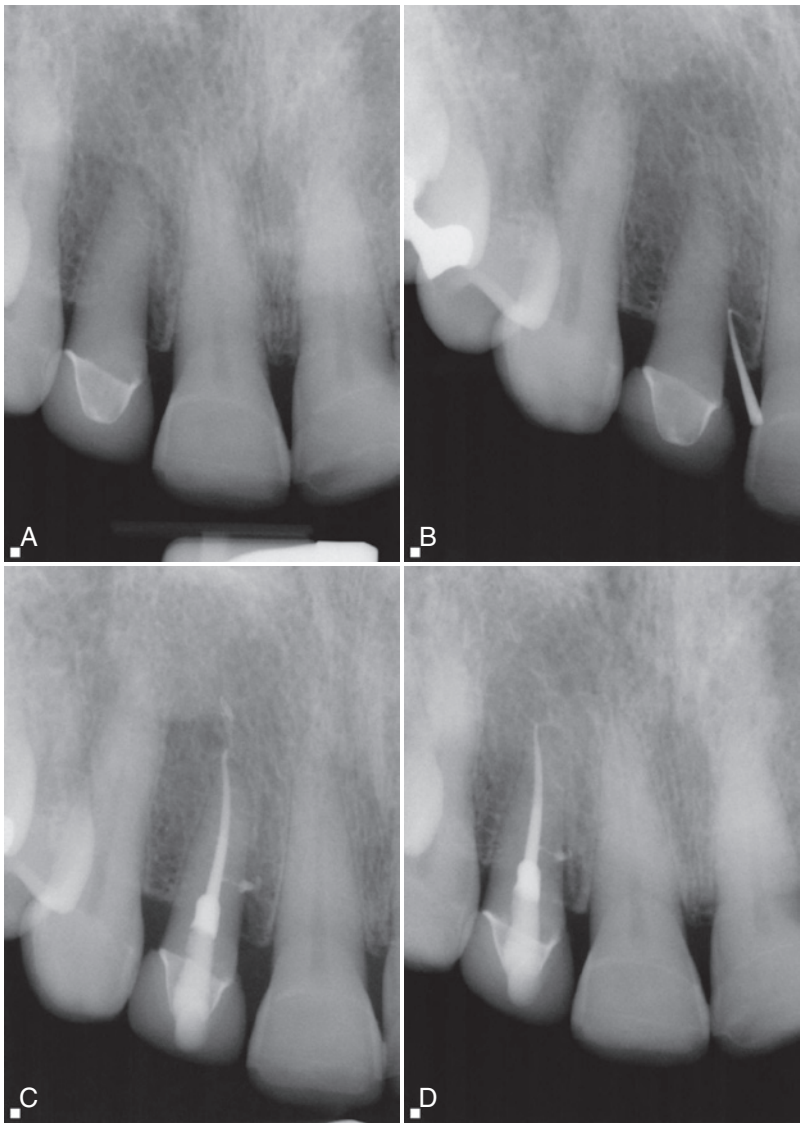

FIG. 26-19 A, This radiograph shows that tooth #7 has a well-fitting crown, but it also has periradicular inflammation along the mesial root surface. B, The origin of a sinus tract is traced with a gutta-percha point. C, Very challenging root canal treatment saved this valuable tooth. D, At the 6-month recall examination, the sinus tract was healing, and the patient was asymptomatic.

FIG. 26-20 The cellularity of older pulp tissue gradually decreases. Odontoblasts decrease in size and number and may disappear altogether in certain areas, particularly on the pulpal floor in multirooted teeth.

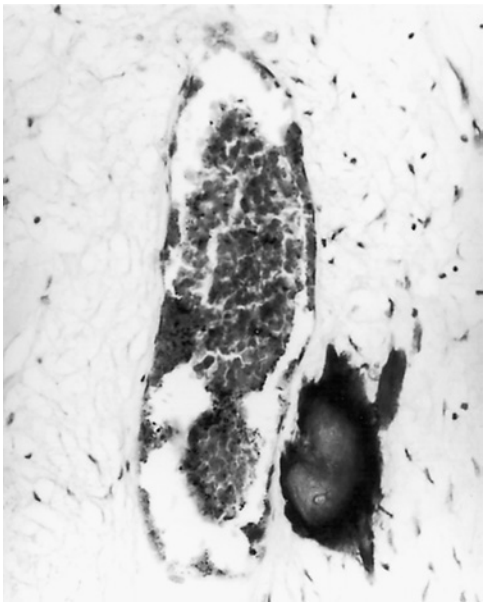

FIG. 26-21 Fibrosis appears to occur along the pathways of degenerated vessels and may serve as foci for pulpal calcification.

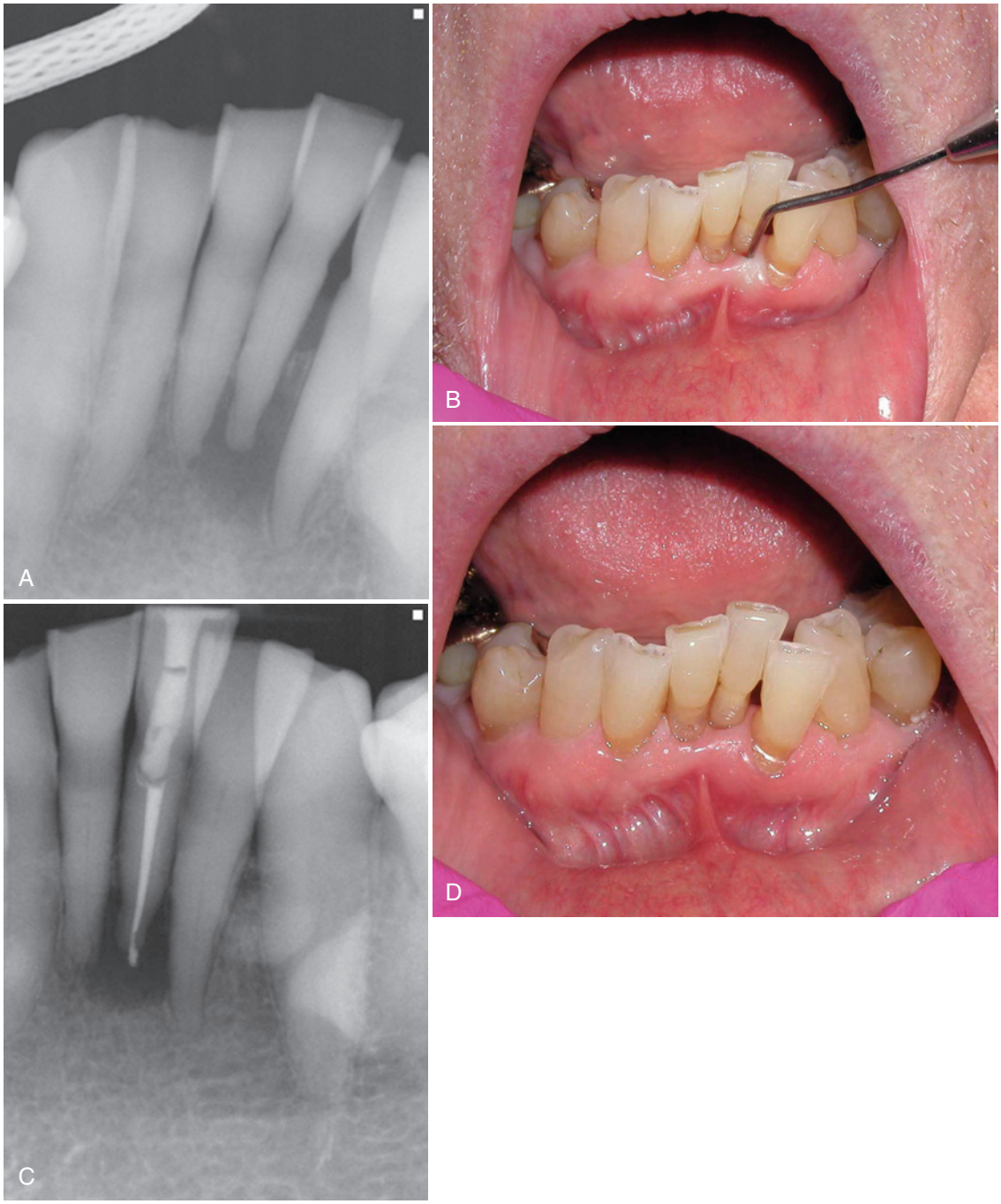

FIG. 26-22 A and B, Vital pulp testing was inconclusive for this 91-year-old patient. C and D, Treatment was performed without local anesthesia to confirm pulp status.

Discoloration of single teeth may indicate pulp death, but this is a less likely cause of discoloration with advanced age. Dentin thickness is greater, and the tubules are less permeable to blood or breakdown products from the pulp. Dentin deposition produces a yellow, opaque color that would indicate progressive calcification in a younger pulp, but this is common in older teeth.

Radiographs

Indications and techniques for taking radiographs do not differ much among adult age groups. However, several physiologic and anatomic changes can significantly affect their interpretation. Film placement may be adversely affected by tori but can be assisted by the apical position of muscle attachments that increase the depth of the vestibule. Older patients may be less capable of assisting in film placement, and holders that secure the position should be considered. The presence of tori, exostoses, and denser bone (Fig. 26-23) may require increased exposure times for proper diagnostic contrast. The subjective nature of interpretation can be reduced with correct processing, proper illumination, and magnification.

The periapical area must be included in the diagnostic radiograph, which should be studied from the crown toward the apex. Angled radiographs should be ordered only if the

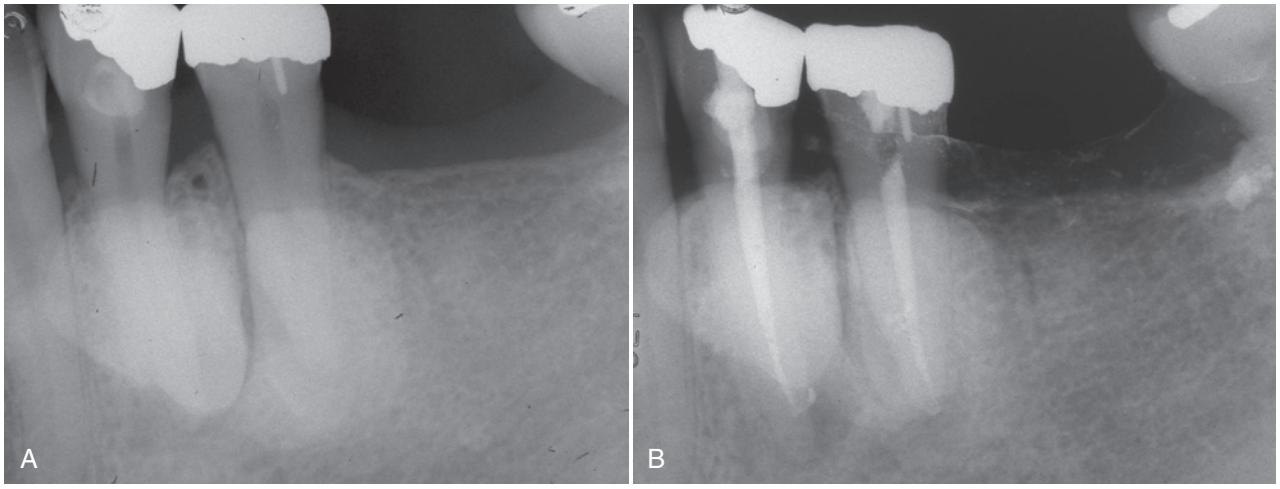

FIG. 26-23 Mandibular tori can make periapical radiography challenging. Such dense bone may indicate the need for increased exposure times to improve the contrast needed to see the canal and root anatomy.

original diagnostic radiograph suggests that more information is needed for diagnosis or to determine the degree of treatment difficulty. Digital radiography may be more useful than conventional radiography in detecting early bone changes.[115] Cone beam computed tomography (CBCT) is a very promising adjunct for a more accurate assessment of changes in the root supporting structures.[20,28,108]

In older patients, pulp recession is accelerated by reparative dentin and complicated by pulp stones and dystrophic calcification. Deep proximal or root decay and restorations may cause calcification between the observable chamber and root canal.

The depth of the chamber should be measured from the occlusal surface and its mesiodistal position noted. Receding pulp horns that are apparent on a radiograph may remain microscopically much higher. Deep restorations or extensive occlusal crown reduction may produce pulp exposures that were not expected. The axial inclinations of crowns may not correlate with the clinical observation when tilted teeth have been crowned or become abutments for fixed or removable appliances. Access to the root canals is the most limiting condition in root canal treatment of older patients.

Canals should be examined for their number, size, shape, and curvature. Pulp has been demonstrated histologically even when not visible on radiographs, but in general, the two measurements did not differ even when pulp calcifications were present.[58] Comparisons with adjacent teeth should be made. Small canals are the rule in older patients. Midroot disappearance of a detectable canal may indicate bifurcation rather than calcification. Canals calcify evenly throughout their length unless an irritant (e.g., caries, restoration, cervical abrasion) has separated the chamber from the root canal. Root-end fillings during root-end resection (more common during retreatment of older patients) indicate missed canals and roots as a common cause of failure.[2]

The lamina dura should be examined in its entirety and anatomic landmarks distinguished from periapical radiolucencies and radiopacities. The incidence of some odontogenic and nonodontogenic cysts and tumors characteristically increases with age, and this should be considered when vitality tests do not correlate with radiographic findings. However, the incidence of osteosclerosis and condensing osteitis decreases with age.

Resorption associated with chronic apical periodontitis may significantly alter the shape of the apex and the anatomy of the foramen through inflammatory osteoclastic activity (Fig. 26-24).[23] The narrowest point in the canal may be difficult to determine; it is positioned farther from the radiographic apex because of continued cementum deposition.

A continued normal rate of cementum formation may be demonstrated by a canal or foramen that appears to end or exit short of the radiographic apex, and hypercementosis (Fig. 26-25) may completely obscure the apical anatomy.

DIAGNOSIS AND TREATMENT PLAN

A clinical classification that accurately reflects the histologic status of the pulp and periapical tissues is not possible and not necessary beyond determining whether root canal treatment is indicated. A clinical judgment can be made based on the patient's complaint, history, signs, symptoms, testing, and radiographs as to the vitality of the pulp and the presence or absence of periapical pathologic conditions. This classification has not been shown to be a factor in predicting success, interappointment or postoperative pain, or the number of visits necessary to complete treatment when the objectives of cleaning, shaping, and filling are clearly understood and consistently met.[35] Of great clinical significance in treatment procedures is the assessment of pulp status to determine the depth of anesthesia necessary to perform the treatment comfortably.

One-appointment procedures offer obvious advantages to older patients. No detectable difference was found in the effectiveness of root canal treatment in terms of radiologic success between single and multiple visits.[32] The length of a dental appointment does not usually cause inconvenience, as may more numerous appointments, especially if a patient must rely on another person for transportation or needs physical assistance to get into the office or operatory.

Root canal treatment as a restorative expediency on teeth with normal pulps must be considered when cusps have fractured or when supraerupted or malaligned teeth, intracoronal

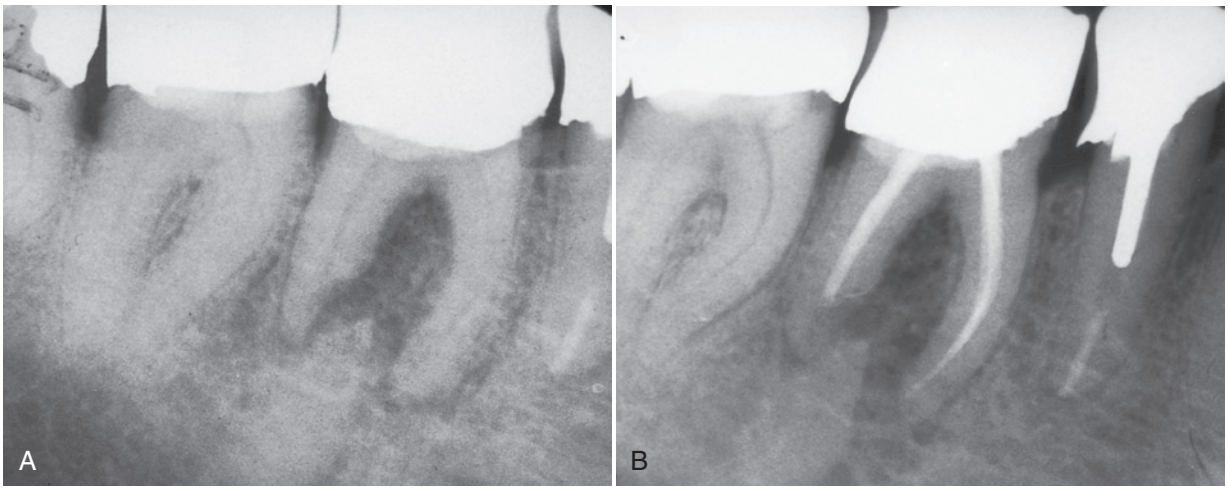

FIG. 26-24 A, Resorption associated with chronic apical periodontitis may alter the shape and position of the foramen through osteoclastic activity. B, The narrowest point in the canal is now positioned farther from the radiographic apex.

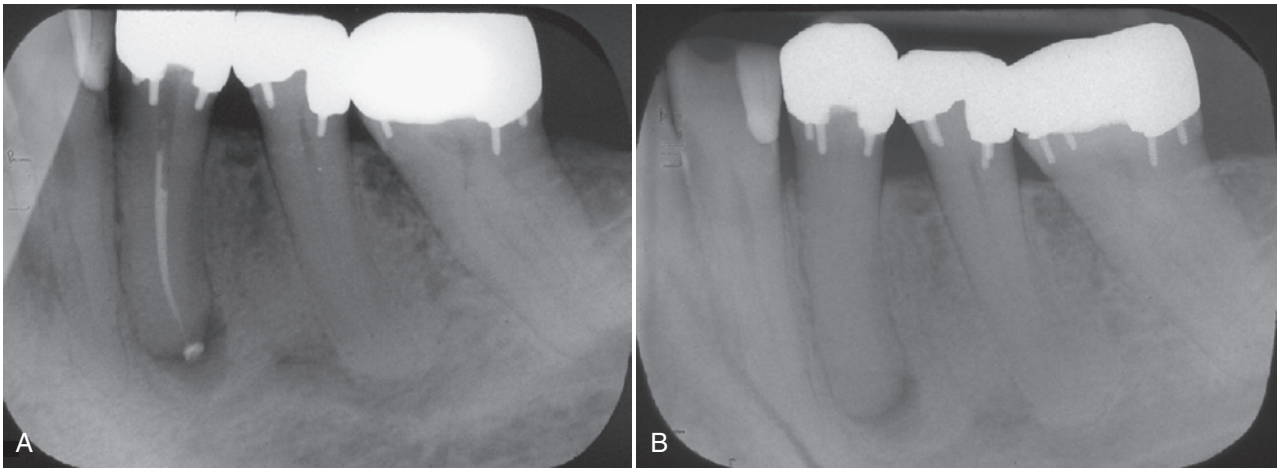

FIG. 26-25 A, Hypercementosis may completely obscure the apical anatomy and result in a constriction farther from the radiographic apex (B).

attachments, guide planes for partial abutments, rest seats, or overdentures require significant tooth reduction (Fig. 26-26). Predicting the need for future root canal treatment and a clinician's ability to perform treatment later are even more important because the risk of losing the restoration during later access preparation increases with the thickness of the restoration and the reduction in canal size (Fig. 26-27). Because of a reduced blood supply, pulp capping is not as successful in older teeth as in younger ones, so it is not recommended. Any risk to the patient's future health and the effect health may have on his or her ability to withstand future procedures should also be considered. Endodontic surgery at a later time is not as viable an alternative as for a younger patient.

Consultation and Consent

Good communication should be established and maintained with all patients, regardless of whether they are physically impaired or unable to make treatment decisions for themselves. Relatives or trusted friends should be included in consultations if their judgment is valued by the patient or needed for consent, but the clinician should direct the discussion toward the patient (Fig. 26-28).

Clinicians should explain procedures in a way that is completely comprehensible and provides an opportunity for questions from the patient. Patient-friendly pamphlets are available from the American Association of Endodontists (AAE) that thoroughly explain and illustrate most endodontic procedures. A signed consent to outlined treatment is required and may be especially useful if the patient is forgetful or feels challenged by his or her understanding of the technology of current communication techniques.

Determining the patient's desires is as important as determining his or her needs, and it is required in obtaining informed consent (Fig. 26-29). Priorities in treating pain and infection to properly and aesthetically restore teeth to health and function should be unaffected by age (Fig. 26-30). A patient's limited life expectancy should not appreciably alter treatment plans and is no excuse for extractions or poor root canal treatment. It is important that all older patients be well informed of risks and alternatives (Fig. 26-31, *A*). Patients deserve a

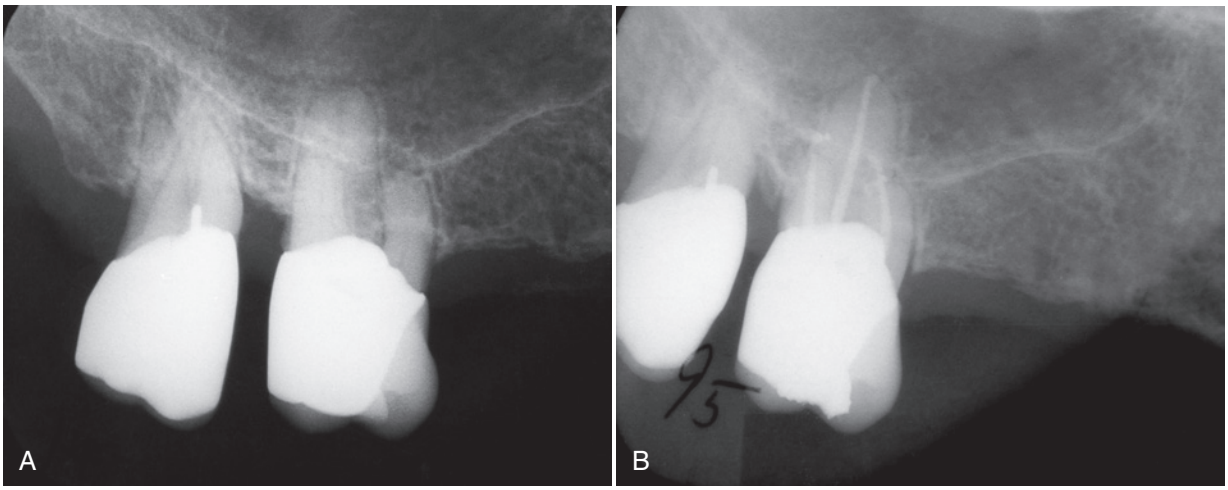

FIG. 26-26 A, Valuable maxillary first- and second-molar abutments in an 85-year-old patient. B, Later need for endodontic therapy was made very challenging because of access abutments.

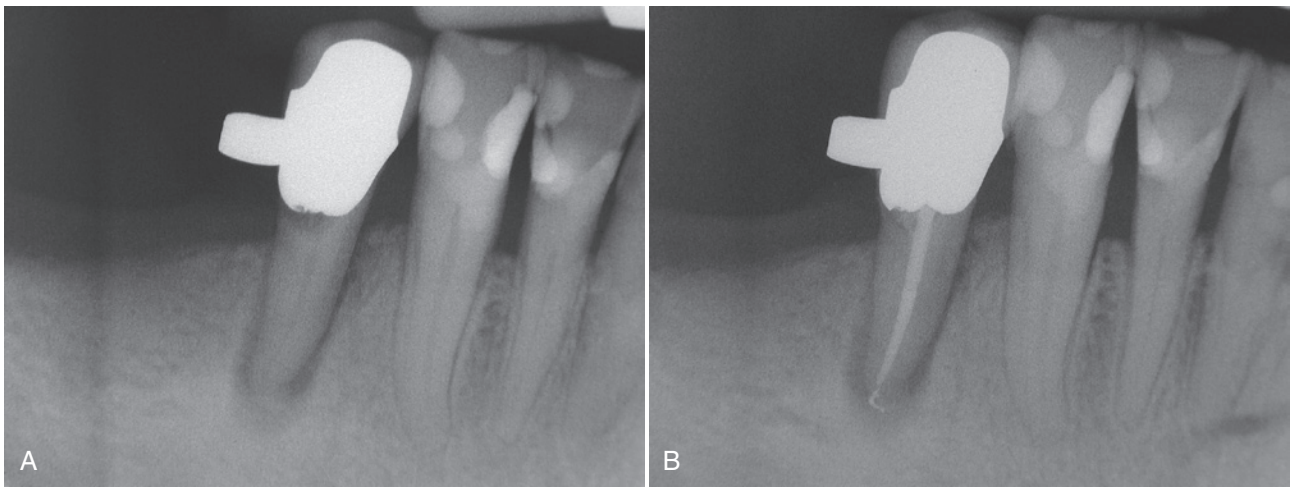

FIG. 26-27 A, Intracoronal attachments, guide planes, and rest seats for partial abutments require significant tooth reduction and a bulk of metal that makes endodontic treatment more likely and difficult. This patient was referred because the root canal could not be located. B, Successful treatment and preservation of the restoration when the canal is small are challenges to the most skilled clinician. An intracoronal post and perforation repair was done for this compromised tooth.

better explanation for their disease than that it is caused by age. When a physician told one patient that the problem with his knee was due to age, the patient replied, "The other knee is the same age, and nothing is wrong with it."

Acceptance of the eventual loss of teeth has been replaced by an understanding of the psychological and functional importance of maintaining dentition. Older patients whose lives have been changed for the worse by a previous surgical procedure may readily recognize the implications of the loss of teeth. Although older patients prefer to save their natural teeth, the retention rate of restored implants compares favorably to restored root canal–filled teeth.[3,48] The implant-retained mandibular overdenture is often the most desired choice for an edentulous mandible (Fig. 26-31, *B*).[22]

The capability of the clinician and the availability of endodontic specialists should also be considered. The obligation to offer root canal treatment as an alternative to extraction may exceed a clinician's ability to perform the procedure efficiently or successfully, and referral may be indicated. Older patients are more likely to be aware that specialists are available who can perform root canal surgery, but patients usually trust and do what their clinician recommends. In advance of the scheduled visit, the referring clinician should provide the endodontist with as much information about the patient as possible (and thoroughly explain the reason for referral to the patient). To support treatment adherence and understanding, the clinician should consider making the referral appointment for the patient while the person is in the dental office and follow up to determine compliance.

Obtaining informed consent before dental procedures usually is not a problem. However, if a patient is medically compromised or cognitively impaired, it may be difficult to acquire valid informed consent. Neuropsychiatric impairment may result in gross manifestations and indicate a reduced level of competency. Physicians or mental health experts should be consulted as needed, and no elective procedures should be

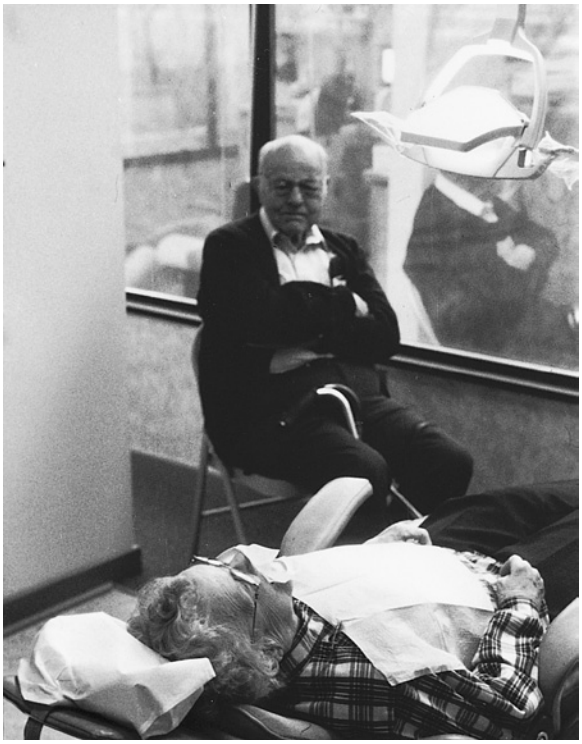

FIG. 26-28 Some patients may request or require that a family member be present during consultation or treatment.

FIG. 26-29 Many geriatric patients benefit from the use of a pillow for neck support.

performed until valid consent is established. Fortunately, acute pulpal and periapical episodes in which immediate treatment is indicated for pain or infection are less common than the low-grade signs and symptoms of chronic disease.

TREATMENT

Most geriatric patients who need and demand endodontic therapy are ambulatory and not institutionalized. Institutionalized and nonambulatory patients require clinicians trained in those environments and facilities designed for access to dental health care. Such access is a benefit required in most

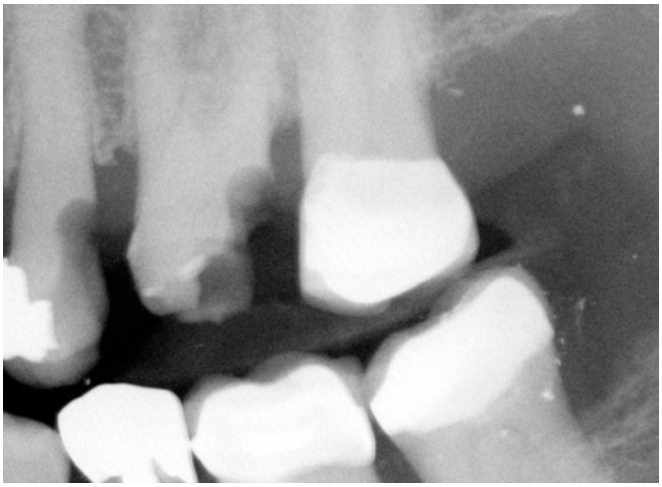

FIG. 26-30 This 86-year-old patient was pleased to find that all these broken-down teeth could be saved with endodontic therapy and crowns. She was eager to make the investment in time and money, despite her limited life expectancy. Her concern focused on why these procedures had not been recommended by her previous dentist, whom she visited regularly.

institutions, but alternatives to root canal therapy may be the only services available. Extended health care facilities are now required to have clinicians on staff before Medicare certification can be obtained. The dental office building, including both the interior design and the exterior approach, must be able to accommodate people with special needs (Fig. 26-32).[75] Access for those who use ambulation aids (e.g., canes, walkers, wheelchairs) should include comfort and safety in the parking lot, reception room, operatory, and restroom. Dental units can be designed to accommodate patients who remain in their own wheelchairs so that they do not require transfer.[101]

A physical and mental evaluation of the patient should determine the ideal time of day and length of time necessary to schedule treatment. A patient's daily personal, eating, and resting habits should be considered, in addition to any medication schedule. Morning appointments are preferable for some older patients, and their comfort, which varies with the procedure indicated, dictates the length of the appointment. Some patients prefer late morning or early afternoon visits to allow "morning stiffness" to dissipate.[29]

Older patients are more likely to tolerate long appointments, although chair positioning and comfort may be more important for older adults than for younger patients. Patients should be offered assistance into the operatory and into or out of the chair, and chair adjustments should be made slowly (and the ideal position established and used if possible). Pillows should be offered, as should assistance in positioning them comfortably. Every effort should be made to accommodate the ideal position, even at some expense to the clinician's comfort or access.

The patient's eyes should be shielded from the intensity of the clinician's light. If the temperature selected for the comfort of the office staff is too cool for the patient, a blanket should be available. As much work as possible should be performed at each visit, and a restroom break should be offered at intervals as the patient's needs indicate. Jaw fatigue is readily recognizable and may be the most limiting factor in a long procedure, requiring periods of rest; however, once such

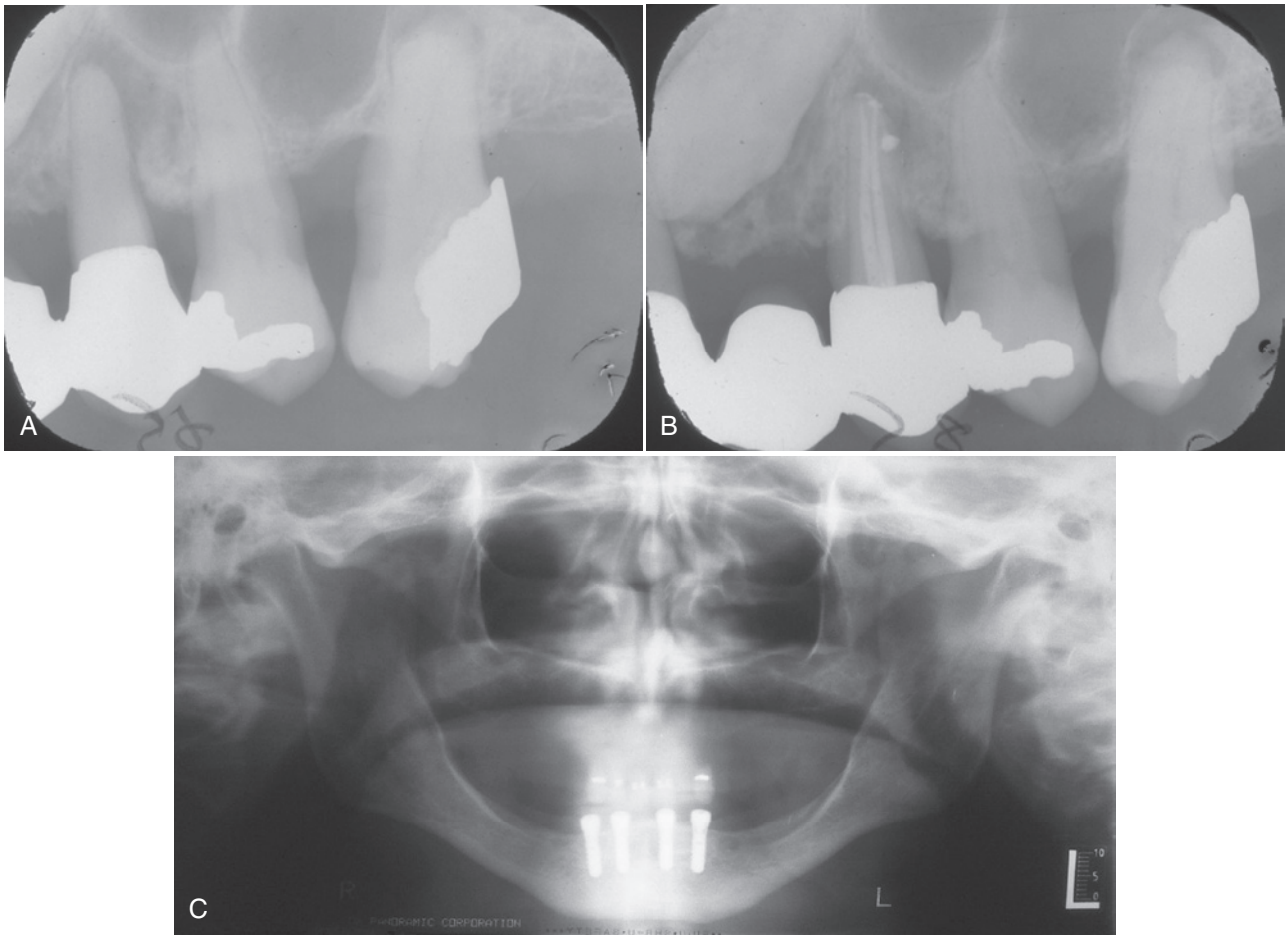

FIG. 26-31 A, Preoperative radiograph showing the calcified canals of this premolar abutment tooth. B, Postoperative radiograph after root canal treatment of the two-canaled premolar tooth, showing conservation of tooth structure. C, A 97-year-old man who had implant placement for mandibular denture retention. He tolerated the procedure well.

FIG. 26-32 A, The office design should consider access for wheelchairs and walkers. B, Staff members should be experienced in assisting patients into and out of the dental chair and operatory.

fatigue is evident, the procedure should be terminated as soon as possible. Bite blocks are useful for comfortably maintaining freeway space and reducing jaw fatigue.

Retired individuals may rely on rides from friends or relatives or public transportation to get to the dental office. Although they often arrive early for appointments, most older patients are in no hurry once they arrive. Enough time should be scheduled to allow some social exchange, and a sincere personal interest should be demonstrated before proceeding. Endodontic specialists need to consider separate consultations for making this initial social contact and evaluating the degree of difficulty that will determine the chair time needed to perform treatment. Staff should allow patients to initiate handshakes, which may be very uncomfortable for people with arthritic joints. From a behavioral and management standpoint, geriatric patients are among the most cooperative, available, and appreciative.

Older, medically compromised patients are at no more risk of complications than those in any other age group, and the chronicity of their diseases can alert the clinician to necessary precautions. Once again, clinicians should recognize that root canal therapy is far less traumatic than extraction for older patients.

The pulp vitality status and the cervical positioning of the rubber dam clamp determine the need for anesthesia. Older patients more readily accept treatment without anesthesia, and sometimes they must be persuaded that anesthesia is necessary for root canal treatment if their routine operative procedures have been performed without it. Generally, older patients demonstrate less anxiety about dental treatment and may have previous experience with root canal treatment. Therefore, anxiolytic medication is rarely necessary.

The cutting of dentin does not produce the same level of response in an older patient for the same reason that a test cavity is not as revealing during examination. The number of low-threshold, high-conduction-velocity nerve endings in dentin is reduced or absent, and the nerve endings do not extend as far into the dentin. In addition, the dentinal tubules are more calcified.[12,96] A painful response may not be encountered until actual pulp exposure has occurred.

Anatomic landmarks used as guides to needle placement during block and infiltration injections are usually more distinguishable in older patients. The effects of epinephrine should be considered when selecting anesthetics for routine endodontic procedures. Anesthetics should be deposited very slowly (and skeletal muscle avoided) if epinephrine is the vasoconstrictor.

The reduced width of the periodontal ligament[54] makes needle placement for supplementary intraligamentary injections more difficult. Placing an anesthetic under pressure produces an intraosseous anesthesia that extends to the apex and to adjacent teeth, but it also distributes small amounts of solution systemically.[91] Intraosseous injections can significantly increase the success of pulpal anesthesia but can be associated with a transient increase in heart rate when anesthetics contain epinephrine. Cortical perforation requires less pressure and rarely causes irritation. Smaller amounts of anesthetic should be deposited during intraosseous injections, and the depth of anesthesia should be checked before the procedure is repeated. Like intrapulpal anesthesia, intraosseous anesthesia is not prolonged; the pulp tissue must be removed within 20 minutes. Most patients receiving an intraosseous injection of 2% lidocaine with 1:100,000 epinephrine (correct ratio) experience a transient increase in heart rate. This would not be clinically significant in most healthy patients, but in the older patient whose medical condition, drug therapies, or epinephrine sensitivity suggest caution, 3% mepivacaine is a good alternative for intraosseous injections (see Chapter 4 for further information).[81]

The reduced volume of the pulp chamber makes intrapulpal anesthesia difficult in single-rooted teeth and almost impossible in multirooted teeth. Initial pulp exposures are also hard to identify. Wedging a small needle into each canal to produce the necessary pressure for anesthesia is the method of last resort. Every effort should be made to produce profound anesthesia. Patients should be encouraged to report any unpleasant sensation, and a prompt response should be made to any complaint. Patients should never be expected to tolerate pulpal pain.

Isolation

Single-tooth isolation is preferred, but multitooth rubber dam isolation may be required. Badly broken-down teeth may not provide an adequate purchase point for the rubber dam clamp, so alternate rubber dam isolation methods should be considered (see Chapter 5). Multiple-tooth isolation may be used if adjacent teeth can be clamped and saliva output is low or a well-placed saliva ejector can be tolerated (Fig. 26-33). A three-holed rubber dam, with the tooth in question in the middle, allows for better maneuverability, especially when the dental operating microscope is used. A petroleum-based lubricant for the lips and gingiva reduces chafing from saliva or perspiration beneath the rubber dam. A reduction in salivary flow or an exaggerated gag reflex reduces the need for a saliva ejector. Tissue can be aspirated into the perforated tip of the saliva ejector and torn upon removal. Artificial saliva is available and should be used just before isolation because it is difficult to apply after the dam is in place.

Canals should be identified and their access maintained if restorative procedures are indicated for isolation. The clinician should not attempt isolation and access in a tooth with questionable marginal integrity of its restorations. Fluid-tight

FIG. 26-33 Multiple-tooth isolation techniques protect the patient from instruments but do not provide the fluid-tight environment preferred for the safe use of sodium hypochlorite.

isolation cannot be compromised when sodium hypochlorite is used as an irrigant. In initial preparation, defects that are difficult to isolate because of root decay present a good indication for the use of sonic handpieces that use flow-through water as an irrigant.

The many merits of single-visit root canal procedures are generally recommended when isolation is compromised (see Chapters 3 and 5). The few minor benefits of multiple-visit treatment are further reduced if an interappointment seal is difficult to obtain or the interval between visits is extended for any reason.

Access

Adequate access and identification of canal orifices are likely the most challenging aspects of providing root canal treatment for older patients. Although the effects of aging and multiple restorations may reduce the volume and coronal extent of the chamber or canal orifice, its buccolingual and mesiodistal positions remain the same and can be predicted from radiographs and clinical examination. In general, there is a good correlation between the size of a canal in relationship to its radiographic image, but there can be large discrepancies, especially when canals appear very small on radiograph.[58] Canal position, root curvature, and axial inclinations of roots and crowns should be considered during the examination (Fig. 26-34). The effects of access on existing restorations and the possible need for actual removal of the restoration should be discussed with the patient before treatment is begun (Fig. 26-35). Coronal tooth structure or restorations should be sacrificed when they compromise access for preparation or filling (Fig. 26-36). Perhaps all restorations should be removed before endodontic treatment so as to remove the common factors (caries, marginal breakdown, cracks) that may have caused the pulp and periradicular disease and to allow assessment of the tooth's prognosis and future treatment needs. One study found that these factors were present in almost all teeth (93%); however, only 56% were detectable clinically or on radiographs.[1] (See Chapter 5 for more details on proper access openings.)

Magnification in the range of ×2.5 to ×4.5 (e.g., Designs for Vision, Ronkonkoma, New York) has become a common tool and can be designed to fit the clinician's most comfortable working distance. The growing acceptance and availability of endodontic microscopes[4,53] offer clear magnification ranging up to ×25 or greater, and these microscopes have obvious advantages in the treatment of smaller and narrower geriatric canals.

Location and penetration of the canal orifice are often difficult and time-consuming in calcified canals. The most important instrument for initial penetration is the DG-16 explorer. The explorer does not stick in solid dentin, but it resists dislodgment in the canal. Once the canal has been distinguished, negotiation is attempted with a stainless steel (SS) #8, #10, or #15 K-file. The #6 file lacks stiffness in its shaft and easily bends and curls under gentle apical pressure. Nickel-titanium (NiTi) files lack strength in the long axis and are not useful for initial negotiation. The canal can be negotiated using a watch-winding action with slight apical pressure. Chelating agents are seldom of value in locating the orifice but can be very useful during canal negotiation. Dyes may distinguish an orifice from the surrounding dentin.

Pain, bleeding, disorientation of the probing instruments, or an unfamiliar feel to the canal may indicate a perforation (Fig. 26-37). The size of the perforation and the extent of contamination determine the success of repair (which should be done immediately) and do not necessarily indicate failure (Fig. 26-38). Supraerupted teeth can be easily perforated (Fig. 26-39) if the reduced distance to the furcation is not noted.

A lengthy, unproductive search for canals is fatiguing and frustrating to both the clinician and the patient. Scheduling a second attempt at this procedure is often productive. Personal clinical experiences and judgment determine when the search for the canals must be terminated and referral or alternatives to nonsurgical root canal treatment considered (Fig. 26-40).

Modifications to enhance access vary from widening the axial walls, to increasing visibility or light, to complete removal of the crown. Alterations may be indicated after canal penetration to the apex if tooth structure interferes with instrumentation or filling procedures.

Teeth with apical periodontitis usually have patent canals, even if they are undetectable on radiographs (Fig. 26-41). Surgical access (Fig. 26-42) may be preferred if the risk of deviation from the long axis exists when canals are calcified and the tooth is heavily restored (Fig. 26-43).

Very few canals of older teeth, even maxillary anterior teeth, have adequate diameter to allow safe and effective use of broaches. The clinical appearance of older pulps may reflect

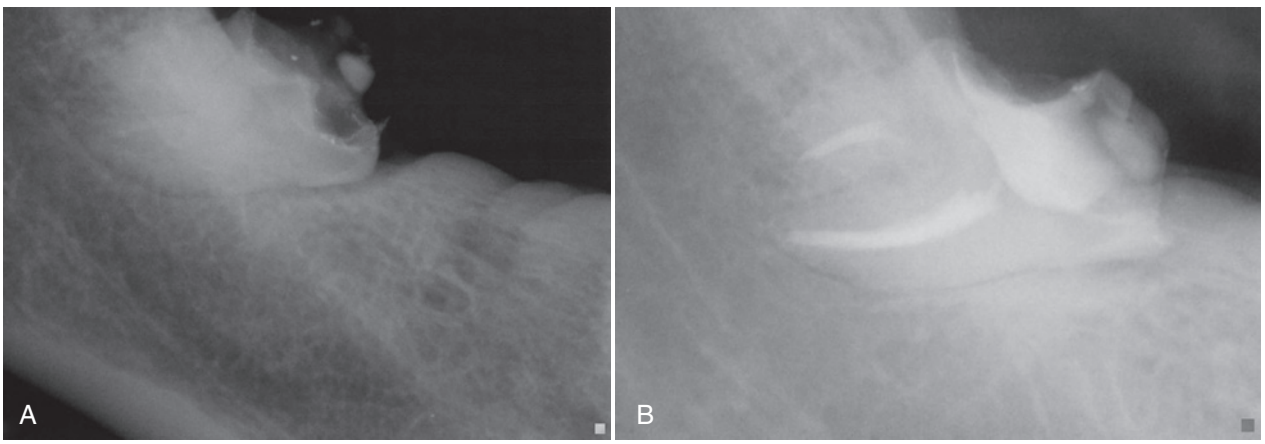

FIG. 26-34 A and B, This severely tilted mandibular third molar, which had a questionable prognosis, was saved in a patient at risk for bone osteonecrosis (BON).

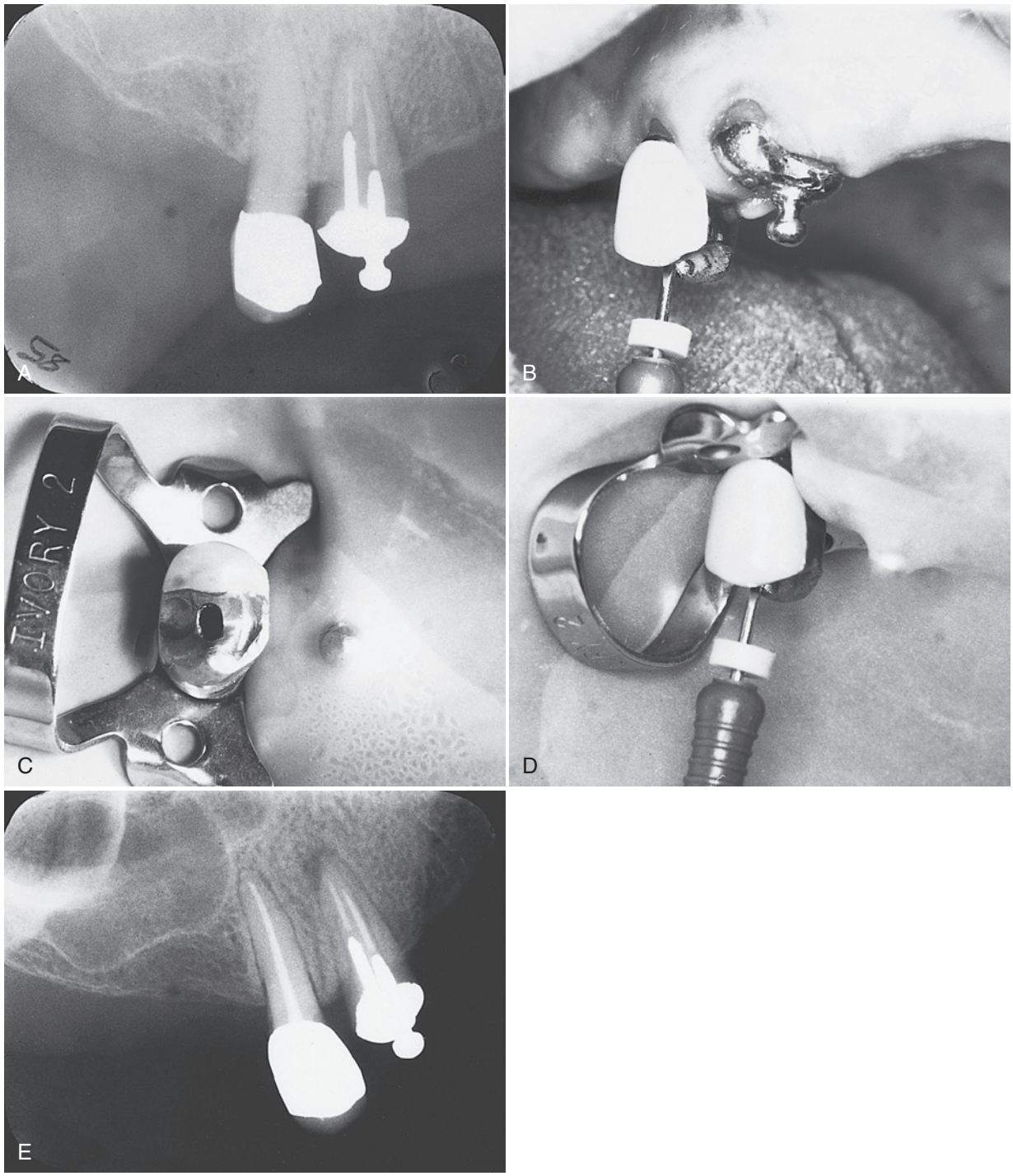

FIG. 26-35 A, Radiograph of a strategic abutment and a calcified canal. B, To increase visibility and alignment, access was initiated before isolation. C, An access opening that conserved tooth structure and provided straight-line access was isolated with the rubber dam (D), and treatment was provided (E).

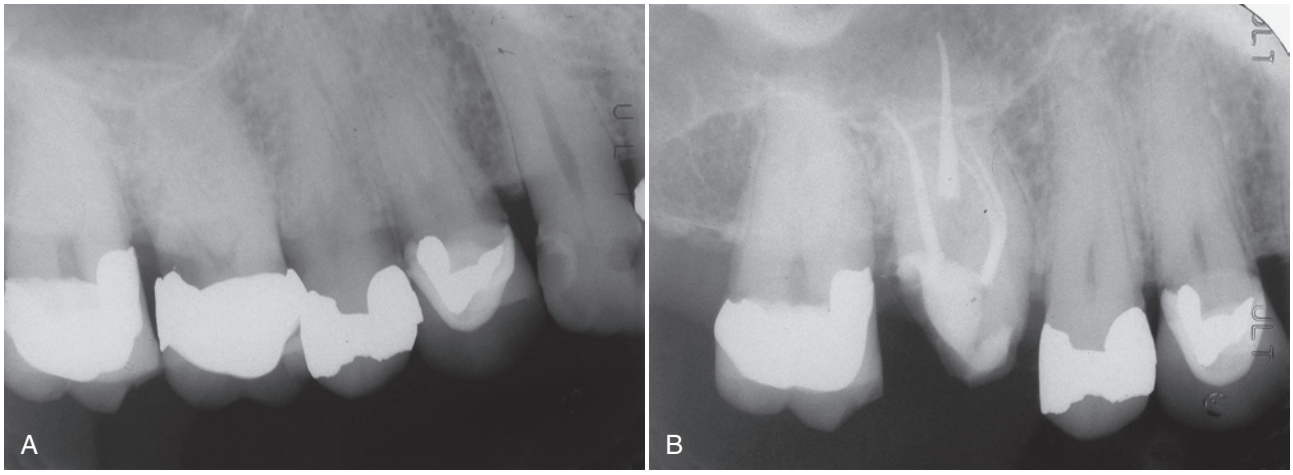

FIG. 26-36 A, Caries control and isolation of this tooth necessitated crown removal. B, Endodontic treatment was completed in one visit.

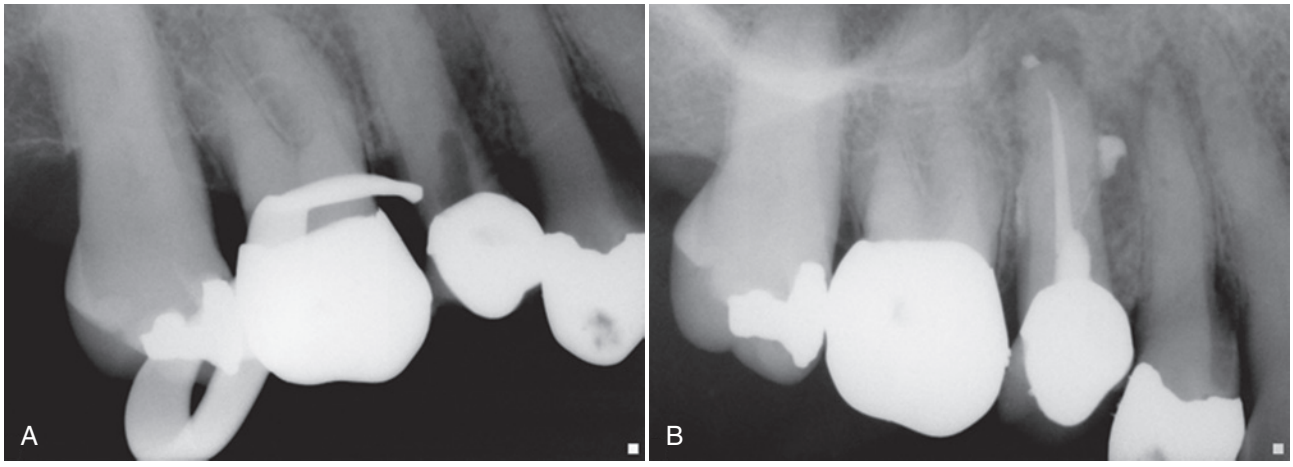

FIG. 26-37 A, Pain, bleeding, or disorientation of the probing instrument may indicate a perforation, which should be immediately investigated with a radiograph. B, Repair has the best chance of success when the perforation opening is pinpoint size and the periodontal tissues are normal.

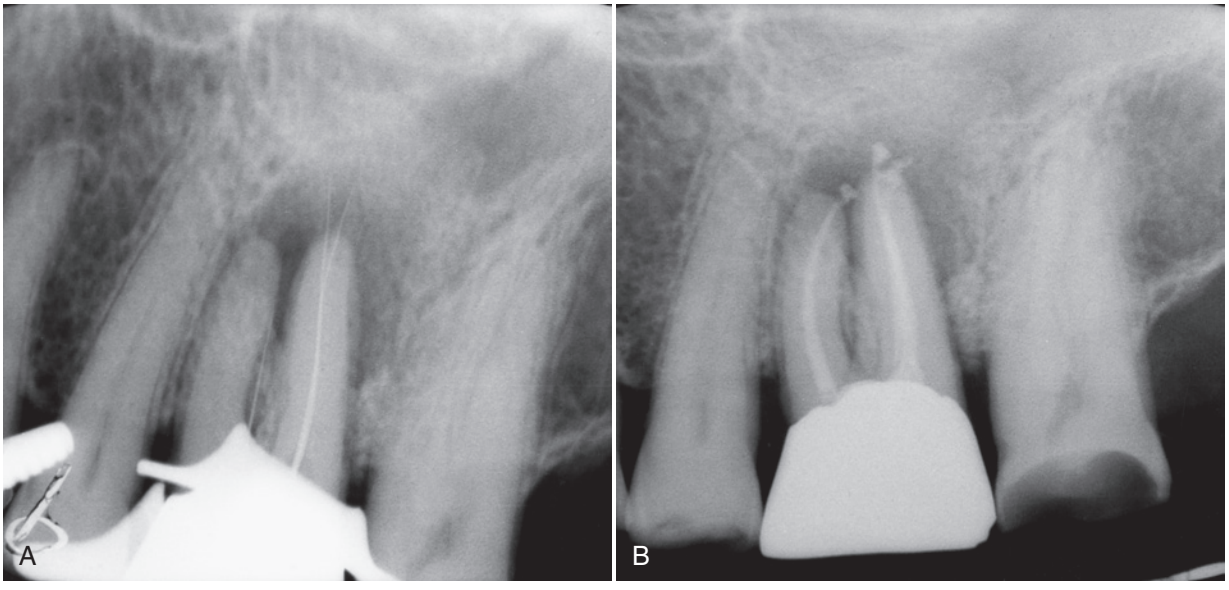

FIG. 26-38 A, Perforation occurred during access of these calcified canals. B, Endodontic treatment was immediately completed after the perforation repair.

their calcified, atrophic state; they may have the stiffened, fibrous consistency of a wet toothpick. Any broached canals should be thoroughly cleaned and shaped at the same appointment.

Preparation

The calcified appearance of the canals resulting from the aging process presents a much different clinical situation than that of a younger pulp in which trauma, pulpotomy, decay or caries, or restorative procedures have induced premature canal obliteration. Unless further complicated by reparative dentin formation, this calcification appears to be much more concentric and linear. This allows easier penetration of canals once they are found. An older tooth is more likely to have a history of earlier treatments, with a combination of calcifications present. (Chapter 6 includes a description of cleaning and shaping root canals.)

The length of the canal from the actual anatomic foramen to the CDJ increases with the deposition of cementum throughout life.[57] The advantage of this situation in the treatment of teeth with vital pulps is countered by the presence of necrotic, infected debris in this longer canal when periapical pathosis is already present. The actual CDJ width or most apical extent of the dentin remains constant with age.[97]

Flaring of the canal should be performed as early in the procedure as possible to provide for a reservoir of irrigation solution and reduce the stress on metal instruments that occurs when they bind with the canal walls. Thorough and frequent irrigation should be performed to remove the debris that could block access. Files with a triangular or square cross section may penetrate into the walls, resulting in greater strain than small files (when used with a reaming action) and create more instrument torque and fracture. Instrumentation techniques used for the preparation of calcified canals require an advanced understanding of the variations in taper, tip geometrics, rake angle, pitch, and metallurgy. Instruments with little or no rake angle and a crown-down technique are recommended.

Because this CDJ is the narrowest constriction of the canal, it is the ideal place to terminate the canal preparation. This point may vary from 0.5 to 2.5 mm from the radiographic apex and may be difficult to determine clinically. Calcified canals reduce the clinician's tactile sense in identifying the constriction clinically, and reduced periapical sensitivity in older patients reduces the patient's response that would indicate penetration of the foramen. An increased incidence of hypercementosis, in which the constriction is even farther from the apex, makes penetration into the cemental canal almost impossible. Achieving and maintaining apical patency are more difficult. Apical root resorption associated with periapical pathosis further changes the shape, size, and position of the constriction. The use of electronic apex-finding devices is

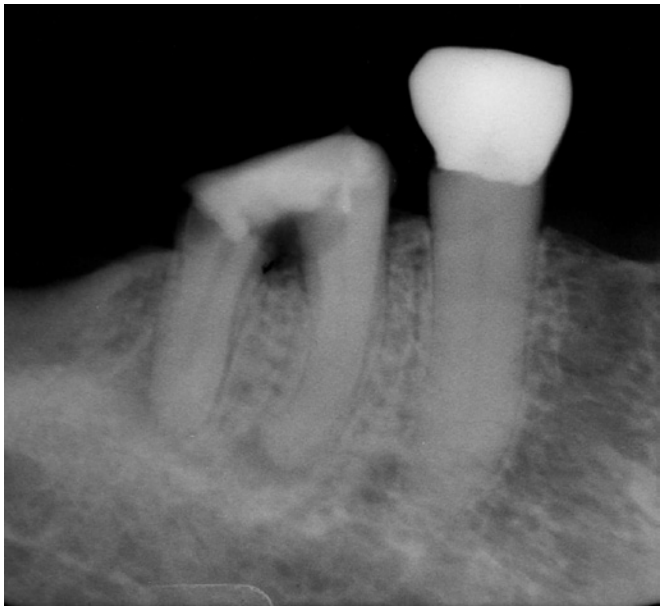

FIG. 26-39 Supraerupted mandibular first molar that was perforated during access.

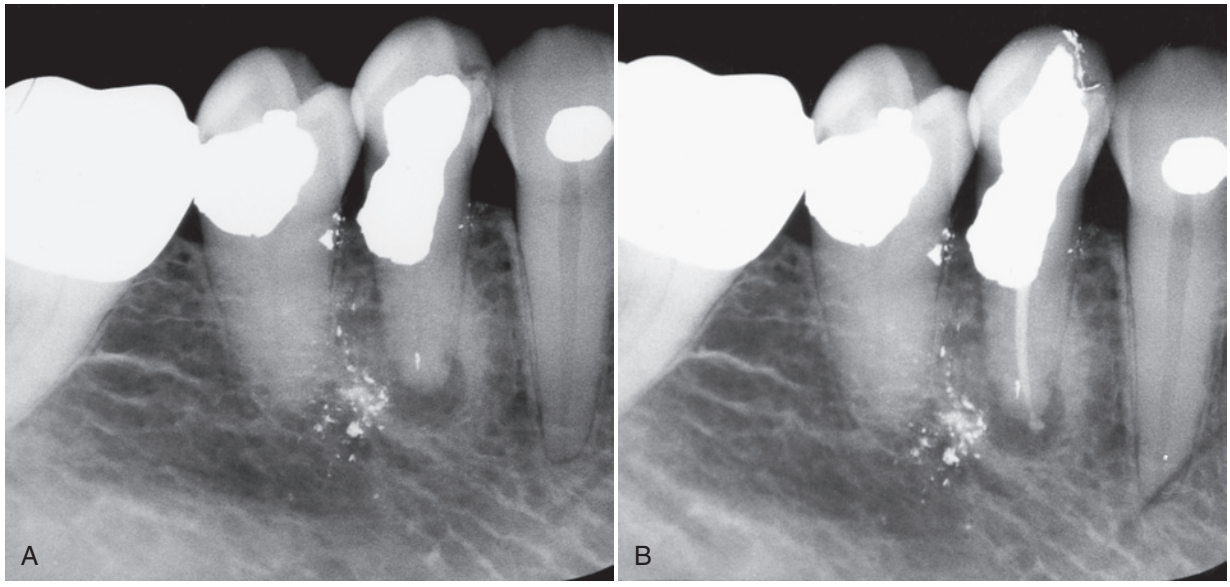

FIG. 26-40 A, Midtreatment referral of a mandibular first premolar after perforation and repair with amalgam. B, The canal was still detectable with enhanced vision techniques, and treatment was completed.

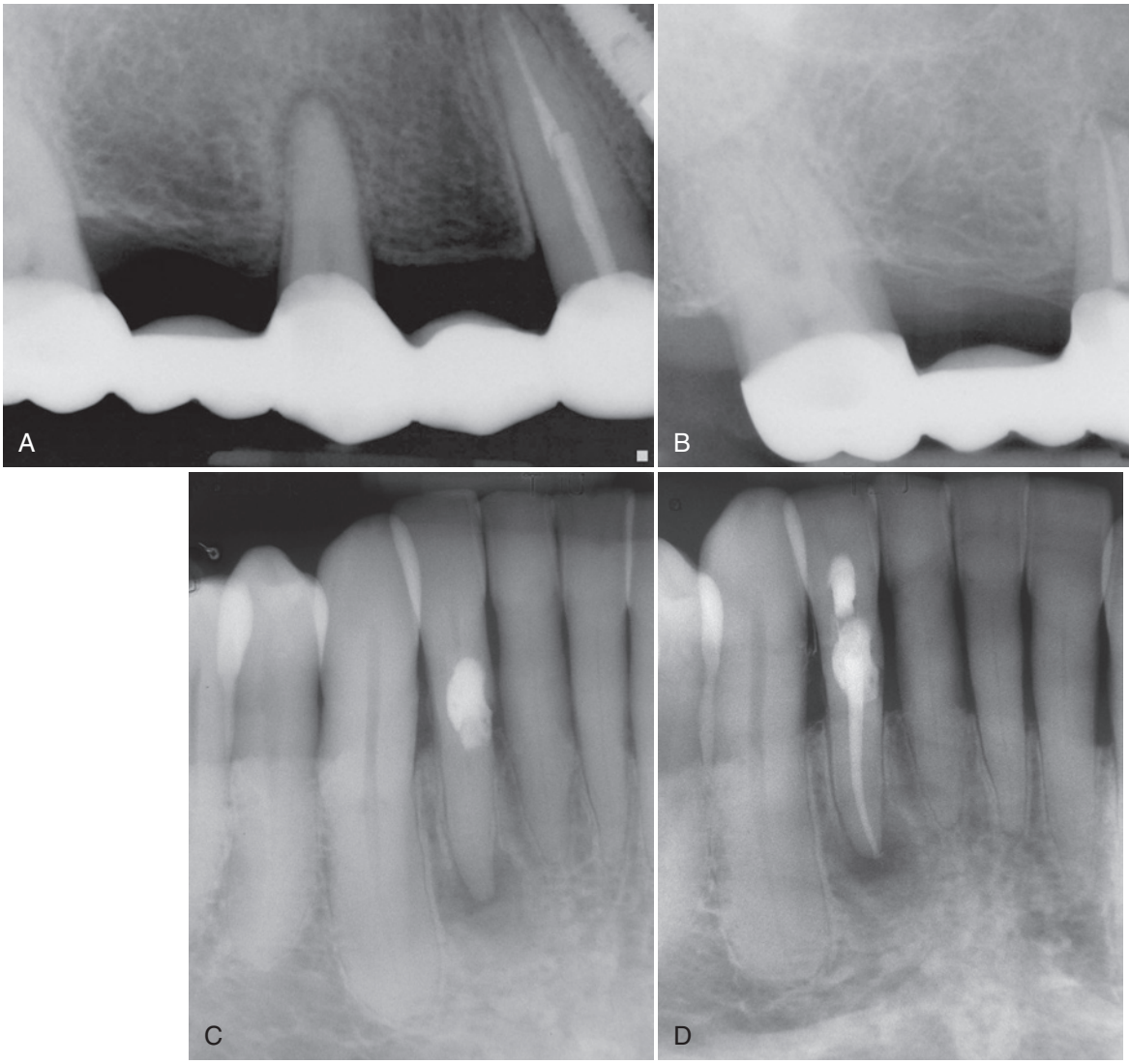

FIG. 26-41 A, Strategic middle abutment for a long fixed bridge presents a challenge to the most skilled clinician for minimally invasive root canal treatment. B, Root canal treatment should have been considered before this longstanding premolar was established as an abutment. C, Calcified canals can usually be detected and treated when a nonvital pulp and chronic apical periodontitis are present, even when the canals are not apparent on the radiograph. Asymptomatic, calcified canals in teeth with vital pulps are much more difficult to find and negotiate. D, External resorption repair before endodontic treatment made negotiation and instrumentation of this incisor tooth more difficult.

sometimes limited in heavily restored teeth when contact with metal can bleed off the current.

The incidence and intensity of flare-ups[103] after cleaning and shaping have not been shown to be related to the amount of preparation, the type of interappointment medication or temporary filling, the pulp or periapical status, the tooth number or age, or whether the root canal filling is completed at the same appointment.[100] The more constricted CDJ permits a much smaller pulp wound and resists penetration, even with the initial small files. Patency is difficult to establish and maintain. Dentin debris creates a matrix early in the preparation[114] and further reduces the risk of overinstrumentation or the forcing of debris into the periapical tissues,[77] which could cause an acute apical periodontitis or abscess. Further access to periapical tissues through the canal is likewise limited with a dentinal plug. Crown-down rotary instrumentation creates less apical dentin than the push-pull motion typical of hand instrumentation.

Obturation

For the older patient, the prudent clinician selects gutta-percha filling techniques that do not require unusually large midroot tapers and do not generate pressure in this area, which could result in root fracture (Fig. 26-44). (See Chapter 7 for the most appropriate technique to use to seal the canal.)

The coronal seal plays an important role in maintaining an apically healthy environment, and it has a significant impact on long-term success.[99] Even a root-filled tooth should not have its canals exposed to the oral environment.[107] Permanent restorative procedures should be scheduled as soon as possible, and intermediate restorative materials should be selected and properly placed to maintain a seal until that time. When

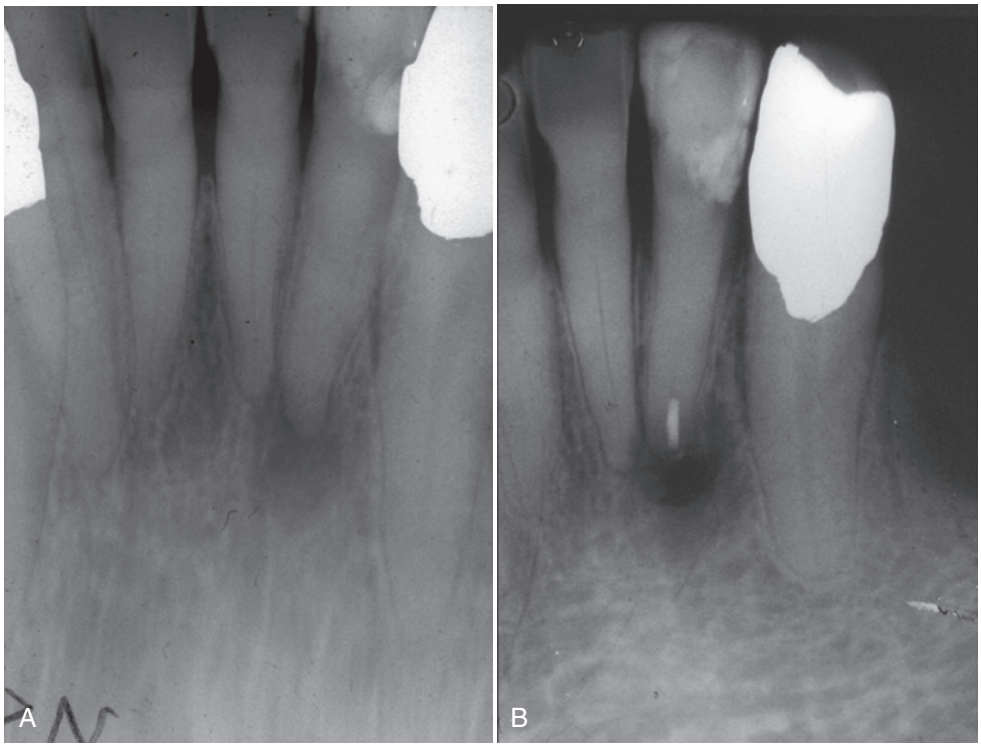

FIG. 26-42 A, Extremely calcified canals and thin roots make the risk of perforation high. B, The operator chose to perform surgical access and root-end filling, rather than weakening the tooth trying to locate the root canal system via an orthograde approach.

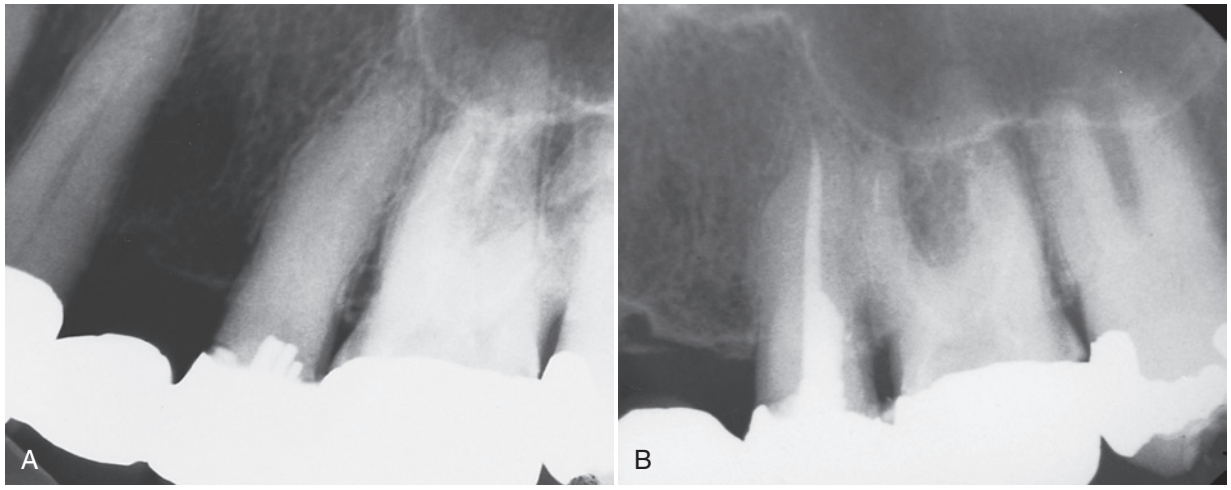

FIG. 26-43 A, Multiple pins were used to restore this middle abutment on a fixed bridge. B, Extreme care and exceptional skill are required to maintain proper alignment to find and treat the canal with a deep access opening.

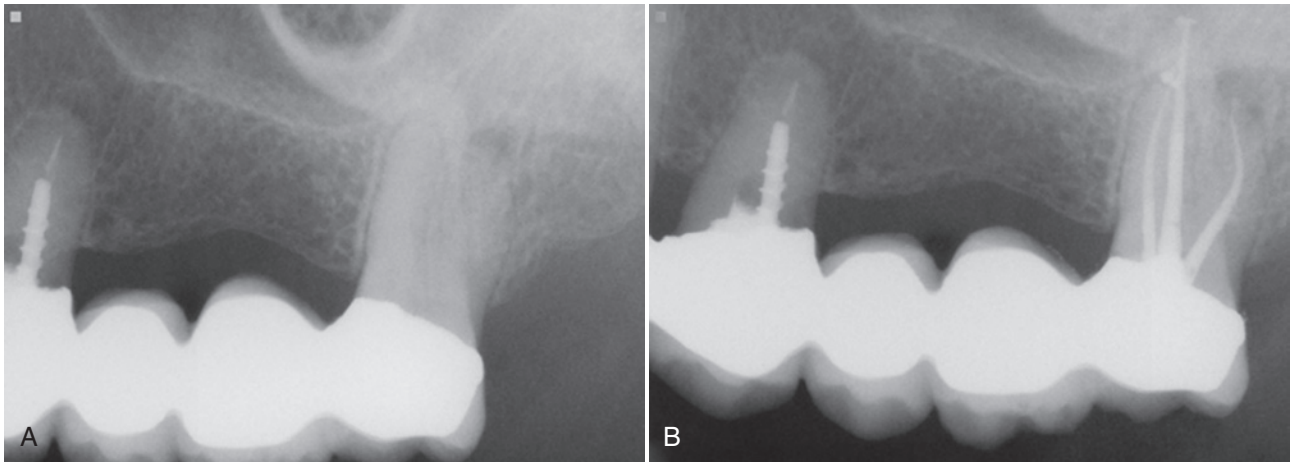

FIG. 26-44 A, Third molar abutment for a seven-unit bridge in an 84-year-old patient with limited opening. B, Instrumentation of this molar was performed without excessive removal of coronal or midroot dentin.

mechanical retention is not ensured with the preparation, glass ionomer cements are recommended.

Success and Failure

Repair of periapical tissues after endodontic treatment in older patients is determined by most of the same local and systemic factors that govern the process in all patients. With vital pulps, periapical tissues are normal and can be maintained with an aseptic technique, confining preparation and filling procedures to the canal space. In infected nonvital pulps with periapical pathosis, this process must be altered in favor of the host tissue, and repair is determined by the ability of this tissue to respond. Factors that influence repair have their greatest effect on the prognosis of endodontic therapy when periapical abnormalities are present. Patients with diabetes have increased periodontal disease in teeth involved endodontically and have a reduced likelihood of success of endodontic treatment in cases with preoperative periradicular lesions.[33] With aging, arteriosclerotic changes in blood vessels increase and the viscosity of connective tissue is altered, making repair more difficult. The rate of bone formation and normal resorption decreases with age, and the aging of bone results in greater porosity and decreased mineralization of the formed bone. Although periradicular tissues heal as readily in the elderly as in young patients,[100] a 6-month recall period to evaluate repair by means of radiographs may not be adequate (Fig. 26-45).[36]

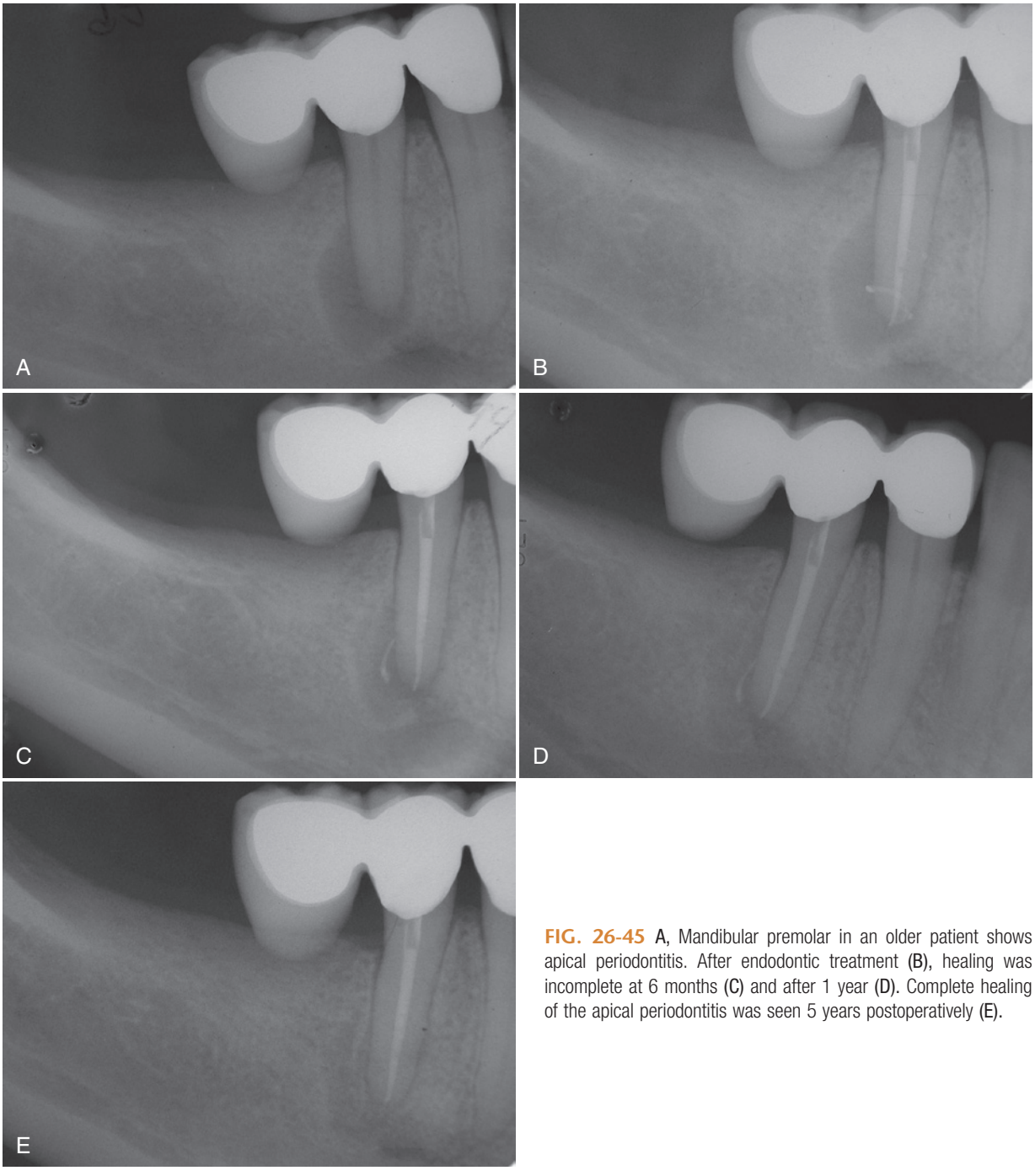

FIG. 26-45 A, Mandibular premolar in an older patient shows apical periodontitis. After endodontic treatment (B), healing was incomplete at 6 months (C) and after 1 year (D). Complete healing of the apical periodontitis was seen 5 years postoperatively (E).

Studies that suggest differences in success among age groups must note the smaller numbers (usually in this older treatment group) and the local factors that make treatment difficult. Overlooked canals are a more common cause of failure in older patients (Fig. 26-46), which explains the increased clinical indications for retrograde fillings when surgical treatment is attempted.[2] As an isolated symptom, heat sensitivity may indicate a missed canal. (See Chapters 8 and 11 for further information on assessing failures and possible retreatment.)

FIG. 26-46 The incidence of root-end filling during periradicular surgery increases with age because calcified canals become more difficult to detect and negotiate, causing failure of nonsurgical treatment.

ENDODONTIC SURGERY

Generally, considerations and indications for endodontic surgery are not affected by age. The need to establish drainage and relieve pain is not a common indication for surgery. Anatomic complications of the root canal system, such as small (Fig. 26-47) or completely calcified canals, nonnegotiable root curvatures, extensive apical root resorption, or pulp stones, occur with greater frequency in older patients. Perforation during access, losing length during instrumentation, ledging, and instrument separation are iatrogenic treatment complications associated with treatment of calcified canals.

Medical considerations may require consultation but do not contraindicate surgical treatment when extraction is the alternative (Fig. 26-48). In most instances, surgical treatment may be performed less traumatically than an extraction, which may also result in the need for surgical access to complete root removal. Many older patients receive low-dose aspirin therapy to prevent blood clot formation and may be subject to embolic formation if the treatment is interrupted. Aspirin therapy should be continued throughout dental procedures, even during extraction or surgery. Local measures are sufficient to control bleeding.[5] Interrupting therapeutic levels of continuous anticoagulation therapy for dental surgery is not based on scientific fact but seems to be based on its own mythology.[109] An international normalized ratio (INR) value recorded the day of the procedure provides the most useful measurement of bleeding risk. Many patients on anticoagulation therapy are aware of their INR value, but a current measurement should be made. In patients receiving combined anticoagulation-aspirin therapy, simple tooth extraction can be performed safely without discontinuing either oral anticoagulant or antiplatelet therapy if the INR is within therapeutic range and appropriate local hemostasis measures are provided.[6] Clinicians should also reassess historical recommendations that

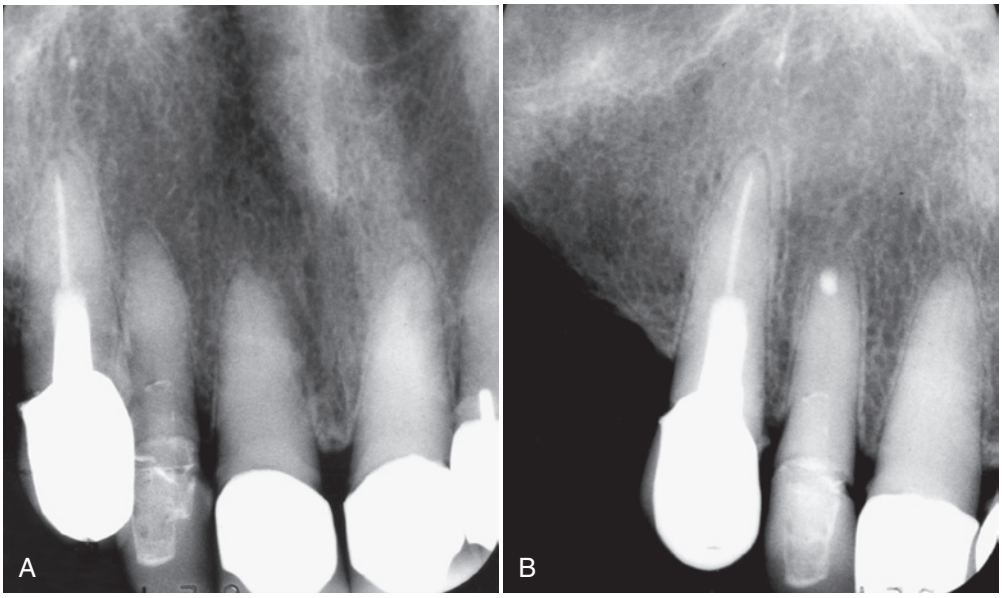

FIG. 26-47 **A**, A small canal is detectable on the radiograph, even though it appeared that an earlier, unsuccessful attempt had been made to find it. **B**, It was decided that the risk of damage to a satisfactory restoration justified surgical treatment for this patient.

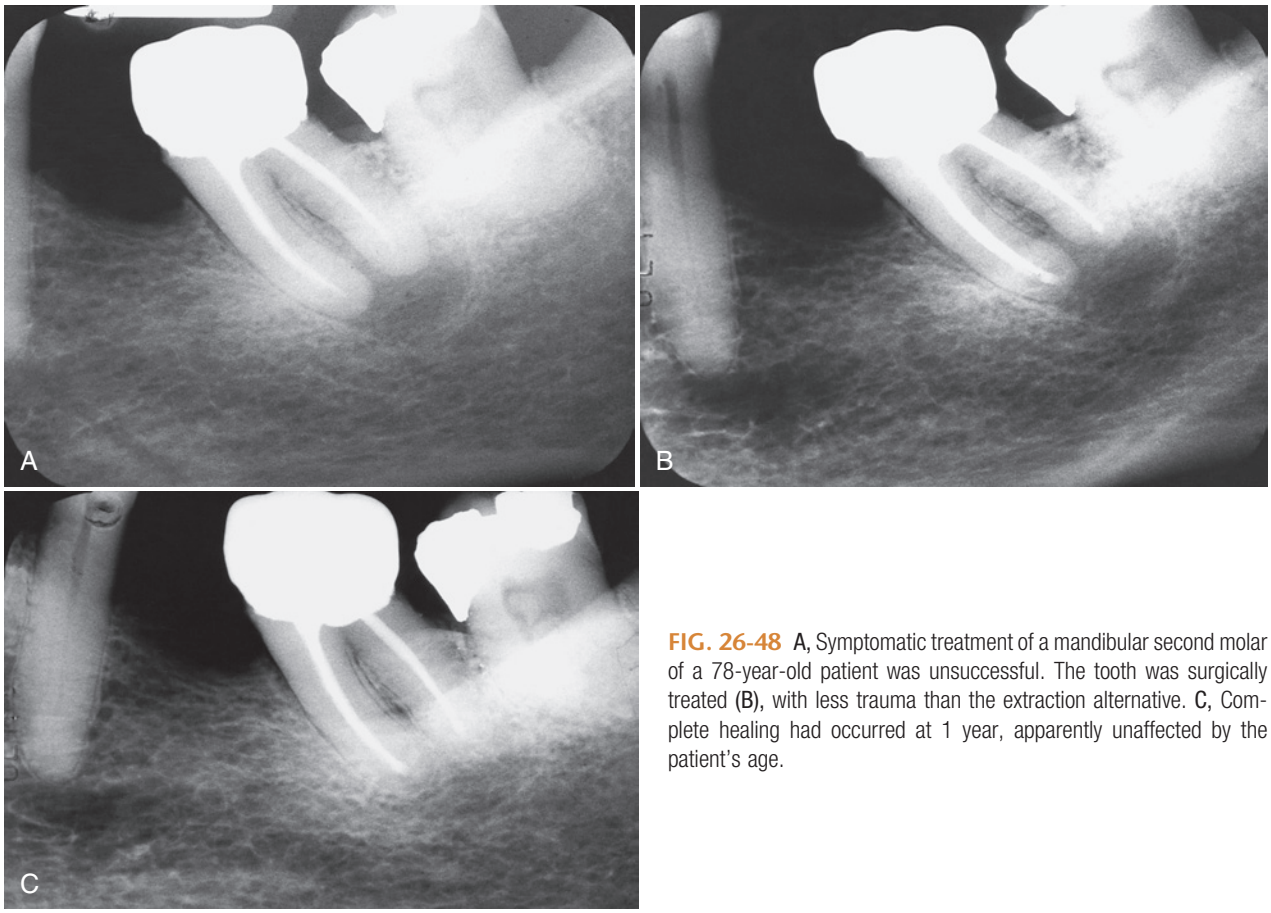

FIG. 26-48 A, Symptomatic treatment of a mandibular second molar of a 78-year-old patient was unsuccessful. The tooth was surgically treated (B), with less trauma than the extraction alternative. C, Complete healing had occurred at 1 year, apparently unaffected by the patient's age.

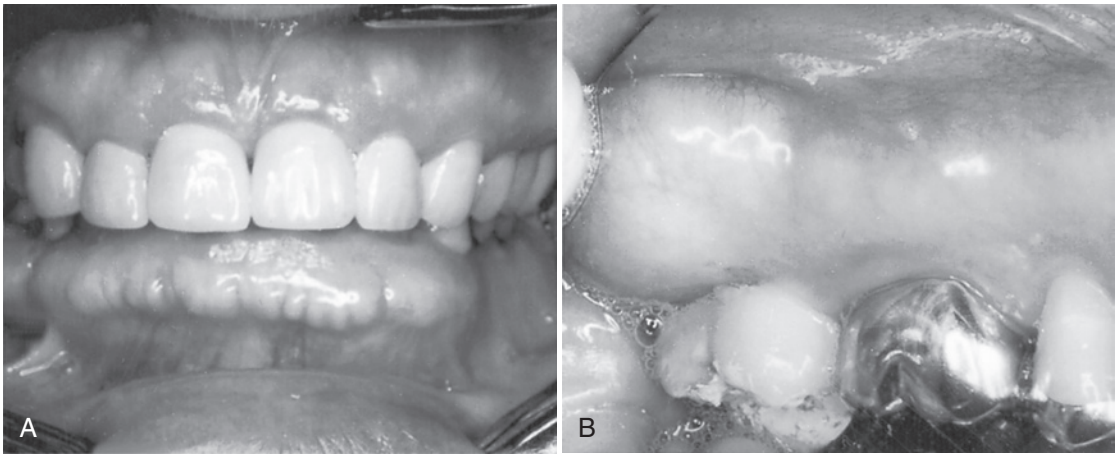

FIG. 26-49 Exostoses are shown in this mandibular anterior tooth (A) and maxillary molar (B), covered with thin tissue that can easily be torn during flap reflection. Another consideration is the more obvious heavy bone and its effect on surgical access.

dental care in older patients be postponed for as long as 6 months after ischemic vascular events.[89] A thorough medical history and evaluation should reveal the need for any special considerations, such as prophylactic antibiotic premedication, sedation, hospitalization, or more detailed evaluation.

Local considerations in the treatment of older patients include an increase in the incidence of fenestrated or dehisced roots and exostoses (Fig. 26-49). The thickness of overlying soft and bony tissue is usually reduced, and apically positioned muscle attachments extend the depth of the vestibule. Smaller amounts of anesthetic and vasoconstrictor are needed for profound anesthesia. Tissue is less resilient, and resistance to reflection appears to be diminished. The oral cavity is usually more accessible with the teeth closed together because the lips can more easily be stretched. The apex can actually be more surgically accessible in older patients. The ability to gain such

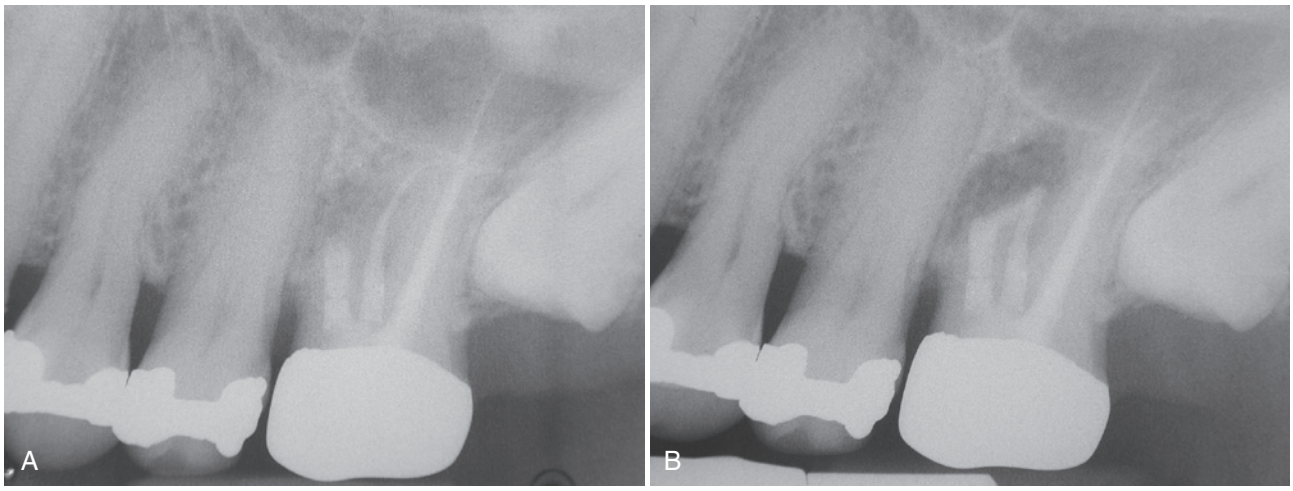

FIG. 26-50 A, Tooth #15 showing failed surgery; it appears that only part of the MB root was resected. B, Apicoectomy of the entire MB root, retrocavity preparation of the MB1 and MB2 canals and joining isthmus, and retrofilling with mineral trioxide aggregate (MTA).

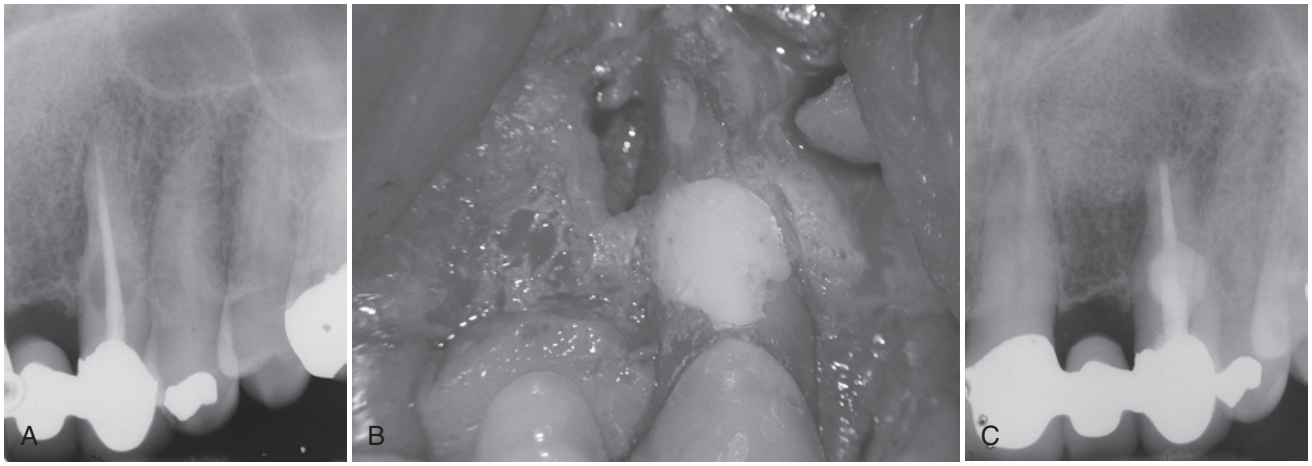

FIG. 26-51 A, External root resorption accompanied by periradicular periodontitis in an important canine abutment tooth. Endodontic retreatment, surgical repair of the resorptive defect (B), and apicoectomy (C) were performed.

access varies with the skill of the surgeon; however, some areas are unreachable by even the most experienced clinicians.

The position of anatomic features (i.e., the sinus, floor of the nose, and neurovascular bundles) remains the same, but their relationship to surrounding structures may change when teeth have been lost. The need may arise to combine endodontic and periodontal flap procedures, and every effort should be made to complete these procedures in one sitting.

When root-end surgery is to be performed, the surgeon must consider whether the root that will be left is long enough and thick enough for the tooth to continue to remain functional and stable (Fig. 26-50). This factor is especially important when the tooth will be used as an abutment (Fig. 26-51). (Detailed surgical procedures are presented in Chapter 9.)

Ecchymosis is a more common postoperative finding in older patients and may appear to be extreme. The patient should be reassured that this condition is normal and that normal color may take as long as 2 weeks to return. The blue discoloration changes to brown and yellow before it disappears. Immediate application of an ice pack after surgery reduces bleeding and initiates coagulation to reduce the extent of ecchymosis. Later, application of heat helps dissipate the discoloration.

RESTORATION

Root canal treatment saves roots, and restorative procedures save crowns. Combined, these procedures are returning more teeth to form and function than were thought possible just a few decades ago. Successful restorative outcomes have been shown to be related to full coverage restorations on molars and older patient age.[93] Special consideration must be given to post design, especially when small posts are used in abutment teeth; root fracture is common in older adults when much taper is used. Post failure or fracture occurs when small-diameter parallel posts are used (Fig. 26-52). Posts are not usually needed

FIG. 26-52 This fracture of a cast post presents a very difficult challenge for removal.

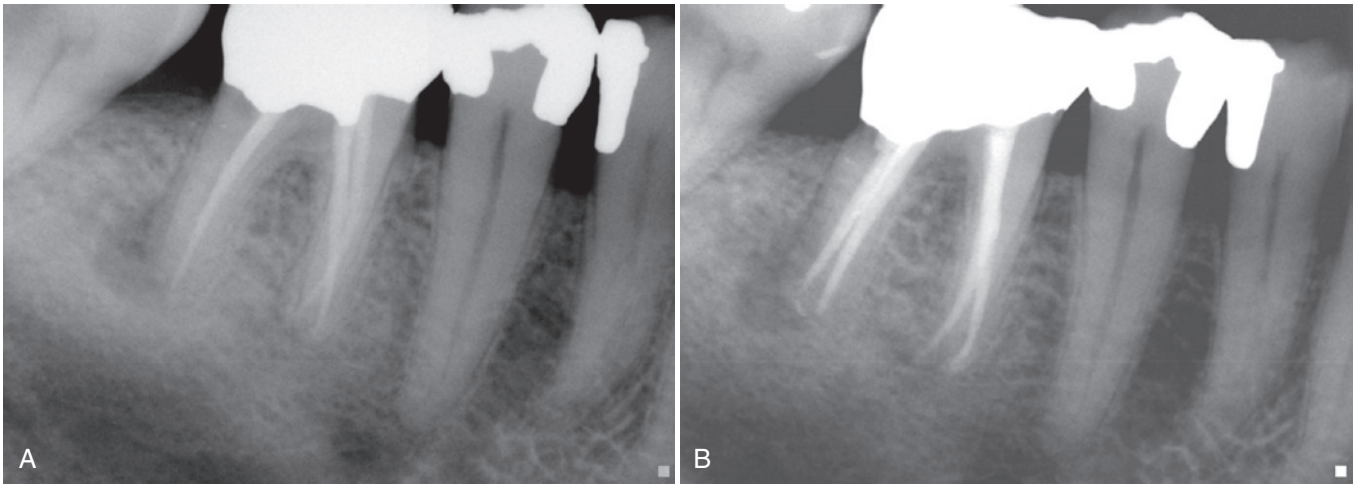

FIG. 26-53 A, Intact margins permitted the continued use of this crown. B, Root canal failure was due to an uncleaned and unfilled second distal canal. The access cavity was restored with an amalgam-bond core.

when root canal treatment is performed through an existing crown that will continue to be used (Fig. 26-53). (General considerations and procedures for postendodontic restoration are detailed in Chapter 22.)

The value of the tooth, its restorability, its periodontal health, and the patient's wishes should be part of the evaluation preceding endodontic therapy. The restorability of older teeth can be affected when root decay has limited access to sound margins or reduced the integrity of remaining tooth structure (Figs. 26-54 and 26-55). There can also be insufficient vertical and horizontal space when opposing or adjacent teeth are missing. The patient's desire to save appliances can sometimes be fulfilled with creative devices that may outlive them (Fig. 26-56).

Geriatric endodontics will gain a more significant role in complete dental care as an aging population recognizes that a complete dentition, and not complete dentures, is part of its destiny. Saving teeth for the aging population is a valuable and important aspect of helping older individuals maintain their quality of life.

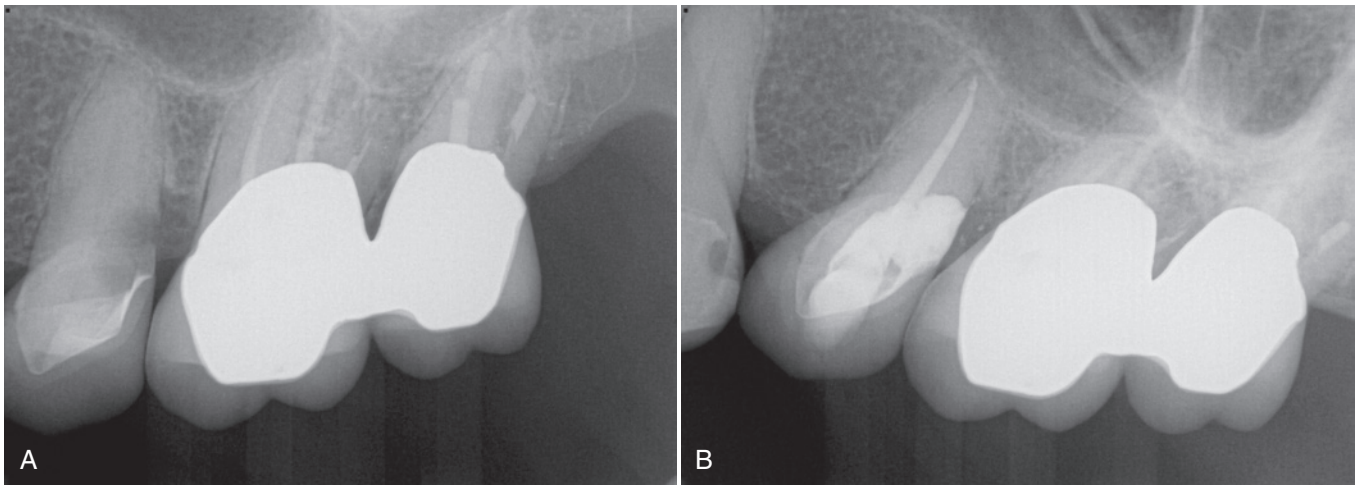

FIG. 26-54 A, This 82-year-old woman insisted on saving her tooth despite extensive root caries on the distal aspect of tooth #13. B, Root canal treatment was performed, followed by a surgical approach to remove the root caries. Restoration was done with Geristore.

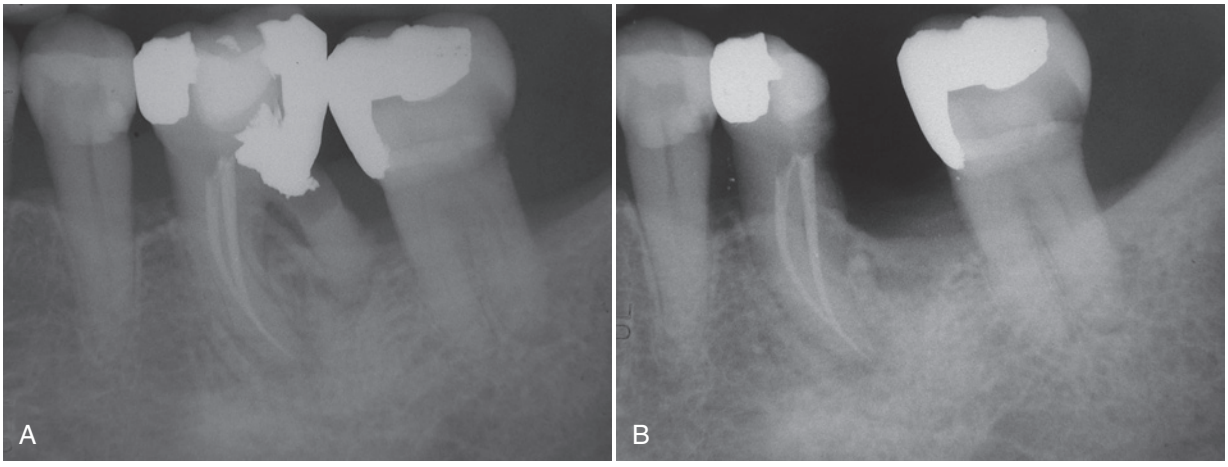

FIG. 26-55 A, Root caries and periodontal involvement of this mandibular molar tooth necessitated hemisection to retain the mesial root for restoration (B).

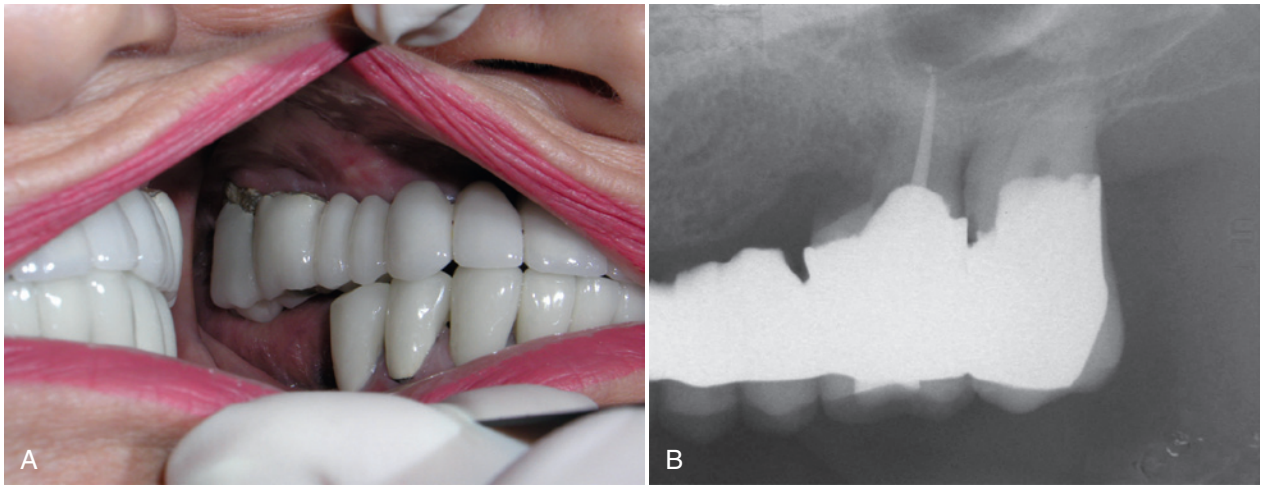

FIG. 26-56 A, Unrestorable caries contraindicated endodontic treatment on this molar abutment. B, Amputation of the buccal roots was performed to extend the life span of this bridge.

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CHAPTER 27

Bleaching Procedures

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CHAPTER OUTLINE

Etiology of Tooth Discolorations
Systemic Intrinsic Causes
Local Intrinsic Causes
Extrinsic Causes
Treatment Planning for Internal Bleaching
Contraindications of Bleaching
Alternatives to Bleaching
Chemistry of Bleaching
Walking Bleach Technique
Thermocatalytic Bleaching
Permanent Restoration of Teeth Following Internal Bleaching
External Bleaching
In-Office External Bleaching
"Power Bleaching"
At-Home External Bleaching
Risks and Complications Associated with Tooth Bleaching
Risks Associated with Internal Tooth Bleaching
Risks Associated with External Tooth Bleaching
Prognosis of Bleaching

Society, in general, is more concerned about beauty and perfect looks than ever before. In particular, the "perfect smile" has gained more and more importance, including straight and light-colored teeth. Tooth whitening has become a veritable business, of both the dental profession that offers vital in-office bleaching procedures, as well as of commercial companies that sell over-the-counter tooth "whitening" products. Whereas vital tooth bleaching aims to lighten the entire arch, nonvital bleaching procedures are used to treat individual discolored teeth so that they will blend in with adjacent teeth.

ETIOLOGY OF TOOTH DISCOLORATIONS

Discoloration of teeth can be attributed to various etiologies. Discoloration was described as "any change in the hue, color, or translucency of a tooth due to any cause."[174] Great differences exist in severity, localization, and appearance. Intrinsic discolorations are incorporated into the tooth structure. Extrinsic discolorations are attached to the tooth surface. Intrinsic and extrinsic discolorations may exist in combination and can affect enamel, dentin, or the pulp. Tooth discolorations may be hereditary, related to patient behavior, age, and disease, or caused iatrogenically by dental and medical treatment (Table 27-1).

Systemic Intrinsic Causes

Genetic Causes

Amelogenesis imperfecta (AI) describes a group of hereditary conditions associated with mutations in five genes—AMEL (amelogenin), ENAM (enamelin), MMP20 (matrix metalloproteinaise-20), KLK4 (kallikrein-4), and FAM83H—affecting the structure and appearance of enamel in the primary and secondary dentition.[66] Enamelin defects may cause variable hypoplasia ranging from local pitting to generalized thinning of the enamel. Amelogenin defects can lead to abnormal maturation and mineralization defects; disorganized, hypoplastic enamel; or other phenotypic variations ranging from hypoplasia to hypomaturation or hypomineralization.[66] Clinical appearance and morphologic structure of the enamel can be affected by discoloration as well as increase sensitivity and fragility. Due to the impaired mineralization resulting from the developmental changes in the enamel matrix formation, discoloring agents in the oral cavity may gain easy access to the mineral structure. The tooth color may be brown or yellow. Bleaching attempts may be, although successful, of a temporary nature and seen as an adjunct to comprehensive restorative treatment.

Dentinogenesis imperfecta (DGI) and dentinal dysplasia (DD) are a group of hereditary autosomal dominant genetic dentine disorders, characterized by an abnormal dentine structure.[18] Either primary or both, primary and secondary dentitions may be affected.[18] Dentinogenesis imperfecta is most commonly categorized into three subtypes: DGI type I, DGI type II, and DGI type III; dentinal dysplasia is categorized into two subtypes: DD type I and DD type II. DGI type I is associated with osteogenesis imperfecta and involves mutations in the collagen type 1 encoding genes COL1A1 and COL1A2. With the exception of DD type I, all other forms of dentinogenesis imperfecta and dentinal dysplasia are related to mutations in the gene encoding dentine sialophosphoprotein (DSPP).[18] All patients affected by osteogenesis imperfecta also suffer from brittle bones. Patients with dentinogenesis imperfecta have weaker teeth that are more prone to fracture. Patients suffering from DGI type I, DGI type II, and DGI type III have teeth with yellow-brown, deep amber, or blue-gray discolorations and higher translucency. Bleaching treatment will have

TABLE 27-1

Overview of Intrinsic and Extrinsic Discolorations

Color of Stain	Extrinsic	Intrinsic (Single Tooth)	Intrinsic (Multiple Teeth)
Black	Betel nut Chromogenic bacteria Coffee Dental plaque Metals Tea Tobacco Wine	Amalgam restorations Glass ionomer restorations Pulpal trauma with hemorrhage Minocycline (revascularization procedures) Metals (silver, gold, alloys)	Minocycline (oral) Tetracycline (oral)
Gray		Metals (silver, gold, alloys) Silicate compounds (e.g., MTA)	Dentinogenesis imperfecta Thalassemia
Brown/reddish	Coffee Dental plaque Doxycycline Drugs (e.g., khat, marijuana) Iodine Metals/halides Oral rinses (e.g., chlorhexidine) Other foods Tea Tobacco Wine	Caries (active, arrested) Cervical root resorption Composite resin, glass ionomer restorations Dental trauma Endodontic irrigation solutions Iodine Minocycline (revascularization procedures) Pulpal trauma with hemorrhage Resorcinol-formaldehyde resin	Amelogenesis imperfecta Congenital erythropoietic porphyria Dental trauma Dentinogenesis imperfecta Fluorosis Infection during enamel formation Tetracycline (oral) Thalassemia
Green	Chromogenic bacteria Metals Tea	—	Hyperbilirubinemia Thalassemia
Orange	Chromogenic bacteria Metals Doxycycline	—	—
Yellow	—	Caries (active) Composite resin, glass ionomer restorations Dental trauma	Aging Amelogenesis imperfecta Dentinogenesis imperfecta Dental trauma Dentinal dysplasia Fluorosis Hemolytic diseases Hemoglobin, heme Hyperbilirubinemia Nutrition deficiency (e.g., rickets, scurvy)
White	—	Caries (incipient) Dental trauma	Calcification due to trauma Fluorosis Infection during enamel formation Nutrition deficiency (e.g., rickets, scurvy)

minimal to no effect; comprehensive restorative treatment is indicated, but it is difficult to execute due to the compromised tooth structure.

Other diseases causing tooth discoloration include porphyria, erythroblastosis fetalis or thalassemia, and sickle cell anemia. Congenital erythropoietic porphyria is a disease resulting from abnormalities in the synthesis of porphyrins, organic compounds essential for the function of hemoglobin. It may result in reddish or brownish discoloration. Erythroblastosis fetalis is a hemolytic disease wherein fetal erythrocytes in Rh-positive fetuses or newborns of Rh-negative mothers are destroyed by antigens that cross the placenta, leading to hyperbilirubinemia. Bilirubin, a yellow breakdown product of hemoglobin, is incorporated into developing teeth, creating yellow-green and blue-green discolorations. Thalassemia, a blood disease causing characteristic malformations of the skull and long bones, and sickle cell anemia, a hereditary blood disorder displaying an abnormality of the oxygen-carrying hemoglobin molecule causing red blood cell malformations, may result in intrinsic blue, green, and brown discolorations.

Disease-Related Causes

High fevers experienced during the ages of tooth development may cause chronologic enamel hypoplasia leading to banding

type discolorations on the tooth surface. Vitamin and mineral deficiencies may induce hypoplasia. Rickets, a vitamin D deficiency, may lead to white hypoplasias resulting from osteomalacia, developmental anomalies in the bones. Scurvy, vitamin C deficiency in conjunction with vitamin A deficiency, and disturbances of phosphorus uptake can cause enamel hypoplasias.

Metabolic Causes

Enamel hypoplasia may also occur after excessive exposure to fluoride during tooth formation. This exposure can lead to alterations in the mineralized tooth structure, especially in the enamel matrix, and give the appearance of a mottled tooth. The extent of hypoplasia directly correlates with the fluoride uptake during tooth formation and will impact the degree of discoloration.[39] Shades of discoloration may vary greatly and can range from white spots with opaque or chalky appearances to yellow or brown stains on the facial side of the affected tooth, in severe cases with surface pitting of the enamel. No damage to the shape of the crown can be found. It can be related to amelogenesis imperfecta or hereditary hypophosphatemia. Tumors, infections, or trauma may be contributing factors. In situations of moderate severity, external bleaching may be sufficient, whereas in severe cases, a restorative approach may be indicated.

Drug-Related Causes

Tetracycline is an antibiotic used to treat diseases such as urinary tract infections, chlamydia, and acne. It was frequently used from the 1950s through the early 1980s. In particular in young children, it causes gray or brown, deep, dark stains that may affect the entire tooth or occur as a pattern of horizontal stripes. Many adults also currently suffer from tetracycline stains on their teeth. Tetracycline induces discolorations in permanent teeth manifest during tooth development, when tetracycline becomes calcified in the tooth, generating the characteristic stains. The discolorations are embedded in enamel and dentin. Children are susceptible to tetracycline discolorations from the time they are in utero until the age of 8. Because tooth development starts prior to birth, women are advised to avoid taking tetracycline during pregnancy. Tetracycline stains are obvious and were viewed as always permanent in the past. However, several studies reported successful cases after application of a walking bleach technique[2,4,7,11,58,82,126,230] and even prolonged bleaching with carbamide peroxide.[111,121,134,156,204,227] The walking bleach technique will, however, require intentional devitalization and endodontic treatment of the tooth to allow for the application of the bleaching agent into the pulp chamber.[2,4] A patient must be thoroughly informed about the risks, advantages; and disadvantages of this treatment.[2,4] Today, "power bleaching" and veneers are among the suggested treatments for tetracycline stains. Nevertheless, great care must be taken to avoid issues with bonding systems after the application of bleaching agents. Severe cases of tetracycline discoloration may require full coverage restorations.

Local Intrinsic Causes

Pulpal Hemorrhage

Dental trauma may lead to the rupture of intrapulpal blood vessels with subsequent intrapulpal hemorrhage and the release of blood components into the dentinal tubules.[12] The hemolysis of erythrocytes will result in the degradation of hemoglobin into globin and the heme protein, containing an iron atom.[145] The iron, in the form of iron sulfides, may reach dentinal tubules, causing stains and discolorations in the surrounding dentin. Depending on whether the pulp recovers or becomes necrotic, the discoloration may revert or persist. If the pulp survives, the tooth may eventually revert to its initial shade; however, in situations where pulp necrosis takes place, the discoloration may remain and become worse. Intracoronal bleaching has proved effective in these situations.[60,184,232]

Pulp Necrosis

Irritation of the dental pulp can happen chemically, mechanically, or by microbial insult, especially by bacteria and their toxic by-products. After initial inflammation, the pulp develops local microabscesses followed by tissue necrosis and eventually complete necrosis. Disintegration products from pulp necrosis may become incorporated into dentinal tubules and cause discoloration of the surrounding dentin. The intensity of the discoloration appears proportional to the time the discoloring agents remain in the pulp chamber. These types of discolorations tend to respond favorably to intracoronal bleaching.[174]

Pulp Tissue Remnants

Remnant pulp tissue that was left behind after endodontic therapy will eventually disintegrate and may discolor tooth structure. As a clinical consequence, pulp horns and all other pulp tissues in the access chambers should be carefully removed during treatment.

Restorative Materials

Metallic filling materials, such as amalgam or gold, may cause discolorations. Gold fillings—for example, gold foil compaction fillings, inlays, or onlays, but also pins or posts—mostly cause color changes, reducing tooth translucency and adding dark hues through thin remaining tooth portions. Amalgam fillings, over time, will undergo corrosive changes and degradation, with by-products causing color alterations in tooth structure.[157] For any metallic filling that remains visible through existing tooth structure, removal of the filling and exchange with an aesthetic filling material is the preferred choice. Effective dentinal discolorations by amalgam can be bleached but may be prone to recurrence. Composite resin restorations may leak at the margins and allow for discoloring agents to penetrate into the tooth and dentinal tubules.

Intracanal Medicaments and Root Filling Materials

A variety of endodontic medications and filling materials may be responsible for discolorations. Silver points, historically used for root fillings, can cause gray, dark discolorations in teeth and the surrounding tissues due to corrosive processes.[3,29] Gutta-percha was reported to cause a light pink discoloration.[164,225] Resorcinol-formaldehyde resin paste ("Russian Red") was reported to cause discolorations ranging from pink to dark burgundy.[146,189] Phenols or iodine containing intracanal medications (e.g., camphorated monochlorophenol [CMCP] or iodine-potassium-iodine [IKI]) may reside in the root canal space and start gradual discolorations by penetration of dentinal tubules through oxidation.

Endodontic sealers, particularly those containing metallic compounds (e.g., silver), can cause dark discoloration, either if the material is incompletely removed from pulp chambers and access cavities or because some components interact with moist dentine over time.[8,163,229] For example, AH26 (Dentsply De Trey, Konstanz, Germany), an epoxy resin cement, contains bismuth trioxide as a filler and radiopacifier. Within dentin, various bismuth compounds that range in color from green to black may occur and cause discoloration.[229] The corrosion of silver in the sealer may cause a gray to black discoloration.[8,49] In addition, incomplete removal of AH26 can cause discoloration in reaction with intracanal medicaments.[208] Epiphany (SybronEndo, Orange, CA), an adhesive root canal cement, showed signs of tooth discoloration.[191]

Mineral trioxide aggregate (MTA), a dental material known for excellent biocompatibility, is used for apexifications, perforation repair, root-end filling, and revascularization procedures. MTA may discolor teeth and adjacent soft tissues.[21,26,27,101,106,155,198,233] The discoloring effects were shown for both gray and white formulations.

Antibiotic pastes in various combinations are used to initiate revascularization of immature necrotic teeth. In particular, use of the triple antibiotic combination of metronidazole, minocycline, and ciprofloxacin has produced cases of dark discolorations of hard tissue structure.[112] Minocycline, a tetracycline derivate, binds calcium ions of the root dentin and forms an insoluble complex. Other medications shown to induce discolorations include corticosteroid preparations[113] or formocresol and iodoform-based medications.[78,123] Repeated applications of formocresol will penetrate dentin and cementum, particularly in the teeth of younger patients.

Discolorations may also occur due to irrigation solutions. Sodium hypochlorite can discolor dentin based on its destructive effect on erythrocytes and the ability to crystallize on the dentin surface.[78] In contact with chlorhexidine, sodium hypochlorite forms a dark brown precipitate containing parachloroaniline, which can only be removed by mechanical action.[19,20,96,117,226] MTAD (a mixture of a tetracycline isomer, citric acid and a detergent; Dentsply Tulsa Dental, Tulsa, OK) can cause brown discolorations,[214] probably caused by dentinal absorption and release of the doxycycline.

Dental Caries

Progressing caries can cause tooth discoloration. Early stages of caries are characterized by white, opaque enamel lesions. If caries arrests, the lesion may darken by taking up pigments from exogenous sources, frequently rendering it a deep dark brown or black color.[207] Explanations for discoloration include the formation of melanin or lipofuscin; the sugar-protein reaction also known as the Maillard reaction or nonenzymatic browning[16]; melanin[116] or food dye[110] uptake into the lesion, or compounds diffusing ahead of the bacterial penetration of demineralized dentine. Dietary chromogens entering the dentine is seen at least as a co-staining factor facilitated by the increased porosity of the hard tissues by the carious process.

Calcific Metamorphosis/Dystrophic Calcification

Calcific metamorphosis can be caused by trauma, resulting in obliteration of the pulp with mineralized tissue. Odontoblasts can become destroyed by the traumatic impact and replaced by cells from adult stem cell populations in the pulp. These cells may initiate rapid deposition of reparative dentin, resulting in yellowish or yellow-brownish discolorations. Anterior teeth are mostly affected. The reparative dentin may occupy the entire pulp chamber, and in certain cases also major parts of the root canal system, resulting in a loss of translucency of the crown. Depending on whether or not the remaining uncalcified portions of the pulp remain vital, endodontic therapy may be indicated, yet difficult to execute, depending on the extent of the calcification.[232] Dystrophic calcification involves the formation of foci of calcification frequently found in the aging pulp, usually in perivascular or perineural locations.

Root Resorption

Cervical root resorptions can lead to pink discolorations of teeth. The granulation tissue of the resorptive defect may be visible through a thin dental enamel layer, giving the characteristic "pink spot" appearance. Treatment involves the removal if the granulation tissue, etching with trichloroacetic acid to remove tissue remnants, and an adhesive restoration of the defect. The treatment may involve a surgical exposure of the resorption or, in severe cases, extraction of the tooth.

Aging

Aging-related tooth discolorations are the result of a physiologic process attributed to the uptake of discoloring agents into the hard tissue structure over time.[227] Age-related changes such as incisal wear, craze lines, or cracks allow for easier penetration. Furthermore, over time, enamel becomes thinner, and the change in ratio between dentin and enamel structure results in additional optical darkening of the teeth. Extracoronal bleaching can partially whiten age-related stains.

Extrinsic Causes

Extrinsic stains are food-, beverage-, or smoking-related superficial stains and discolorations. Pigments from beverages such as coffee or tea or tar from smoking may cause dark, brownish discolorations. Extensive consumption of oranges, carrots, licorice, or chocolate may lead to food-related stains. In areas with diverse populations, betel chewing and areca nut use may be prevalent. In particular, people with origins in Southeast and East Asia, India, Nepal, Bangladesh, Sri Lanka, Pakistan, and parts of China, Melanesia, Taiwan, and the Pacific Islands may be affected. Consistent chewing of betel nut and related substances may cause large-scale black discolorations. Discoloring agents are also included in marijuana. The effects of staining may be accelerated or enhanced if demineralization from acidic food or poor oral hygiene creates rougher tooth surfaces. In general, extrinsic stains respond well to scaling, polishing, or bleaching.

TREATMENT PLANNING FOR INTERNAL BLEACHING

The etiology of tooth discolorations may vary greatly. The outcome of bleaching procedures is also very dependent on the nature of the discolorations. Prior to any bleaching procedures, the situation must be carefully assessed and potential bleaching outcomes discussed with the patient, together with the patient's expectations.[74] If bleaching procedures are indicated, they should be integrated into the overall treatment plan.[194] The referring dentists may have to be made part of the conversation, if applicable. To establish a correct diagnosis, the patient's dental and medical histories need to be assessed, including

family history, genetic or systemic disorders, past and current medications, as well as any history of dental or facial trauma or orthodontic, restorative, and endodontic treatment. Radiographic analysis, including periapical and bitewing radiographs (as well as, if applicable, CBCT) is indicated. The assessment of the pulpal and periapical status is a prerequisite. Particular to treatment planning bleaching procedures, the dentition must be evaluated for caries prevalence and type, plaque control, and tooth surface stains, as well as the type, quality, and extent of existing restorations. Transillumination may aid in finding dental decay or pulp chamber calcifications.

CONTRAINDICATIONS OF BLEACHING

Tooth bleaching is not indicated for every situation and every patient. A thorough investigation and diagnosis of the patient's history and clinical situation may surface issues with the underlying stain that imply that a good aesthetic outcome is either impossible or difficult to achieve.[38,167] The patient's aesthetic expectations should be critically evaluated and the prognosis for the treatment discussed thoroughly. This includes potentially unrealistic expectations by the patient, either due to the clinical facts or the patient's over-expectations. The clinician should be aware of any psychological or emotional problems that a patient may have with the current clinical situation and should advise patients if a bleaching procedure is not in their best interest. All findings and discussions with the patient should be well documented for legal purposes and future reference.

The patient must be made aware of additional dental procedures, such as restorative, periodontal, endodontic, or orthodontic treatments that may be necessary to achieve a desired aesthetic goal.[167,194] Bleaching of teeth with white[23,76] or brown spot[109] discolorations could increase the existing contrast.[201] Teeth with existing decay or discolored or leaking older restorations may require technique variations[53] or new restorative procedures to remove diseased hard tissue structures to match the adjacent tooth aesthetically.

Patients with confirmed severe attrition, abrasion, abfractions or erosion, caries, or insufficient restorations may experience dentin hypersensitivity prior to a bleaching procedure. This sensitivity could be strongly enhanced by an in-office or at-home external bleaching procedure, particularly if patients can control the amount of bleaching agent used. Clinicians are advised to resolve the underlying issues prior to engaging in tooth whitening procedures. Patients with suspected or confirmed bulimia should not receive bleaching procedures. The underlying psychological condition must be resolved prior to treatment. Most patients suffering from bulimia require veneers or full coverage restorations.

If orthodontic treatment with fixed orthodontic appliances is scheduled, no bleaching procedures should take place 2 to 3 weeks prior to any adhesive placement of brackets.[201] The oxygen released from the peroxide compounds will interfere with the bonding strength of the composite resins to hard tissue structure.[53,153]

ALTERNATIVES TO BLEACHING

Microabrasion is an alternative to bleaching with peroxide compounds for superficial irregularities and shallow intrinsic stains in the enamel surface. Briefly, the technique involves mixing 18% hydrochloric acid solution with fine pumice powder to form a thick paste and applying it to the stained tooth surface. A rotating rubber cup in a slow speed handpiece with light pressure is used to distribute the paste. Liquid or sheet rubber dam placement is mandatory. Prior to using the rubber cup, the paste should be placed on the tooth for about 5 seconds, followed by the rubber cup activation for 15 seconds. While the enamel is carefully monitored for any damages, this procedure is repeated for up to 12 to 15 times, depending on the effectiveness of the stain removal. If the stain is not fully removed after this many applications, the procedure should be terminated to avoid permanent enamel damage. Copious irrigation with water is used to remove excessive paste material. The application of topical fluoride for several minutes concludes the procedure.[170] Several case reports with successful long-term follow-up have been reported.[199]

CHEMISTRY OF BLEACHING

The most common classification for tooth discoloration was described by Nathoo[157] and defines three classes:

Nathoo type 1 (N1): The discoloring agent (chromogen) binds to the tooth surface, with a color similar to that of dental stains caused by chromogenic bacteria, coffee, tea, wine, and metals.

Nathoo type 2 (N2): The discoloring agent is a Nathoo type 1 food stain that changes color to darker after binding to the tooth surface for a certain amount of time.

Nathoo type 3 (N3): A prechromogen or in its base state colorless material binds to the tooth and causes a stain after a chemical reaction. These types of stains are often due to carbohydrate-rich foods (e.g., apples, potatoes), stannous fluoride, or chlorhexidine.

Tooth bleaching involves the alteration of light-absorbing or light-reflecting properties of enamel and dentin stains, resulting in apparent whitening. In-office bleaching, internally and externally, has been practiced since the early 20th century, employing peroxide compounds. At-home bleaching procedures were only introduced in the 1980s. Historically, a variety of bleaching materials has been tried, including substances such as oxalic acid or chlorine.[23,72,157] Requirements for active tooth bleaching ingredients include a quick reaction with the stain components, fewer reactions with oral hard and soft tissues, ease of use, and an acceptable shelf life. Alkaline bleaching solutions may cause less demineralization damage than acidic solutions and are less likely to penetrate the pulp.[64,65] Bleaching materials should cause as minimal oxidative damage to tissues as possible[59,178] (Fig. 27-1).

Contemporary bleaching techniques rely on oxidative processes that actively break down pigmenting molecules. Oxidation is initiated by active peroxide compounds.[73,83] Peroxides and oxygen radicals are reactive free agents that are formed in all body cells that use oxygen. Reactive oxygen is, however, usually only found in low concentrations. Natural antioxidants, such as vitamin E, prevent the accumulation.[34,137] Cell damage may occur if reactive oxygen reacts with iron or copper ions.[59]

The active peroxides in concurrent bleaching techniques primarily derive from hydrogen peroxide and carbamide peroxide for external, vital bleaching and sodium peroxide for internal, nonvital bleaching. Hydrogen peroxide (H_2O_2) is the simplest peroxide, and in its pure form is a colorless liquid of

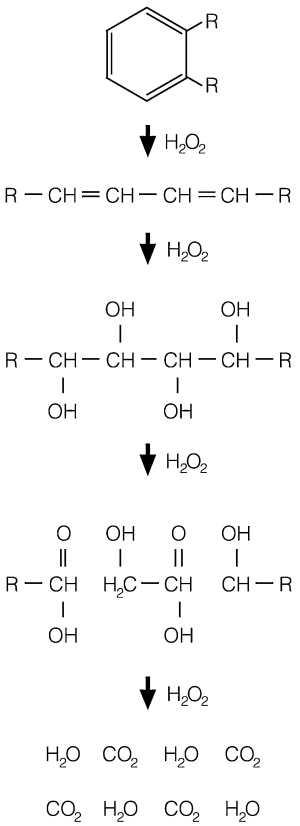

FIG. 27-1 Chemistry of tooth whitening with hydrogen peroxide. Breakdown of pigmented organic materials consisting of carbon ring structures with unsaturated, double carbon bonds to hydrophilic, nonpigmented carbon structures with saturated carbon bonds, resulting in effective whitening. Further degradation over this step may complete disintegration of the organic matrix with the release of carbon dioxide and water. (Modified from Cohen S, Burns C: *Cohen's pathways of the pulp*, ed 8, Part III, Chapter 21, Fig. 21-1. St. Louis, 2002, Mosby.)

a slightly higher viscosity than water. It can be utilized for in-office as well as at-home bleaching procedures. An increase in temperature accelerates the speed of the bleaching reaction. Hydrogen peroxide is more effective as a bleaching agent at pH values that are close to the dissociation constant.[56] Enzymes such as peroxidases can break down hydrogen peroxide into oxygen and water. High concentrations of hydrogen peroxide must be handled with great care. If accidentally brought into contact with soft tissues, it is caustic and may cause chemical burns by free oxygen radicals. For external bleaching procedures, the use of a rubber dam with additional blockout isolation around the gingival margins is mandatory to avoid iatrogenic complications. Typically, at-home concentrations range from 3% to 7.5%; however, some exceptionally strong concentrations can reach 14%. In-office concentrations may range from 25% to 38%. Hydrogen peroxide was shown not to induce significant changes in the organic and inorganic relative contents of enamel, but to whiten teeth by oxidizing the organic matrix.[54]

Carbamide peroxide ($CH_6N_2O_3$), or urea hydrogen peroxide, will break down into carbamide and hydrogen peroxide in aqueous solution.[157] It is an efficient bleaching agent.[223] The carbamide portion urea is well tolerated by the human body.[180] Carbamide peroxide is available as crystallized powder or white crystals that contain H_2O_2 in a concentration of about 35%. Most common home bleaching products contain carbamide peroxide at about 10% strength, but it can reach up to 30% (equivalent to 3.5% to 8.6% hydrogen peroxide). Carbamide peroxide can be used for internal bleaching.[223] At 35%, it shows very low extraradicular diffusion rates compared to hydrogen peroxide and sodium perborate.[128,129,141] Common bleaching preparations may contain glycerin or propylene glycol, sodium stannate, phosphoric or citric acid, and flavor additives. Carbopol, a water-soluble polyacrylic acid polymer, may be added as a thickening agent. It prolongs the release of active peroxide. At room and oral temperatures, the reaction of carbamyl peroxide takes place at a slower rate compared to hydrogen peroxide.[85] The release of oxygen from hydrogen peroxide occurs within seconds of contacting tooth surfaces, whereas carbamide peroxide remains active for 40 to 90 minutes.[157]

Sodium perborate ($NaBO_3$) comes in different preparations, mostly as a stable, dry powder or as a gel. It is available as a monohydrate, trihydrate, and tetrahydrate, with varying contents of oxygen. Depending on the oxygen content of the preparation, the compounds have a different bleaching efficiacy.[234] The perborate ion comprises 95% of the molecule and provides close to 10% of the available oxygen. Acids, water, and warm air initiate its decomposition to sodium metaborate, hydrogen peroxide, and nascent oxygen. The pH of sodium perborate solutions used for dental bleaching is alkaline. Because sodium perborate can be more easily controlled, it is considered safer to work with than hydrogen peroxide and more popular for intracoronal bleaching.

In an aqueous solution, due to heat or in an acidic environment, both carbamide peroxide and sodium perborate decompose and release hydrogen peroxide. Hydrogen peroxide then diffuses through enamel and dentin. Subsequently, the oxidative process provides for lightening of stains in comparison to the surrounding tooth structure. Double bonds of complex molecular chains with high molecular weight pigments are broken down by the chemical reactions with the free radicals. As a consequence, the change in pigment configuration and size leads to a change in the wavelength of the reflected light and the change of the stain to a lighter color.[64,65,157] In a best-case scenario, this leads to an effective reduction or elimination of the discoloration. However, after the bleaching process a shade rebound effect may be observed, resulting from a reformation of double bonds of the molecules.

Walking Bleach Technique

The walking bleach technique was described early on as leaving a mix of sodium perborate and distilled water in the pulp chamber for a period of several days, with the access cavity sealed with a temporary restoration.[186,196] Later on, the use of 30% hydrogen peroxide instead of water was suggested to provide better bleaching effectiveness.[161] The mix of sodium perborate with water or, alternatively, anesthetic solution remains the most commonly used technique for internal bleaching of nonvital teeth. The enhancement of the mixture with 30% hydrogen peroxide is being used less due to concerns of cervical root resorption but remains an option for stains resistant to whitening that require stronger chemical compounds to achieve good bleaching results.[15,24,152,162,190]

Walking Bleach Clinical Protocol

1. Prior to initiating the bleaching procedure, a diagnosis must be made following thorough evaluation of the etiology and the origin of the stain. The patient must be informed about the history, the procedures involved in the treatment, the expected outcome of the procedure, and the potential for rediscoloration.
2. The endodontic status of the tooth must be assessed clinically and radiographically. In the case of symptoms, the presence of periapical pathology, or an insufficient root filling, retreatment of the existing endodontic filling is advised, unless the apical periodontitis is on a tooth with a history of recent, well-executed root canal treatment or is verifiably in the process of healing.
3. Existing coronal restorations need to be checked for quality and shade and replaced if defective. In certain situations, the reason for discoloration may be a leaking restoration or discolored filling materials. Replacement of a defective restoration and cleaning of the access chamber may resolve the discoloration.
4. Clinical photographs should be taken at the beginning, throughout, and at the end of the treatment with the tooth next to a shade guide to act as a reference for the practitioner and the patient.
5. Rubber dam isolation is mandatory. It is important to provide a seal at the gingival margin to prevent leakage of bleaching agents into the surrounding soft tissues. Depending on whether a single tooth or multiple teeth are being isolated, this may involve the placement of clamps, wedges, ligatures, floss, or light curing blockout material. Local or topical anesthesia may be applied if the patient is sensitive to the discomfort exerted during the isolation procedures.
6. The access cavity needs to be prepared so that all restorative filling materials, root-filling material and sealer remnants, and necrotic pulp tissues remnants are completely removed. This procedure requires good visualization through cavity refinement and the elimination of all undercuts, especially in the area of pulp horns. The practitioner should exercise care to avoid labial perforations.
7. Materials should be removed to a level just below the labial gingival margin. Solvents such as xylene, eucalyptus oil, or orange solvent may be used to help clean the pulp chamber. Also, rinsing the cavity with sodium hypochlorite was suggested for cleanliness and disinfection or, alternatively, with alcohol to reduce surface tension.[15] Although some studies have recommended cavity treatment with 37% phosphoric acid or EDTA (ethylenediaminetetraacetic acid) to allow for deeper penetration of the bleaching agents, others have suggested that phosphoric acid was unnecessary and offered no better prognosis and no improvement in the bleaching effectiveness of either sodium perborate or a high concentration of hydrogen peroxide.[15,35] Acidic pretreatment may increase the risk of adverse effects on the periodontium.[97]
8. A protective layer of at least a 2-mm thickness should be placed over the root-filling material. To avoid further pigmentation the protective layer should be white or tooth colored. Without this protective layer, bleaching materials may penetrate the root filling toward the apical foramen.[45,80,195] Materials that had been suggested include glass-ionomer cements, intermediate restorative material (IRM), Cavit and Coltosol, resin composites, photoactivated temporary resin materials such as Fermit, zinc oxide–eugenol cements, polycarboxylate cements, and zinc phosphate cements.[197] Cavit and IRM provide better sealing than zinc phosphate cement,[148] and Cavit offers a better leakage barrier than IRM.[80] Cavit and Coltosol also provided the most favorable seal when tightly compacted and proved to be superior to Fermit.[98] A protective barrier at the cementoenamel junction (CEJ) will furthermore reduce the risk of cervical root resorption and damage to the periodontal ligament.[197] Ideally, the vertical outline of the protective barrier should be 1 mm coronally to the CEJ to cover lingual, labial, and proximal CEJ areas.[185] Clinically, however, this may be difficult to achieve in confined spaces. A barrier level with the labial CEJ will leave a substantial area of proximal dentinal tubules unprotected. Clinically, Cavit is also easily removable using ultrasonic instruments and chlorhexidine, thus minimizing the amount of tooth structure loss. Glass-ionomer cements may remain in place after the bleaching procedure is completed and do not need to be removed (Figs. 27-2 and 27-3).
9. Sodium perborate (tetrahydrate) is mixed with an inert liquid (distilled water, saline, or anesthetic solution) to achieve the consistency of wet sand. Mixing the paste instead with hydrogen peroxide is an option to accelerate the speed of the bleaching process, yet this technique is risky due to the increased possibility of cervical root resorption. If it is required, due to the severity of the stain, it should be used at 3% strength,[161] and it should be refrained from a 30% solution. The long-term results of a sodium perborate and water mix do not significantly differ from mixes with hydrogen peroxide.[95,180,184] A plastic instrument or amalgam carrier can be used to place the material in the pulp chamber. It is then carefully

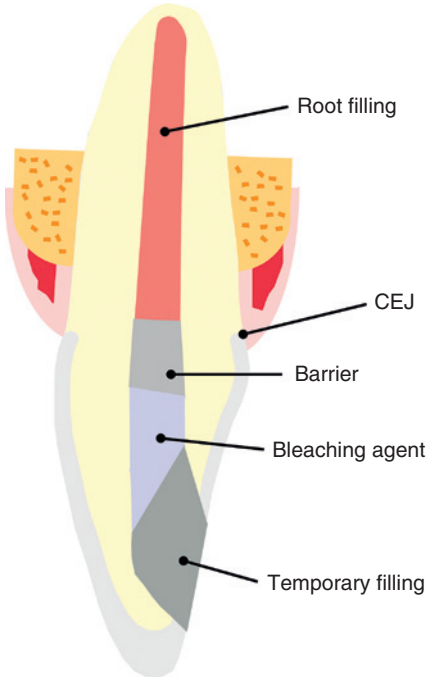

FIG. 27-2 Walking bleach technique: Placement of barrier material at the CEJ level, bleaching agent, and coronal seal with temporary filling material.

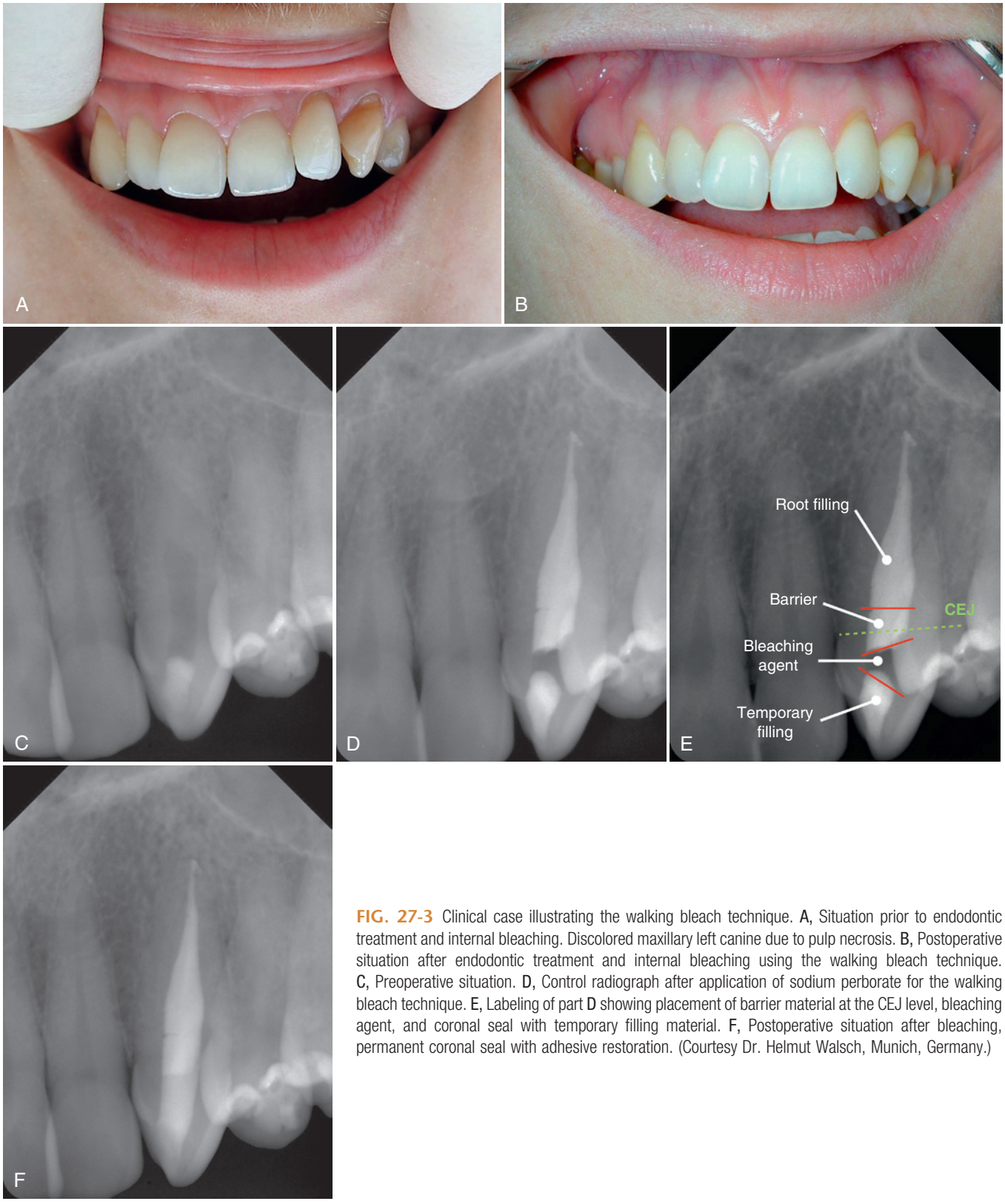

FIG. 27-3 Clinical case illustrating the walking bleach technique. **A**, Situation prior to endodontic treatment and internal bleaching. Discolored maxillary left canine due to pulp necrosis. **B**, Postoperative situation after endodontic treatment and internal bleaching using the walking bleach technique. **C**, Preoperative situation. **D**, Control radiograph after application of sodium perborate for the walking bleach technique. **E**, Labeling of part **D** showing placement of barrier material at the CEJ level, bleaching agent, and coronal seal with temporary filling material. **F**, Postoperative situation after bleaching, permanent coronal seal with adhesive restoration. (Courtesy Dr. Helmut Walsch, Munich, Germany.)

FIG. 27-4 Clinical case illustrating clinical steps of the walking bleach technique. **A,** Situation prior to endodontic treatment and internal bleaching. Discolored maxillary left central incisor due to dental trauma. **B,** Postoperative situation after endodontic treatment and internal bleaching using the walking bleach technique. **C,** Access cavity after barrier placement. **D,** Application of sodium perborate. **E,** Temporary coronal seal with adhesive restoration. (Courtesy Dr. Mohammed Al-Harbi, Philadelphia, PA.)

condensed with a plugger, without missing areas such as pulp horns (Fig. 27-4).

10. Excessive bleaching material should be removed, the paste carefully dapped with a cotton pellet without removing too much moisture, and a temporary filling material that is ideally 3 mm thick with well-sealing margins placed (Cavit, 3M ESPE, St. Paul, MN). Though some have advocated the use an acid-etched adhesive as a temporary filling to avoid recontamination, restaining, and to offer the best possible prevention from leaking walking bleach,[228] this may not be practical in small cavities where it becomes difficult to selectively etch the enamel without the risk of opening dentinal tubules for hydrogen peroxide and the subsequent risk of cervical resorption. Moreover, because this procedure would have to be repeated at every visit, it would add to the amount of tooth structure loss. Any walking bleach remnants on the external tooth surfaces or the rubber dam isolation need to be removed to avoid irritation of the soft tissues.
11. The rubber dam is removed and all tissues inspected.
12. The patient should be informed about the procedure. Depending on the reaction of the stain to the bleaching agent, the time to expect results may vary. Usually, a patient should be instructed to return between 3 to 10 days to evaluate the results and to change the walking bleach if necessary. To avoid overbleaching,[15] the patient needs to be instructed to self-monitor the color status of the tooth and to report back earlier than the scheduled appointment date if the color change has reached the desired result or if the temporary filling material is starting to wash out.

Although a rarely documented event, it was advocated to instruct the patient about an increased susceptibility to tooth fracture in the time that the tooth is filled with a bleaching agent and not a supportive buildup.[17]

13. If desired results cannot be obtained, a mix with 3% H_2O_2 can be considered. However, the patient needs to be informed about the risks and that most good bleaching results can be accomplished by a sodium perborate and water/anesthetic mix.[184] If the aesthetic outcome with the results is acceptable, a potential for resorption may be minimized.
14. If a clinical case does not show improvement after three to four attempts, the diagnosis and treatment plan should be reevaluated for a different etiology.
15. At the conclusion of the bleaching procedure, a postoperative radiograph should be taken to check for any adverse effects of the bleaching agents. A permanent adhesive restoration should be placed about 1 to 3 weeks after the last appointment[36,192,193,221] (see next section, Permanent Restoration of Teeth Following Internal Bleaching).
16. The patient should be scheduled for yearly follow-ups to include a clinical examination and periapical radiographs.[42]

Thermocatalytic Bleaching

Heat-activated Superoxol had been advocated for its increased bleaching effect for internal bleaching of endodontically treated teeth.[99] Hydrogen peroxide with a concentration of 30% to 35% is placed in the pulp chamber and activated via heat application with electric heating devices or special lamps.[24,25,32,100,118,145,206] All preparatory steps follow the clinical protocol described previously for the walking bleach technique. However, in lieu of placing a sodium perborate/water mix, the pulp chamber is filled with 30% to 35% hydrogen peroxide and a heating device applied (e.g., Touch'n Heat, System B; SybronEndo, Orange, CA; SuperEndo Alpha, B&L Biotech, Fairfax, VA). The heat application will create foaming of the hydrogen peroxide, with a subsequent release of free radical oxygen. This procedure is repeated in three to four office visits. In addition, some protocols advocate the placement of 30% to 35% hydrogen peroxide in the pulp chamber between the chair-side appointments.[40,143,235] Temporary filling placement follows the same steps as outlined earlier for the walking bleach technique. If this technique is used, great care needs to be taken to avoid overheating of teeth, periodontal ligament, and gingival tissues. Regular cooling breaks are recommended. Products such as Vaseline, Orabase, or cocoa butter have been suggested for additional thermal insulation. However, due to the increased risk of cervical root resorption when using this clinical technique, the walking bleach technique is seen as more favorable today. If a decision is made to apply the thermocatalytic technique, the patient must be thoroughly informed about the risks and potential long-term consequences.

PERMANENT RESTORATION OF TEETH FOLLOWING INTERNAL BLEACHING

A good permanent restoration is one of the foundations of long-term success, as it prevents coronal microleakage from the access cavity and avoids the risk of renewed discoloration through open dentinal tubules.[17,238] It is generally accepted that an acid-etched and bonded composite resin restoration provides the most favorable results. For preexisting resin composite restorations, a rebonding of the filling has been suggested to reduce the risk of microleakage at the dentinal margins after bleaching with 15% carbamide peroxide.[151] The restoration must be placed at a depth that provides both an adequate seal for the access cavity as well as incisal support. The placement of a white base material below the resin restoration was viewed favorably in situations where the composite resin restoration may compromise the translucency of the tooth. Lighter shade composites were suggested for teeth, with a not entirely satisfactory bleaching result.[4,70] Consensus exists that the adhesive bond strength between glass-ionomer cements and composite resins to dentin and enamel is temporarily compromised following bleaching procedures[52,67,104,210-212] by remnants of peroxide or free oxygen, which inhibit resin polymerization.[52,215] Several remediation techniques have been suggested to overcome the negative influence of hydrogen oxide–containing bleaching agents, including dehydrating agents such as 80% alcohol and the use of acetone-containing adhesives,[105] the application of sodium hypochlorite to dissolve remnants of peroxide,[180] catalases,[173] the antioxidant sodium ascorbate,[107,125] or the use of alpha-tocopherol.[236] At least 1 week of contact with an aqueous solution was recommended to achieve adequate bonding.[5,210,216] Optimal bonding to bleached dentin and enamel was shown to be reestablished after 3 weeks.[36,193] Calcium hydroxide medication in the pulp cavity was advocated to achieve a buffering effect for an acidic pH that may be present after internal bleaching[17,108]; however, it may be unnecessary after a walking bleach technique, due to the weaker nature of sodium perborate compared to Superoxol. Light curing of the composite resin restoration should occur from labial so that shrinkage toward the axial walls can reduce a potential risk of microleakage.[130]

EXTERNAL BLEACHING

External bleaching may involve single teeth or entire arches. Whereas internal bleaching requires endodontic treatment of the tooth, external bleaching can also be applied to vital teeth. Steady advancements have been made in external bleaching techniques since its inception.[87,102] Various techniques exist, including in-office and at-home methods.

In-Office External Bleaching

The in-office or chair-side techniques are completely in the hand of the dental professional. Almost all techniques involve the application of hydrogen peroxide gels of concentrations between 25% and 38%. Liquid solutions at higher concentrations are associated with higher complication rates of soft tissue damage, as they are more difficult to control in terms of handling. Furthermore, high concentrations of hydrogen peroxide solutions are thermodynamically unstable and may explode if not stored in dark bottles in a refrigerator.

Although early techniques also employed heat activation, electric currents, or combinations with other chemicals to make hydrogen peroxide more effective,[136] contemporary techniques commonly use bleaching gels applied on their own or in combination with light activation by special lamps. Patients should undergo a hygiene treatment with cleaning and polishing of the tooth surfaces prior to external bleaching, as well as a careful evaluation of the nature of the stain or the possibility that the discoloration is related to an existing restoration.

If external bleaching is indicated, the treatment involves the placement of a rubber dam, as described in the protocol for the walking bleach technique. Great care must be taken to avoid soft tissue damage. If liquid hydrogen peroxide solutions and no gels are used, the additional placement of gauze to act as a reservoir for hydrogen peroxide is recommended to keep the solution in the proximity of the teeth. More gauze can be placed underneath the rubber dam as an additional safety to catch excess solution before it can damage gingiva, mucosa, or lips. However, these complications can be greatly reduced by a gel application with or without light activation.

Light sources used for bleaching include conventional ultraviolet (UV) bleaching lights, tungsten-halogen and Xe-halogen lights, plasma arc lamps, light-emitting diodes (LED), or laser lights. The wavelengths of the various light sources may range from the high ultraviolet to low visible blue light spectra, and invisible infrared light, such as employed by a CO_2 laser.[32,172] Although all of these lights have been used to activate the bleaching agent or expedite the whitening effect, there are great differences among them. Limited research on bleaching lights exists, with sometimes controversial results. Although some studies emphasized certain effects after light-activated bleaching, others reported no differences between teeth bleached with and without light activation.[32,122,172,203] In the 1980s, the Fuji HiLite dual-cure material containing 35% hydrogen peroxide was used. When the paste was mixed, it had a green color that would turn white upon activation with a standard UV light upon the release of oxygen. In comparison to other light activation techniques, this was a more time-consuming process and less comfortable for the patient. In addition to light activation alone, various photocatalysts were suggested, including methylene blue and ultraviolet or visible light-activated titanium dioxide.[200]

Tungsten-halogen curing lights are the standard dental curing lights that provide heat and stimulate the initiation of the chemical reaction by activating light-sensitive chemicals in the bleaching agent. Depending on manufacturer recommendations, the individual bleaching time with activation of the gel is 30 to 60 seconds per application per tooth, which is rather time consuming for the patient and the dental professional because up to three passes are recommended. Some products available were based on a premixed 35% hydrogen peroxide gel with carotene that converts light energy to heat upon activation and increased the breakdown of the bleaching agent into active free radicals. Xenon-halogen curing lights are newer generation dental curing lights used to the same effect. Some specialized bleaching lights offer full-arch adaptors. A randomized clinical trial of in-office tooth whitening identified blue light–activated bleaching as the one technique with results most favored by patients.[9]

Xenon plasma arc lamp systems are primarily based on thermal activation and the activation of chemical catalysts in the bleaching gel. The light employed is of high intensity, produces great heat energy, and limits the application to 3-second intervals for up to three passes. Due to the high heat intensity, there is a potential for dental trauma to the pulp and the surrounding tissues, which is why these lights have to be used with great care. Some manufacturers produced lights that allowed an on-off mode setting over 5-second bursts to have a safer and lower-intensity output.

LED lamps emit cold blue light with a wavelength of around 465 nanometers, which allows activation of hydrogen peroxide gels and accelerates the bleaching process. LED lights are placed close to the patient's teeth for 15- to 20-minute treatments, which may have to be repeated up to three times. Cosmetic specialists with no dental background have adopted this method of bleaching because it offers low risk, as no heat activation is involved.

Lasers are used for tooth bleaching at 830 nm and 980 nm wavelengths for tooth bleaching in combination with 30% to 35% hydrogen peroxide gel. Gels prepared for laser activation may contain fumed silica and a blue dye, the latter absorbing the laser wavelength leading to an increase in temperature and a controlled breakdown of the hydrogen peroxide. Gels are commonly applied in a 2- to 3-mm thickness over the teeth. An application of 1 to 2 W of laser energy for 30 seconds per tooth is recommended. Dental professionals and patients are required to wear protective eyewear while operating lasers. Some studies reported no significant advantage to using laser light for bleaching,[32] whereas others showed a decrease in color regression after a combination of at-home bleaching with an in-office laser-assisted bleaching session in comparison to an at-home bleaching regimen on its own.[6]

"Power Bleaching"

The technique normally termed power bleaching refers to accelerated vital in-office tooth whitening procedures that employ either xenon plasma arc-curing lights or lasers. Either dental hygienists or dental assistants may carry out this procedure, depending on individual licensing issues state by state. In particular, the use of laser lights might be more strictly regulated and may not be allowed to dental professionals other than the dentist. For a delegated procedure, liquid rubber dam is often applied in lieu of a sheet of rubber dam, which would require active retention techniques, such as wedges, floss, or ligatures. Liquid rubber dam can be applied easier without assistance, but it does not give additional protection to gingival tissues or the mucosa.

Power Bleaching Clinical Protocol

1. Prior to initiating the bleaching procedure, a diagnosis must confirm that the discolorations can be resolved by external bleaching. The patient must be informed about the history, the procedures involved in the treatment, the expected outcome of the procedure, and the potential for rediscoloration.
2. A thorough clinical examination should be performed to detect caries, developmental defects, endodontic or periodontal diseases, and other pathologic conditions in the oral cavity. Existing restorations need to be checked for quality and shade and replaced if defective. For certain teeth, the reason for discoloration may be leaking restorations or discolored filling materials. Replacement of a defective restoration may resolve most of the discoloration.
3. Patients should have a hygiene appointment prior to the bleaching session to check for sound gingival tissues and ensure that areas to be bleached are free of calculus or plaque.
4. Clinical photographs should be taken in the beginning, throughout, and at the end of the treatment with the tooth next to a shade guide to act as a reference for the practitioner and the patient.

5. The patient and dental professionals should wear proper protective eyewear (sunglasses). The patient should be prepared to signal if a tingling sensation to the gingiva, mucosa, lips, or teeth is felt or the temperature is perceived as too hot. As a precaution, vitamin E capsules, a powerful antioxidant, should be ready at hand. In case of an emergency, these can be cut open and the oil inside the capsules applied to the injured tissues with a cotton pellet.
6. The teeth should be cleaned again with rubber cups and pumice. The cheeks should be retracted with photo retractors or cotton rolls. The cotton rolls serve as an additional safety feature if any hydrogen peroxide is leaking. Rubber dam isolation is mandatory. This can be either liquid rubber dam (light-cured resin dam) around the gingival margins or ligated sheet rubber dam. Liquid rubber dam bears a higher risk of tissue injury. It is important to create a tight seal at the gingival margin to prevent leakage of bleaching agents. If a sheet rubber dam is used, the inversion of the rubber dam in the sulcus with an air syringe followed by dental floss ligature is recommended.
7. The power bleach gel is mixed according to the manufacturer's recommendation and applied on the labial surfaces of the teeth in a 2- to 3-mm thickness with a disposable brush.
8. Depending on the light source, each individual tooth is exposed for up to three passes for 3 to 10 s, according to manufacturer's recommendations. The gel may stay on the teeth for another 3 to 5 minutes without light activation.
9. The gel should be removed with a wet gauze and copious amounts of water, then cleaned with pumice for a second time, and rinsed again to ensure all remnants of the bleaching gel were removed.
10. The rubber dam and any remaining cotton rolls and retractors are removed. Water rinses are used again, and all tissues inspected.
11. The teeth should be polished and a neutral pH sodium fluoride gel applied.
12. The patient should be informed about the procedure. Depending on the individual situation, results may vary. The patient should be instructed that increased sensitivity of the teeth may be present for 2 to 3 days.
13. The patient ought to be instructed to refrain from tobacco, coffee, tea, cola, and wine for a period of 2 weeks.[44]

At-Home External Bleaching

For patients who do not want to undergo an in-office procedure, the possibility of at-home external bleaching with custom-made bleaching trays exist. These may be readily available from a pharmacy, requiring only crude adaptation after softening in hot water, or can be fabricated in a dental laboratory. Over-the-counter products most commonly come with bleaching gels, although some come with pastes. Some products are offered as simple, low-strength hydrogen peroxide strips or pastes that can be brushed onto the teeth. Occasionally, pre-rinses are offered. However, these may come at a rather acidic pH, as these solutions may become unstable more easily.

Professional home bleaching involves the fabrication of a well-adapted custom tray after impressions are taken and models are poured. The patient will wear gel loaded in a tray that slowly releases the bleaching agents for several hours or overnight every day for 2 to 6 weeks until a satisfactory result has been achieved.[38,83-87]

Trays are usually made using alginate impressions. Upon delivery, trays must be checked for fit, smoothness at the margins, and occlusion. The patient should place several drops of bleaching gel into the tray before every application. The most common bleaching agent for at-home bleaching is 10% to 22% carbamide peroxide with an effective yield of 4% to 7.5% hydrogen peroxide. Patients who wear the trays during the daytime may choose to replace the gel every 2 hours. If any disturbance (e.g., thermal sensitivity, abnormal taste, or tissue irritation) occurs, the patient should stop the procedure and seek advice from the dentist. Patients who use the trays in the daytime should be seen once per week for 3 weeks; nighttime users should be seen every other week for 6 weeks.

The success of at-home bleaching mostly depends on the patient's cooperation. Risks include potential compliance issues and the possibility of overuse of the bleaching agent. Results with at-home bleaching techniques tend to remain stable for 1 to 10 years.[132]

RISKS AND COMPLICATIONS ASSOCIATED WITH TOOTH BLEACHING

Risks Associated with Internal Tooth Bleaching

Tooth bleaching may cause localized adverse effects on hard and soft tissues, such as the risk of external cervical resorption, interaction with adhesive bonding systems, and dental material solubility.[10]

Cervical Root Resorption

External or cervical root resorption is a serious complication of whitening procedures with peroxide compounds. Cervical root resorptions were seen in 6% to 8% of cases when 35% hydrogen peroxide was used, and 18% to 25% if the hydrogen peroxide was heat activated.[47,62,63,81,91,124,182] The etiology for external root resorption to occur is complex.[75,81,120,124] It is suggested that a combination of predisposing factors—such as a cementum deficiency exposing dentin, a periodontal ligament injury that is triggering an inflammatory response, or an infection that is sustaining inflammation—has to be present.[17,63,160,219] Cervical resorptions are also often seen in patients who had bleaching treatment at a young age[7,11,63,91,95,124] or who suffered from a traumatic injury.[81,217]

Hydrogen peroxide itself was suggested to be responsible for the induction of the resorptive process.[81] The body can handle hydrogen peroxide effectively at lower concentrations.[79]

Proinflammatory agents activate reduced nicotinamide adenine dinucleotide phosphate oxidase, which produces superoxides that can react with hydrogen peroxide in the presence of inflammation.[77] This may allow the formation of hypochlorous acid, N-chloramines, and reactive hydroxyl ions and cause a resorptive process.[77] Hydrogen peroxide may reach the bone by diffusion via dentinal tubules, cementum, and the periodontal ligament.[77,166,231] Within the bone, the acidic environment induced by the low pH value of hydrogen peroxide at high concentrations may facilitate osteoclastic activity.[63,224] After application to the enamel surface, 25% hydrogen peroxide was also shown to cross enamel and the dentinoenamel junction (DEJ), reaching the dentin.[220]

About 10% of all teeth have a natural defect at the cementoenamel junction, exposing dentin.[205] These cementum

defects, as well as unusual morphology at the CEJ, can favor the penetration of hydrogen peroxide into the cervical area of a tooth.[119,159,182] The placement of 30% hydrogen peroxide in the pulp chamber of teeth with missing parts of the cementum layer resulted in the diffusion of 82% of the bleaching agent.[182] Dentin exposure may also be a result of defects in the cementum layer following traumatic injuries or an incomplete formation of the cementoenamel junction.[144,150] The permeability of the dentin can then facilitate the penetration of bacteria or bacterial endotoxins from an infected root canal system or pulp chambers to the surface of the root, potentially causing inflammatory processes in the periodontal ligament.[92,152] If highly concentrated bleaching agents are used, in particular, 30% to 35% hydrogen peroxide, a diffusion of highly reactive hydroxyl radicals via unprotected dentinal tubules and cemental defects was suggested.[119,182]

Free oxygen radicals are also able to break down constituents of connective tissue, in particular collagen and hyaluronic acid, which may be another likely mechanism responsible for periodontal tissue destruction and root resorptions after internal bleaching.[48] The oxidizing agents were shown to induce dentin protein denaturation,[124] either by local changes in the pH[50,69,150] or by heat.[60,81,144] Pretreatment is with EDTA, a chelating complex binding iron. Heat activation of 30% to 35% hydrogen peroxide is the characteristic step of the thermocatalytic method. It may result in widening of the dentinal tubules, allowing for a diffusion of free radicals into the dentin[165,181] and an enhancement of resorptive processes.[181] The hydroxyl radicals released from hydrogen peroxide are extremely reactive and have been shown to degrade components of connective tissue.[48] As a consequence, the thermocatalytic technique is nowadays disfavored due to the high risk of external root resorption.

The potential for cervical resorption may be minimized by using sodium perborate with water instead of hydrogen peroxide for internal bleaching techniques, because similar acceptable outcomes have been reported[184] and because it did not cause cervical resorption, even 1 year after bleaching.[144] Sodium perborate–water suspensions were also shown to be less toxic to periodontal ligament cells than 30% hydrogen peroxide or sodium perborate mixed with 30% hydrogen peroxide.[115,181] Moreover, a strong association was found between tooth discoloration related to blood components and an increased presence of hydroxyl radicals.[48]

Treatment of cervical resorption may range from simple fillings to extraction, depending on the severity of the resorptive defect. An early detection of a resorptive process increases the chances of avoiding unnecessary tooth loss, because the prognosis for cases with advanced resorptions is doubtful.[51] Evaluation by means of a CBCT may greatly enhance the ability for correct treatment planning.[55] If resorptions are related to internal bleaching, the placement of calcium hydroxide into the root canal space has been suggested[63] to change the low pH present in inflammatory osteolytic processes characteristic for hard tissue resorption.[147] Reparative hard tissue formation was suggested to take place following this treatment.[218] Some cervical resorption can be repaired with direct restorations, such as composite resin or glass ionomer–composite compomers (e.g., Geristore, DenMat, Lompoc, CA). This treatment requires a surgical exposure of the defect, followed by degranulation by curettage and the application of 90% trichloroacetic acid to remove remnant tissues prior to adhesive restorations.[61,149] Patients should be informed about the potential for gingival recession if the surgical access requires a full thickness flap in the anterior area.[55,69] Rapid orthodontic extrusion in combination with fiberotomy and restoration was suggested for resorptions with a substantial subcrestal component.[90,127]

The free radicals released by peroxides can cause chemical burns and tissue sloughing on gingiva, mucosa, or other oral soft tissues. This may occur during the internal bleaching procedure in the dental chair during both walking bleach or thermocatalytic technique appointments after a patient has left the office if a walking bleach technique is used. In any case, prevention is the best cure, and avoiding accidents is the priority. In general, the use of higher concentrations of peroxides, such as 30% to 35% hydrogen peroxide, in particular in liquid form, is more dangerous than the use of a mixed paste or gel of sodium carbamide peroxides that have lower effective hydrogen peroxide concentrations and can be used more controlled. However, in the dental office great care has to be taken so that the bleaching agents do not leak through the rubber dam and all excessive bleaching material is carefully removed after the end of the procedure (see details in Walking Bleach Clinical Protocol). For emergency situations, clinicians should have vitamin E on hand, because this tough antioxidant reverses the strong oxidation processes on oral soft tissues and minimizes damage (see details in Power Bleaching Clinical Protocol). To avoid out-of-office accidents, it must be assured that the temporary filling material is sealing tightly and is placed in an adequate thickness to cover the bleaching agent. Patients should be advised to return immediately after a temporary restoration starts to fail or wash out.

Enamel and Dentin Damage

A number of in vitro studies reported changes in enamel microhardness and morphology,[22,37,142,154,175,177,237] as well as in the cementum.[213,239] It has been suggested that peroxide components alter the proportions of organic to inorganic hard tissue components.[83,92,135,169,213] A treatment with 10% carbamide peroxide and 7.5% hydrogen peroxide did alter the enamel surface micromorphology, but not its microhardness.[187] Hydrogen peroxide demonstrated a different chemical affinity to each specific dental tissue, rather than just to diffuse through interprismatic spaces into the dentinal tubules when applied to the enamel surface.[220] It was also shown to modify the organic compounds of dentin.[220]

At least in vital teeth, however, these changes appear not to be permanent[14,68] and can be reduced by the application of fluoride before or during remineralization.[31,68] The bleached enamel surface may show an increased porosity and slight erosion when observed under scanning electron microscopy (SEM). These changes were mostly seen when acidic prerinses or bleaching gels with a low pH had been applied.[222] One study found no differences between bleached and unbleached human enamel over a 6-month period when a 10% carbamide peroxide gel was used.[89] No significant clinical evidence of negative effects on enamel by professionally administered at-home bleaching techniques was seen. A few case reports described significant enamel damage after over-the-counter products had been applied by patients, possibly the result of overuse.[46]

Inhibition of Adhesive Bonding

Polymerization and bonding strength of composite resins to dentin and enamel are temporarily compromised by the oxygen

released during a bleaching process.[13,52,202,209,211] Scanning electron microscope images have shown an increased porosity of the enamel after bleaching.[211] This may present a clinical issue in terms of leaking restorations with the potential for caries activity and rediscoloration. Several remedial solutions were suggested, including rinsing the cavity with sodium hypochlorite,[180] which, however, was demonstrated to have similar effects on bonding, alcohol,[105] chlorhexidine, catalase, and sodium ascorbate.[41,107,125] Clinicians are generally advised to wait for 2 to 3 weeks prior to placing a permanent adhesive restoration to allow time to reverse the effects and have all remnants of hydrogen peroxide removed from the pulp chamber.[41]

Risks Associated with External Tooth Bleaching

External tooth bleaching (including professional at-home tray bleaching techniques) is, generally speaking, a safe procedure that allows tooth whitening to be retained for several years.[132] Nevertheless, concerns have been raised since the introduction of external bleaching,[88,137-140] mostly related to the caustic effects of free oxygen radicals that may cause tissue damage in oral soft and hard tissues. The reaction with proteins, lipids, and nucleic acids may eventually lead to cell damage. At-home external bleaching did not demonstrate significant long-term complications if 10% carbamide peroxide was used as the bleaching agent. However, since at-home bleaching does not give the practitioner control over the patient's behavior, higher concentrations of carbamide peroxide than recommended may be used and could more likely cause damage to the gingival and periodontal tissues.[93] In general, caution is advised in regard to the quality of product, techniques used, and treatment rendered.

Tooth Sensitivity

The presence of hypersensitivity is an indication of inflammatory changes in the dental pulp.[139] Histologically, however, few pulps have demonstrated actual signs of inflammation after external bleaching with hydrogen peroxide gels.[114] Nevertheless, changes in sensitivity after external bleaching of vital teeth have been reported in up to 50% of the teeth.[88,136] Most of the time the change in sensitivity is mild to moderate and is transient over a period of 2 to 3 days.[103,133,168] In the case of at-home bleaching, the incidence of hypersensitivity increases with the frequency of changing the bleaching trays, particularly if they are changed more than once daily.[133] For the best immediate remediation, the bleaching periods should be shortened. Both topical fluoridation and desensitizing toothpaste may aid in reducing the hypersensitivity after bleaching.

Hydrogen peroxide that is released from the bleaching trays or from the gel during power bleaching may reach the pulp chamber via craze lines, enamel defects, as well as enamel and dentin cracks.[28,43,71,171] However, it was shown that 50,000 µg of hydrogen peroxide are required to inhibit pulpal enzymes.[79] From bleaching gels containing 12% carbamide peroxide, less than 30 µg of hydrogen peroxide were shown to reach the pulp chamber after tray applications for up to 7 hours. Therefore, at least for at-home external bleaching, it is unlikely to create long-term or significant damage. Nevertheless, to avoid complications, active bleaching should be avoided whenever restorations with ill-adapting margins or exposed dentin areas are present. It has been suggested that desensitizers be used in addition to the bleaching gel. Caution should also be applied regarding children and adolescents due to the larger pulpal volume present in immature, respectively younger teeth.[129]

Gingival Irritation

The most commonly observed adverse effect after external bleaching is gingival irritation, which may be associated with an increase in tooth sensitivity.[131,183,188] Most of the gingival irritations are mild to moderate and disappear after 2 to 3 days without causing significant discomfort for the patient. For in-office bleaching, these issues are mostly related to soft tissue exposures to excessive bleaching gel or liquid hydrogen peroxide in amounts less than necessary to cause severe discomfort or tissue damage. However, when a patient reports tissue irritation, the soft tissues should be checked immediately. Warning signs other than the patient's sensation are air bubbles rising from the gingival margins. The area should be copiously rinsed without delay. A vitamin E preparation should be on hand to serve as an emergency dressing that will provide an immediate antioxidative effect. If an at-home technique is used, gingival irritations may be related to badly adapting trays, and the issue should be resolved after correcting the defective trays. As for gingival hypersensitivity, the increase of gingival irritation is related to the frequency of changing the gel in the custom trays. For more severe cases, topical anesthesia, limited movement, and good oral hygiene can increase the speed of soft tissue healing.

Mercury Release from Amalgam Restorations

Several studies have reported an increased mercury release from amalgam restorations after the application of bleaching agents, in particular carbamide peroxide.[176,179] Although mercury is toxic and can cause damage in larger doses, no health implications due to the induced release of mercury by tooth bleaching has been reported.

PROGNOSIS OF BLEACHING

Most studies addressing the prognosis of internal bleaching refer to the immediate postoperative whitening result.[4,7,11,95,99,145,160] A long-term follow-up of teeth bleached with the thermocatalytic technique reported an 80% success rate after 1 year and a 45% success rate after 6 years, confirming the redarkening of bleached teeth in many instances.[57] Discolorations induced by trauma or pulp necrosis appear to have a good prognosis of around 95% that bleaching reaches the desired result, whereas discolorations due to medications or restorations have a much less successful outcome.[158] In general, discoloration caused by restorative materials has an uncertain prognosis,[225] particularly if metallic ions such as silver, mercury, copper, or iodine are involved. Although some authors disagree,[1,30,100] there may be a higher possibility for successful bleaching in younger patients due to the wider open dentinal tubuli when compared to older patients.[2,94,160] Gray or light yellow discolorations may be easier to bleach than other discolorations.[1] Well-sealing composite restorations are recommended to prevent renewed discoloration.[1] External bleaching may show color regressions after 1 to 3 years.[33]

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CHAPTER 28

Understanding and Managing the Fearful Dental Patient

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CHAPTER OUTLINE

Anxiety and Fear
Mechanisms
Intervariability and Intravariability in Responses
Variability in Stress Response
Variability in Pain Response
Management of Pain and Anxiety
Patient Communication
Behavioral Interventions
Pharmacologic Management of Fear
Summary

The hand froze as she started to dial, almost as if some other life form had seized control of her body. She stood there for what seemed like minutes before she could bring herself to dial the number. Her heart was beating faster—she just couldn't do it. As she fumbled to disconnect, she heard a cheery voice on the other end of the line: "Dr. Rodriq's office, dentistry with a personal touch. May I help you?" She muttered under her breath, "I can't do it now. I'll try tomorrow."

This scenario is familiar to many people. The mere act of making a dental appointment requires greater courage than they can summon up, and except when oral pain becomes unbearable, pursuing dental treatment is out of the question.[80]

Many fears are regarded as socially unacceptable. Dental fear, however, is widely accepted and carries little social stigma. Several studies have confirmed that dental anxiety and fear are common among the general U.S. population and are a worldwide problem.[68,69,75] About 50% of adults in the United States report some dental fear, and 8% to 12% are fearful enough that simply making a dental appointment is problematic.[84] Although women account for more than half of fearful patients, clinicians should not assume that the men they treat as patients are without dental fear and anxiety. Men may not express their fears as openly as women, but exaggerated emotions, such as anger or impatience, may be masking their dental fear.[33] As the fictional scenario demonstrates, many clinicians have attempted to make their practices inviting by presenting a warm and friendly image to the patient who calls. Yet data show that in the United States, the rate of dental anxiety or fear has remained stable during the past 50 years.[83]

Worldwide, the prevalence of dental phobia (an extreme form of fear) is also common. In Australian adults, the level of dental fear ranges from 10% to 14%,[31] whereas in Iceland, Taiwan, and Japan, the percentages of adult respondents who reported at least some dental fear were 21%, 50%, and 82%, respectively.[25,72,77] Studies of children have shown considerable global variation in the prevalence, with some estimates as high as 43%.[30] Given the international prevalence and universal acceptance of dental fear and anxiety, it is impossible for any clinician to avoid fearful patients. Consequently, it is worth the clinician's time and energy to understand the nuances of dental fear and anxiety.[4] This chapter presents general information about fear and anxiety, including proposed mechanisms, and highlights individual differences in response and management strategies. It begins with a section describing the biologic and psychological characteristics of anxiety and fear.

ANXIETY AND FEAR

Anxiety and fear play an adaptive role as an alarm in response to danger. Both are crucial and adaptive components of the overall behavioral and autonomic stress response. In response to dangerous situations that threaten to disturb homeostasis, some biologic systems are activated and others are inhibited in a largely predictable manner. A short-term reaction proportional to the challenge elicits an appropriate response (often escape and avoidance) and is of fundamental importance as a survival strategy. However, this survival strategy taxes the patient's resources, and stress-induced dysregulation may result in a diverse set of health risks ranging from increased susceptibility to viral infection[13,14,89] and activation of latent virus[7,58] to impaired wound healing after surgery[63] and increased mortality.[10]

Anxiety and fear lie at the interface of neurobiology and psychology and are controlled by a highly complex system of both inhibitory and facilitatory mechanisms. These stress mechanisms are dynamic and redundant.[70] Their purposes include maintaining an appropriate degree of emotion under nonthreatening conditions; efficiently responding to potential threats; producing behavioral responses, such as "fight or flight"; and, finally, allowing the patient to rapidly return to

"baseline" after the danger has passed. Authors have attempted to make a distinction between anxiety and fear, and for the purposes of this chapter, those distinctions may be helpful in understanding dental patients' responses to actual and potential treatment.[69,91]

Fear is described as an individual's physiologic and emotional response to a perceived immediate and identifiable threat or danger. It is accompanied by an unpleasant cognitive or affective state of impending doom marked by physiologic changes involving the sympathetic branch of the autonomic nervous system (e.g., hyperventilation, enlarged pupils, gastrointestinal upset, changes in salivary flow, elevated catecholamines) and overt behavior that may involve pacing, shaking, combativeness, or attempts to escape the situation.

Anxiety may or may not differ in the degree of physiologic and emotional arousal compared with fear, but the source of the threat is typically ambiguous, ill defined, and not immediate. Using this paradigm, the emotional and physiologic response a patient reports when imagining a dental appointment would be classified as anxiety, whereas the elevated psychological and physiologic arousal of a patient receiving an injection of local anesthetic while seated in a dental chair would be characterized as fear.

Dental phobia is a special case of dental fear, characterized as a consistent and persistent fear that interferes with a person's social life or role functioning and often leads to avoidance of dental treatment of almost any type. Although the distinction between anxiety, fear, and phobia is useful to consider, most patients do not make such a distinction; consequently, the clinician should be aware that the terms are often used interchangeably.

For most people, the level of anxiety and fear expressed is commensurate with the level of danger or threat present, and the individual quickly adapts to the threatening event, either through reassessment of the level of danger or development of more effective coping skills. For most dental patients, the level of threat from treatment is balanced by the potential to benefit from maintaining good oral health, leading the patient to seek and receive treatment. For others, the fear may be disproportionate to the level of threat, and the individual's behavior is perceived by an outsider to be maladaptive (avoiding treatment until the last moment). For still others, the anxiety experienced is paralyzing with respect to the stimulus and persists in the absence of threats, producing high levels of intrusive thoughts and memories.[21] At its most extreme, this reaction qualifies as a phobia, and the avoidance is of such a proportion that it produces extreme distress and may provoke an immediate and intense anxiety response at the mere mention of the word *dental*.

MECHANISMS

The paradox, that the systems activated by stress-inducing stimuli cannot only protect and facilitate, but also damage the body, was recognized nearly 70 years ago.[26] Since that time, the interactive roles of the autonomic nervous system, the hypothalamic-pituitary-adrenal axis, and the cardiovascular, metabolic, and immune systems during stressful responses have been documented, and the price of the continual accommodation to stress has been characterized.[65]

The stress system coordinates the generalized stress response, which has both central nervous system and peripheral components. The main elements of the stress system's central components, which are located in the brainstem and the hypothalamus, include the arginine vasopressin neurons of the paraventricular nuclei of the hypothalamus; the parvocellular neurons of corticotrophin-releasing hormone; the corticotrophin-releasing hormone neurons of the paragigantocellular and parabrachial nuclei of the medulla and the locus ceruleus; and other, largely noradrenergic cell groups in the medulla and pons (LC/NE). (The reader is referred to an excellent paper by Charmandari et al.[9] for details of the endocrinology of the stress response.)

INTERVARIABILITY AND INTRAVARIABILITY IN RESPONSES

Variability in Stress Response

The stress system receives and integrates a diversity of inputs; these arrive through distinct pathways arising from the individual's unique biology, psychology, and environment. Activation of the stress system is remarkably similar across species, but individual differences do exist that arise from variations in genetics and individual experiences. The knowledge resulting from research into the effects of stressful responding is far-reaching and shows associations across a broad range of health conditions, including wound healing[82]; the severity of infectious diseases[14,59]; inflammatory response[60]; chronic diseases such as cancer[85]; and pain levels.[20,71,73,93]

For instance, psychological stress has been shown to impair human wound healing in a number of well-controlled studies. In one study,[46] the investigators placed 3.5-mm punch biopsy experimental wounds on the arms of chronically stressed caregivers of patients with Alzheimer's disease, in addition to well-matched controls, to study dermal wound healing. In the Alzheimer caregivers group, healing of standardized wounds took an average of 24% longer, with group differences in wound size appearing within the first week of healing. The ability to mount an inflammatory response was tested using an ex vivo endotoxin-stimulated interleukin 1 beta (IL-1β) gene expression assay in whole blood. Alzheimer caregivers produced significantly less IL-1 than did the controls. These results indicate that stress begins to have an impact in the early phases of wound repair, specifically the inflammatory phase, resulting in delayed wound closure. In an oral wound model, with wounds placed 3 days before examinations, dental students healed an average of 40% slower than did the same students with wounds made during summer vacation, and the differences were quite reliable. No student healed as rapidly during examinations as during vacation.[63] These data show that an event as transient, predictable, and relatively benign as examination stress may have significant consequences for wound healing, even in young adults.[62]

It seems quite likely that similar phenomena would be found in fearful dental patients after surgery, but no study to date has tested the question. It is beyond the scope of this chapter to review this important body of work,* but two important factors largely determine individual responses to potentially stressful situations: the way a person perceives the situation and the person's general state of health, which is

*References 11, 40, 42, 44, 47, and 88.

influenced by both genetic factors and lifestyle.[66] Under this paradigm, it is not surprising that both interindividual and intraindividual differences exist in response to potentially aversive stimuli.[23] Nowhere are variations in an individual's response to a stressor more apparent than in a dental office. The following section describes one highly relevant dental outcome that has been shown to be influenced by fear, anxiety, and stress and that exhibits remarkable interindividual variability.

Variability in Pain Response

Two patients with comparable dental disease may report vastly different levels of pain associated with their disease and treatment. One patient may be distressed and report high levels of pain. The other may seem less bothered by the problem and report low to moderate levels of pain. Is one patient an outlier and the other typical? Or are both legitimate expressions of the individual's experience?

The role of these individual differences in the response to stress is complex, and the part these differences play in pain perceptions is only partially understood. Investigators showed that experimentally induced anxiety led to increased pain reactivity, whereas high levels of fear led to decreased pain.[79] Whether the dental setting produces fear or anxiety is not clear, but within the dental care context, patients may have worried for days about a scheduled treatment, resulting in elevated levels of autonomic arousal and high levels of anxiety. This elevated arousal may produce heightened levels of plasma catecholamines, which in laboratory studies have been shown to lower the pain threshold and tolerance.[8] It is not surprising that many highly anxious patients show elevated levels of pain reactivity during dental treatment compared with less anxious patients.[49] Therefore, clinicians should anticipate that anxious patients will have more sensory and affective distress during dental treatment than less anxious patients and treat them accordingly.[61]

A body of literature shows that pain is strongly influenced by factors that include the person's affective, cognitive, and psychosocial history and social learning (see also Chapter 4). One study[56] demonstrated that patients' perception of pain was associated with the amount of personal control they believed they would have during dental treatment, coupled with the amount of control they wished to have. Dental patients with the largest discrepancy between the amount of control they desired and the amount they believed was available were at greatest risk for elevated levels of expected, experienced, and recalled pain during dental treatment.[36] *Catastrophizing,* a form of coping characterized by expecting the worst, predicts greater severity of postoperative pain; it has been linked with both experimental pain sensitivity and clinical pain symptoms.[45] In addition, negative mood predicts greater severity of acute pain and is related to greater pain sensitivity.[37,57]

Negative mood also influences the level of pain that is recalled. Overall, pain is remembered inaccurately and becomes exaggerated over time, with the amount of exaggeration strongly influenced by the intensity of the negative emotions at the time of the painful encounter. Moreover, the elevated recall of pain influences the level of pain experienced at a second painful encounter, such as in dental treatment.[35] These research findings reinforce the significance of efforts to reduce a patient's level of negative emotion before dental treatment.

Increasing evidence indicates that women with high levels of dental fear are more likely to have a history of sexual or physical abuse (or both) than other women. The current estimate of childhood sexual abuse in the general U.S. population is 5.8% to 34% for women and 2% to 11% for men, which is considered by many to be a conservative estimate.[29,94] The sheer number of patients who have experienced sexual abuse strongly suggests that clinicians frequently treat sexually abused patients, and a greater understanding of the impact of abuse on dental attitudes and reactions may help both the patient and clinician to form an effective treatment partnership.

A study of sexually abused European women who were categorized by whether they had been exposed to sexual touching, intercourse, or oral penetration showed that women in the oral penetration group scored significantly higher on dental fear than women in the other two groups.[95] In addition, women with a history of childhood sexual abuse and high levels of dental fear considered interpersonal factors related to the clinician as more important than did women with high levels of dental fear but without a history of childhood sexual abuse. These important interpersonal factors included not feeling that the clinician could be trusted and not feeling in control.[96] It is not hard to understand the parallel between some elements of abuse and receiving dental care, including discomfort and pain, in addition to a reluctance by abused patients to believe that the clinician can be trusted.[87]

From a more general perspective, one study[28] found that a self-reported history of physical or sexual abuse among a nonclinical sample was associated with increased pain complaints and lower perceived health status. Not surprisingly, health care utilization and psychological distress, including the tendency to catastrophize about upcoming events, was greater among women with an abuse history compared with other women.[28] The clinical implications of the association between sexual abuse and dental fear are that clinicians should be prepared to talk to their patients about sexual and physical abuse and the influence of that abuse on receiving dental care.

Fear of pain differs greatly among dental patients and is associated with elevated levels of anxiety.[93] Of particular concern to the clinician is the finding that anxious patients tend to be more fearful of dental pain *after* they experience a procedure than before, whereas less anxious patients tend to report less fear of pain after the procedure has been experienced. For highly anxious dental patients specifically, experiencing treatment tends to make their fear of pain even greater.[93] Individual differences in pain response may correlate to individual biological changes, which may be evaluated objectively using techniques such as functional magnetic resonance imaging (fMRI). Using neural imaging, researchers in one study[73] found that fear of pain predicted activation of the right lateral orbital prefrontal cortex during a heat pain task. This region of the brain has been linked to attempts by fearful individuals to regulate pain by determining how much threat the stimulus represents. This phenomenon of assessing the primary threat level, in conjunction with the coping resources available, is well documented in dental patients[4,15,54] and, in conjunction with recent research using fMRI, provides biologic evidence to support behavioral observation that differences in pain perception are true representations of the individual's experience.[73]

Although a variety of cultural, psychological, and physiologic factors contribute to variability in both clinical and experimental contexts, the role of genetic factors in human pain sensitivity is increasingly recognized. The most dramatic manifestation of individual variations in pain response is congenital insensitivity to pain, caused by single mutations of the *NTRK1* gene. This well-documented genetic defect, which causes pain insensitivity by influencing tyrosine kinase, the receptor for nerve growth factor, underscores the role of individual genes in the reaction to painful stimuli.

One study[48] evaluated genetic influences on variability in human pain sensitivity associated with gender, ethnicity, and temperament. Loci in the vanilloid receptor subtype 1 gene *(TRPV1)*, delta opioid receptor subtype 1 gene *(OPRD1)*, and catecholamine *O*-methyltransferase gene *(COMT)* were genotyped using 5′ nuclease assays. These researchers demonstrated that gender, ethnicity, and temperament contribute to individual variation in thermal and cold pain sensitivity by interacting with *TRPV1* and *OPRD1* single-nucleotide polymorphisms. Another study[24] found that the *COMT* gene was related to experimental pain sensitivity and the risk of developing temporomandibular pain and disease (TMD).

In a fascinating study,[78] 207 cancer patients receiving morphine treatment were genotyped with respect to the Val158Met polymorphism. The study compared the morphine doses, serum concentrations of morphine, and morphine metabolites between the genotype groups. Patients with the Val/Val genotype (n = 44) needed more morphine (155 mg ± 100 mg/24 h; n = 96) and the Met/Met genotype (95 ± 99 mg/24 h; n = 67) ($P = 0.025$). This difference was not explained by other factors, such as the duration of morphine treatment, performance status, time since diagnosis, perceived pain intensity, adverse symptoms, or time until death. These results show that genetic variation in the *COMT* gene may contribute to variability in the efficacy of morphine in cancer pain treatment.

Further evidence has emerged of a genetic basis for differences in patients' response to pain control agents. One study[71] demonstrated a significant link between the melanocortin-1 *MC1R* gene sequence, pain tolerance, and the efficacy of morphine-6-glucuronide in both humans and laboratory animals. In addition, the distinctive molecular processes associated with different types of pain and changes in gene expression over time support the idea that heterogeneous responses to pain stimulus are typical, and "one size fits all" attempts at pain management are unrealistic. It follows that failure to achieve anesthesia in some dental patients may reflect real anatomic and genetic differences. Limited research on the effectiveness of local anesthetic agents has been conducted in the past 50 years, and almost none has focused on individual differences in genetic composition and anatomic structure. One in seven patients reports inadequate anesthesia when the teeth are stimulated with a drill, even after administration of local anesthetic[69]; therefore, it may be time to launch research into the impact on drug efficacy of individual differences in genetic composition.[90]

Brain imaging studies provide additional support for the need to adapt to individual differences in pain mechanism. One study[12] used fMRI to evaluate cerebral responses and, through this approach, confirmed that identical experimental pain stimuli produced different activation in pain-related regions of the cortex. These differences systematically varied in subjects who were sensitive to the pain and those who were not.[12] This study provides additional and persuasive evidence that individual differences in pain responses reflect actual variability in the experience of pain. Incorporating pain management strategies into dental treatment, with the understanding that individual differences in pain response are rooted in true biologic, psychological, and social differences, will lead to better outcomes for patients and less stress for clinicians.

Considerable evidence has been presented supporting the existence of differences among individuals in their responses to dental treatment. It follows that a single management approach for anxious patients is less effective than adapting the strategy to the needs of the individual patient. In the next section, a few selected strategies are outlined, with special emphasis on tailoring communication to the fearful patient.

MANAGEMENT OF PAIN AND ANXIETY

Patient Communication

Individualizing clinician-patient communication, so as to identify, acknowledge, and reassure a distressed patient, is one of the most effective anxiety-reducing strategies available to the dental team. This is a three-pronged strategy involving (1) establishment of rapport with the patient, (2) use of a standard interview protocol, and (3) use of effective nonverbal communication. Each of the prongs of communication requires a dialog with the patient, rather than either of two extremes—one-sided listening to the patient, or a monologue by the clinician that does not provide opportunities for the patient to comfortably speak up. For most patients, having the clinician listen attentively sets a context for building a trusting clinician-patient relationship.

Establishing Rapport

Fearful patients often cite poor communication with the clinician as a factor in maintaining anxiety. In particular, patients report that they do not believe their clinician adequately listens to their concerns.[34] Accurate diagnostic information is the foundation of any treatment, but to obtain that information, the clinician must first develop a trusting relationship with the patient so that a free exchange of information is possible. The first step in building a trusting relationship with a fearful patient is to develop rapport.

Rapport development can begin in the first few minutes of getting acquainted. In fact, because many first impressions are unconsciously made, the clinician may be wise to carefully consider patients' first impressions of the dental provider and his or her staff. There is probably no better way to make a positive first impression than to demonstrate a sincere and genuine interest in the patient. What is the first question the patient is asked by the staff? Often, it concerns the payment policy of the practice and how the patient will be paying. Although it is essential to know about the patient's financial status, making that the first question the patient hears may immediately set an impersonal tone. Research shows that the clinician who initially talks about nondental topics is more likely to be perceived by the patient as friendly, and *friendly clinicians are more likely to have satisfied patients*.[3] An important exception to the use of nondental topics to establish rapport is the patient who immediately initiates conversation about dental-related issues associated with his or her fear. Failing to respond to such an initiative may suggest to the patient that the clinician is avoiding the topic of fear or lacks compassion.

The clinician should also be aware of the impression he or she is making when addressing the patient. The use of a first name assumes or implies a familiarity not all patients are comfortable with. Because the manner in which we address each other frequently sets the context for future interactions, dental providers and their staff should be cautious and sensitive to the individual needs of the patient. Addressing the patient as Mr., Mrs., or Ms. demonstrates respect, and this form of address should be used unless the patient has expressed a preference for use of his or her first name. Older patients and fearful patients may be offended if younger clinicians or staff members address them by their first name. Developing rapport and trust may require additional time and attention with the fearful patient, who is already on guard. It is worth the effort, however, because satisfied, formerly fearful patients become excellent ambassadors for the dental practice. Another effective tool with anxious patients is active and engaged listening. In the following paragraphs, the challenge of listening to a distraught patient in a busy practice is discussed.

Listening

Listening is sometimes viewed as analogous to sending an audio signal to a receiver that faithfully reproduces the signal as it was transmitted; however, listening is not a passive act. Anxious and fearful patients may express their feelings to the clinician or staff in the form of anger and blame. Rather than reacting to the patient's anger, the clinician should show reflective listening. Reflective listening is an effective way to respond to anger, often defusing the exchange regardless of the cause of the anger. When people are angry, one of their primary goals is to convey their feelings to the listener. If a patient is expressing strong emotions, he or she cannot listen very well, so it is best not to offer advice at that moment. Both patients and clinicians listen through a filter of biases and prejudices that influence their interpretation of what is being said. In this kind of interaction, it is especially important that the clinician not assume that he or she understands what the patient is saying or how the patient "feels." Clinicians should encourage the patient to explain expressed fear with statements such as, "It sounds like you have very strong feelings about what happened. I want to be sure that I understand. Please go on." This gives the patient an opportunity to clarify, and the clinician has demonstrated that the emotional content or tone of the concern is being heard.

Strong feelings do not vanish just because they are ignored; they do diminish in intensity and lose their sharp edges when the listener accepts them with respect and understanding and allows the patient to express these feelings. Often the best initial step for either the clinician or the staff member is to *LISTEN!* Once the nature of the problem has been clarified and the patient's strong emotions have diminished, the usual techniques for effective communication can be used, such as open-ended questions with appropriate follow-up questions based on the content and tone of the message. ***Developing a dialog of trust between the clinician and the patient is necessary and cannot be delegated to a staff member.***

Building Empathy

Fearful patients are sensitive to any communication that can be interpreted as belittling or disrespectful. This includes dental jargon the patient does not understand or terms the clinician or staff members do not clearly explain. Fearful patients may be insulted by a statement such as, "There is no reason for you to be afraid," when the patient is convinced there are abundant reasons for fear. The more empathetic the clinician can be, the more likely it is that the fearful patient will accept treatment. The term *empathy* refers to the capacity and willingness to understand a situation from the other person's point of view and communicate that understanding to the person.[19] This is not the same as sympathizing, which can widen the gap between patient and provider and has been shown to be counterproductive in establishing a therapeutic alliance.[52] Instead, reflecting back but not parroting what the patient has said can help the clinician show empathy. For example, if the patient says that he or she hates going to the dentist, a reflective statement might be, "Sounds like you have had some unpleasant experiences with dentists in the past." The patient may elaborate, and at the appropriate time, the clinician can offer reassurance by saying that he or she will do whatever can be done to make the treatment as comfortable as possible. Once a clinician has developed a good rapport with the patient, the rest of the patient's visit is more likely to go smoothly. The clinician will also find that a good rapport with the patient facilitates a smooth standard interview and future treatment. After a trusting relationship has been established, the patient is more likely to accept the moments in which the clinician is distracted as a forgivable event and not take it as a personal insult or lack of interest.

Demonstrating clinical empathy can be more of a challenge in a diverse society. Clinicians frequently treat patients from ethnic, racial, and cultural groups different from their own. It is unreasonable to expect that clinicians can identify or understand what it is like to be from another ethnoracial cultural group. It is still possible, however, to demonstrate clinical empathy if empathy is understood as a feedback loop much like hypothesis testing.[19] Successive cycles of conversation establishing what the patient believes about the nature of dental disease and what needs to be done can allow the patient and clinician to move closer to a shared understanding about an acceptable course of treatment. If nothing else, the clinician's honest attempt to understand the patient's perspective facilitates trust. Trust cannot be earned if the patient feels a lack of concern from the clinician.

Using a Standard Interview Protocol

The use of a standard interview protocol facilitates the dialog with distressed patients. Not infrequently, the anxious patient becomes sidetracked by the emotion-driven recollection of past dental experience, and the responses to questions in the patient history meander, lacking logic and clarity. To keep the interview on track and in professional focus, the clinician may need to gently, smoothly, and efficiently return to the standard protocol and format for the information-gathering process. This may be accomplished by using a statement such as, "I am very interested in that part of your experience, but to be sure we have enough time, please tell me about … Remind me later, and we will return to what you were starting to tell me." Similarly, it may become necessary to pause and deal with the patient's fear in the middle of the interview.

The use of a standard set of questions allows the clinician to concentrate on the patient and the response to each verbal question, confident that all relevant information will be gathered. Depending on the patient's comfort level and curiosity, it is often useful for the clinician to provide a rationale for

particular questions or requests for specific information. The rationale need not be long or detailed but should clearly establish, in lay language, why obtaining the information is in the patient's best interest. Stressed or fearful patients are frequently not attentive to the details of a message, so it is a good practice for the clinician to verify clarity and understanding by summarizing each phase of the interview. The clinician may wish to say, "Sometimes my statements are pretty confusing. [A little personal joke can help make the point, such as a reference to the clinician trying to clarify with his or her teenager what time to come home.] Because much of what we are going to talk about is important if I am to provide optimum care for you, I may ask you to repeat what you think I said just to reassure myself that I am being clear." Patients usually respond positively to such an approach.

Another helpful portion of the standard interview protocol is a question already established on most patient medical/dental health history questionnaires, which is, "Are you anxious about receiving dental treatment?" The question provides two benefits for the patient: It allows the patient to verbalize his or her anxieties, and it also reassures the patient that the clinician is aware he or she may be anxious about the visit. The answer to the question also provides a benefit to the clinician; if the patient responds positively, a series of follow-up questions regarding the patient's perception of the cause or causes of the anxiety can lead to fruitful revelation of specific adverse issues, materials, and techniques. This knowledge can then be very helpful in strategizing how treatment will be planned and carried out to mitigate the patient's aversion and anxiety.

The type of questions the clinician uses in talking with a fearful patient can influence the degree of satisfaction with the interaction for both. For example, if the fearful patient is not talkative, the clinician should consider whether responses could be encouraged through use of more open-ended questions. If the patient is talking excessively, closed questions help the clinician control the interview. Incomplete or inaccurate information may result from questions that lead patients to an answer they believe the clinician is seeking. For example, "I don't suppose there have been any changes in your medications," may suggest to the patient that the clinician does not want to be bothered with any new information. A mix of open and closed questions produces the most effective interview and facilitates an ongoing dialog. Effective communication, however, is not based solely on words. Nonverbal communication can be an effective strategy in building an empathetic and effective relationship with a patient. The following section describes an approach many clinicians find helpful, beginning with silence, which is rarely thought of as an important communication tool.

Using Nonverbal Strategies

There is a strong tendency in the commercial model of health care to think that giving advice and providing treatment is the sole task of the clinician. Periods of silence are often avoided, but silence can be a useful tool when listening to any patient, especially a frightened one. Conversely, good care cannot be provided without spending the time to build a trusting relationship between the patient and the clinician. The silence may feel a bit clumsy or uncomfortable, but patients often report that they are not given adequate time to respond to the clinician's questions. This is especially true with older patients who may need a bit more time to understand what is being said or asked of them. Giving the patient adequate time to collect his or her thoughts and avoiding the tendency to comment too quickly gives the impression that the patient is the focus of attention at that moment. It also helps if eye contact is maintained with the patient; a nod of the head shows that the message is being heard and is important. Permitting the silence to continue may encourage the patient to provide additional relevant information that would have otherwise been missed. Within limits, the clinician should encourage the fearful patient to talk more rather than less.

The clinician's physical orientation toward the patient also provides important messages about the level of interest in what the patient is saying. It is unlikely the patient will continue talking if the clinician appears not to care about what is being said. Making direct eye contact and nodding as the patient speaks can be affirming and can serve as encouragement for more conversation. During the interview, it is most effective for the clinician to face the patient, with the chair at about the same level as the patient's chair. These initial moments are often the time when the fearful patient assesses the clinician's trustworthiness and the extent to which expressed concerns are being taken seriously.

In Western culture, eye contact is the principal means of demonstrating involvement with another human being. Eye contact should be steady and frequent (without staring). Glancing elsewhere is acceptable, but the patient's face should be the focus of the clinician's attention. The clinician may take notes, but it is important to reestablish eye contact after each note is taken to convey the impression that what the patient is saying is important. Showing care in this way is one avenue of demonstrating clinical empathy. Patients are very sensitive to discordant messages between our words of caring and aspects of nonverbal communication, such as eye contact. Maintaining eye contact until the patient has stopped speaking provides a powerful statement of respect and concern.

The fearful patient is best served if the context in which care is received is based on mutual respect and concern. Many fearful patients have not received routine preventive care and may have extensive treatment needs, including some requiring complex, invasive treatment. If so, the patient will need reassurance that the clinician will make every effort to provide comfortable treatment.

No promises should be made that the treatment will be painless or free of discomfort. Pain-free dentistry is not a promise that can always be kept. It is also important to acknowledge the patient's feelings. Acknowledgment does not represent endorsement; it simply confirms that what the patient said was heard. During treatment, frequently expressing sincere interest in the patient's level of comfort and keeping the patient informed of progress helps relieve anxiety. The expression of concern should be sincere, not automatic.

As part of the framework for care of a fearful patient, the clinician should work quickly and systematically without appearing hurried. Planning ahead and informing the patient about the next step can be reassuring. Keeping promises made to the patient maintains the patient's trust. Such promises may include the length of the appointment or the frequency of breaks. Being honest about what is and is not realistic helps both the clinician and the patient. Fearful patients appreciate a frequent review of what has been accomplished, what remains to be completed, and any unanticipated deviations from the

original plan. Setting a context of predictability is important to most patients, but with fearful patients, the appearance of predictability projects organization and control.[22] The basis of trust often rests in the patient's sense of personal control.

Research has shown that fear about dental care increases when the fearful patient wants control during treatment and believes he or she will not have it.[56] A sense of personal control can be provided through information, predictable sequences, and choices about the treatment and the treatment process. One strategy that returns some level of control to the patient involves inviting the individual to raise a hand when he or she would like to take a timeout. It should not be surprising if a patient tests the clinician's willingness to give up control during the treatment by frequently raising a hand. Other options may include giving patients a choice of music to listen to during treatment, which implies control over the environment.[2,39] (See de Jongh et al.[22] and Logan and Marek[55] for reviews of individualized dental anxiety management strategies.)

Patients who manage their apprehension about dental treatment often have identified coping strategies that have worked well for them in the past. Fearful patients may not have such well-defined strategies. Asking about preferred coping strategies and helping the patient identify one that usually works has proven useful in reducing anxiety.[54] Dental anxiety can also be managed with conscious sedation techniques,[67] but it is important to remember that these agents do not treat anxiety, they only facilitate treatment. When conscious sedation techniques are used, they are most effective when combined with behavioral interventions.[43,53] (See de Jongh et al.[22] for a review of individualized management strategies for dental anxiety.)

In short, if anxiety reduction is to be possible, a trusting relationship needs to be in place whereby (1) the patient's feelings of anxiety are acknowledged, (2) a safe and secure environment is in place, and (3) the patient believes that nothing will happen that the patient and clinician have not agreed on.[22] In addition to the strategies described before, numerous other behavioral interventions are available. Although no one strategy has emerged as best, most have shown that dental anxiety is treatable, and the effects of the behavioral treatment are long lasting.[50] Next, four interventions with particular relevance to dental care are highlighted in somewhat greater detail.

Behavioral Interventions

Distraction

Distraction as an anxiety-reducing strategy has broad appeal because of the ease of use. Music represents one of the simplest distraction techniques to use and is particularly effective if the patient selects the music and listens over individual headphones. Regardless of their anxiety level, patients often report that they prefer to have music in the background because music makes them more comfortable.[3] In a randomized clinical study, patients who listened to music during dental treatment reported less pain, less discomfort, and a greater sense of personal control.[2] Thus music can be both distracting and, under certain conditions of choice, provide the patient with a sense of control.

Portions of the office environment can also divert a patient's attention. It has often been reported that interesting artwork, decor, and tropical fish tanks are useful in distracting the patient from feared stimuli.[3] Some clinicians have used video games or a television set in the operatory as an effective and comforting distraction for anxious patients.[16,32] Manual distraction-stimulating devices have also been used during injection of dental anesthetics to reduce dental anxiety.[92] The clinician should be aware that simple distraction techniques may be ineffective if the perceived intensity of dental pain is too high.[5] Distraction is largely effective in the short-term management of anxiety but less effective for long-term behavior change.

Relaxation

The goal of relaxation is to achieve both muscular and mental relaxation. Research shows that relaxation is an effective method of reducing patient anxiety.[17,18] Deep breathing coupled with muscle relaxation can be effective in stress reduction. Many fearful adults tend to hold their breath during basic procedures such as applying a rubber dam, giving injections, or forming impressions. When the person's blood becomes poorly oxygenated because of an insufficient amount of fresh air entering the lungs, states of anxiety, depression, and fatigue arise, contributing to the already stressful situation. Deep breathing exercises to help reduce this unwanted stress can consist of as little as 2 to 4 minutes of breathing in deeply, holding the breath, and then exhaling completely. Demonstrating for patients that deep breathing is something they can do on their own provides a new sense of control, in addition to the calming effects from the breathing techniques. It may even be helpful to attach a heart rate monitor so that both clinician and patient can assess this aspect of stress arousal and success in controlling it.

Pausing during the procedure and suggesting that the patient briefly repeat the deep breathing techniques can be helpful. Muscle relaxation is also very useful in calming the patient. This method includes repeated tensing and relaxing of specific muscle groups in the body. Breathing and muscle techniques can be combined by tensing the muscles while breathing in and relaxing them while breathing out. Practicing this rhythmic coordination of relaxation techniques quickly and effectively improves the patient's ability to relax.[69] Periodic reinforcement of muscle relaxation during treatment helps the patient regain composure. Pausing during the procedure and suggesting that the patient take several slow, deep breaths can also be useful. For some fearful patients, the mere act of deeply inhaling and exhaling completely can help dispel negative reactions to receiving care. Some caution should be used in attempting to combine music and relaxation therapy because some types of music may have unanticipated effects on the patient and may actually increase autonomic arousal.

Hypnosis and Guided Imagery

Hypnosis is a guided, self-controlled state of mind in which concentration and focus are directed inward. An altered level of consciousness is reached, similar to "zoning out" while daydreaming or reading a book.[86] Guided imagery is a form of mild hypnosis that can be useful with fearful patients.[6] It produces a light trance from which patients easily awaken, and the procedure is less time-consuming for the clinician than guiding patients into a deep hypnotic state. Asking the patient to focus on a place in which he or she feels very relaxed, comfortable, or safe is a good starting point for guided imagery and can be effectively combined with relaxation training.[81]

Information about appropriate images can be gathered during the examination process. Current research shows that patients have reduced pain and distress when they themselves choose the place to be imagined. The patient should be asked to choose imagery that is associated with little movement so that movements do not interfere with the provision of care. During guided imagery, the patient achieves an altered state similar to daydreaming or focused attention. By focusing on a calm and safe scene, positive emotions are elicited that can block or mitigate the anxiety arising from the dental treatment. An analysis of imagery topics chosen by patients shows that the topics were highly individual; this further supports the efficacy of guiding patients to a "safe and comfortable place" of their own choosing.[27,53] Guided imagery is effective in managing pain during outpatient procedures and can be delivered by the dental team without disrupting the workflow in the patient care setting.[6,51]

In the past, the success of hypnosis was believed to depend on the hypnotizability of the subject and was associated with a specific "phobic" portion of the population.[38] More recently, researchers have shown that nearly all patients are equally able to engage in imagery during invasive outpatient procedures, and the imagery has resulted in reduced pain and anxiety.[51] Although nearly all patients are equally susceptible to hypnotism, their attitudes, motivations, and fears from common misconceptions may interfere with or impede their willingness to engage in a hypnotic state.[27] The most successful conditions involving hypnotism in the clinical setting include a well-trained hypnotherapist and a patient who is highly motivated to overcome a problem.[1,14] *For most mildly or moderately anxious patients, the most effective management strategy for the clinician is taking time, actively listening to the patient's concerns and fears, and emphasizing the building of a trusting relationship between the patient and the clinician.*

For more serious cases, deep breathing techniques and hypnosis or guided imagery are among the most useful. The effectiveness of hypnosis in treating dental fear has been demonstrated.[38] Hypnosis, however, requires specialized training and experience, so a brief "how to" belies the complexity of the strategy. For clinicians who are interested in training in hypnosis, resources can be accessed through the Internet[74] or through other printed materials.[69] Information about training in the specific area may be available through the local dental society or mental health center.

Referral to a Mental Health Professional

In extreme cases, refining the dialog and using previously mentioned strategies are inadequate to reduce the patient's distress and fear. The clinician may want to exercise the option to refer a fearful patient to a mental health worker for professional counseling and therapy. This decision may depend on the clinician's own skills and interests in working with fearful patients. One criterion used for referral is whether the fear imposes a significant barrier to successful completion of care. If this is the case, referral may be warranted. If a therapist's name is not readily available, a call to a county or state psychological or psychiatric association will yield the names of professionals who specialize in anxiety disorders. If the patient is currently in therapy, it is appropriate to ask the patient's permission to speak with the therapist. If the patient gives permission to contact the therapist, the clinician should consider maintaining an ongoing dialog with the therapist during the course of dental treatment. The following are questions the dental provider and the therapist may discuss.

- Do adjustments need to be made in the sequence of the dental treatment plan?
- Do adjustments need to be made in the anxiety management strategies as the treatment progresses?
- Should new anxiety treatments be tried?

Based on the conversation with the therapist, the clinician can decide whether further referral is necessary or if the patient is being treated adequately for his or her dental fear. If the patient's dental fear and anxiety do not subside, other measures may be needed, including pharmacotherapy. As with all treatment, careful documentation of the anxiety treatment is important. The dental team should be reminded to assiduously adhere to the standards established by the Health Insurance Portability and Accountability Act (HIPAA) in discussing the patient's care among themselves and in sharing information unless directly relevant to provision of dental care.

PHARMACOLOGIC MANAGEMENT OF FEAR

If clinician-patient dialog is not adequate to calm the fearful patient, sedative-hypnotic drugs that treat anxiety and insomnia may be an option. Agents used to treat anxiety are known as *antianxiety agents* or *anxiolytics*. An older term is "tranquilizer." Agents used to promote sleep are known was *hypnotics*. The benzodiazepines are the most commonly prescribed group of drugs used to treat anxiety. This class of drugs has four distinct effects: anxiolytic, anticonvulsant, muscle relaxant, and sedative-hypnotic. The distinction between an anxiolytic effect and a hypnotic effect is often a matter of dose or length of action. Lower doses relieve anxiety, whereas higher doses induce sleep. In addition to these actions, the benzodiazepines are known for producing anterograde amnesia.

The benzodiazepines are by far the largest and most important group of drugs used as anxiolytic and hypnotic agents. The antianxiety and calming effect of this class of compounds was discovered in the late 1950s. The basic 7-membered ring can be modified to alter half-life, number of active metabolites, overall duration of action, and therapeutic use.

The benzodiazepines act selectively on gamma-aminobutyric acid A ($GABA_A$) receptors throughout the central nervous system. The benzodiazepines work by enhancing the inhibitory effects of GABA. Short-term use of these medications is generally considered safe and effective; however, long-term use is controversial because of the potential for tolerance and dependence, cognitive impairment, and falls, especially in the elderly or when these drugs are combined with opioids.[41]

The many different benzodiazepines vary in potency, speed of metabolism associated with half-life, number of active metabolites, and approved indications (Table 28-1).

These drugs are classified as U.S. Food and Drug Administration (FDA) Pregnancy Category D or X, depending on the drug. They should not be used in pregnant patients because the risk outweighs the benefit; potential harm to the unborn has been demonstrated.[64]

The benzodiazepines are one of the few classes of drugs with a commercially available competitive antagonist, romazicon (Flumazenil). This drug, which is administered by

TABLE 28-1
Frequently Prescribed Benzodiazepines

Drug Name	Half-Life (Hours)
Alprazolam (Xanax, generics)	12-15
Chlordiazepoxide (Librium, generics)	>100 with active metabolites
Clonazepam (Klonopin, generics)	20-50
Clorazepate (Tranxene, generics)	>100
Diazepam (Valium, generics)	>100
Estazolam	10-24
Eszopiclone (Lunesta, generics)	6
Flurazepam (Dalmane, generics)	>100
Lorazepam (Ativan, generics)	10-20
Oxazepam (generics)	5-14
Temazepam (Restoril, generics)	3.5-18.4
Triazolam (Halcion, generics)	1.5-5.5
Zaleplon (Sonata)	1
Zolpidem (Ambien, generics)	2.5[76]

injection, is used to reverse excess sedation after a benzodiazepine has been used for diagnostic or therapeutic procedures or in the case of an overdose. Romazicon's short half-life requires multiple dosing and careful monitoring of the patient to ensure that resedation does not occur.

Because all benzodiazepines have essentially equivalent pharmacologic actions, selection is largely based on the difference in the course of action. For example, if a patient needs medication to accelerate falling asleep, a benzodiazepine with a rapid onset is preferable. For treatment of anxiety and fear for a dental appointment, a shorter acting agent, such as lorazepam, triazolam, or alprazolam, may be preferable.

SUMMARY

Individual patients differ dramatically in how they respond to dental treatment. These differences arise from multiple biologic, psychological, and social sources, which are influenced by both genetic factors and lifestyle. How successfully a clinician adapts to these differences by tailoring treatment options depends on the depth of his or her understanding of the way a patient perceives the situation and the person's general state of health. Incorporating this understanding into the clinician-patient dialog can produce improved clinical outcomes.

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CHAPTER 29

Endodontic Records and Legal Responsibilities

EDWIN J. ZINMAN

CHAPTER OUTLINE

Endodontic Record Excellence
- *Importance*
- *Patient Information Form*
- *Medical Health History*
- *Dental History*
- *Diagnostic and Progress Records*
- *Electronic Records*
- *Radiographs*
- *Evaluation and Differential Diagnosis*
- *Diagnostic Tests*
- *Treatment Plan*
- *Informed Consent Form*
- *Treatment Record: Endodontic Chart*
- *Preoperative and Postoperative Radiographs*
- *Digital Radiography*
- *Radiation Safety*

Endodontics and Heart Disease
- *Abbreviations*
- *Computerized Treatment Records*
- *Health Information Technology for Economic and Clinical Health (HITECH) Act Pitfalls*
- *Health Insurance Portability and Accountability Act (HIPAA)*
- *Record Size*
- *Identity of Entry Author*
- *Patient Record Request*
- *Patient Education Materials*
- *Postoperative Instructions*
- *Recording Referrals*
- *Dental Fees Related to Quality Care*
- *Record Correction*
- *Spoliation*
- *False Claims*

Legal Responsibilities
- *Malpractice Claims*
- *Incidence of Negligence*
- *Malpractice Prophylaxis: Importance of Records*
- *Standard of Care*
- *Standards of Care: Generalist versus Endodontist*
- *Ethical Guidelines*
- *Ordinary Care Equals Prudent Care*
- *Customary Practice versus Negligence*
- *Employment Law*
- *Dental Negligence Defined*
- *Locality Rule*
- *Continuing Education*
- *Scientific Research Evaluation*
- *Evidence-Based Endodontics*
- *New Products*
- *FDA Approval*
- *FDA Drug Approval*
- *FDA Clearance*
- *Adverse Drug Events (ADEs)*
- *Primary Medication Nonadherence: Unfilled Prescriptions*
- *FDA MedWatch*
- *Compounding Pharmacies*
- *Dietary Supplements*
- *Negligence Per Se*
- *Prescription Drugs*
- *Ability to Foresee Unreasonable Risk*
- *Consent*
- *Alternative Technique Choices*
- *Referrals to Other Specialists*
- *Surgical versus Nonsurgical Endodontics*
- *Microscopic Endodontics*
- *Separated Instruments*
- *Equipment and Supplies*
- *Clinician's Liability for Staff's Acts or Omissions*
- *Abandonment*
- *Expert Testimony*
- *National Practitioner Data Bank*
- *Professional Negligence Incidence*
- *Managed Care and the Employment Retirement Income Security Act*

Malpractice Incidents
- *What to Do If You Are Sued*
- *Screw Posts*
- *Paresthesia Prevention*
- *Treatment Failure*
- *Full Disclosure*
- *Slips of the Drill*
- *Fear Factor*
- *Leakage*

Electrosurgery
Implant Risk Factors
Fluoride Varnish for Caries Prevention
Xerostomia and Diet
Reasonable versus Unreasonable Errors in Judgment
Prophylactic Endodontic Practice
Malpractice Prophylaxis
One-Visit versus Two-Visit Endodontics
Prevention of Orthopedic Joint Infections
Disclosure Errors
Future of Antibiotics and Resistance
Temporomandibular Disorders
Patient Rapport
Fees
Post Perforation
Perforation Prevention
Bleach Injury
Bleach Usage
Cores
Absorbable Hemostatic Agents
Pneumomediastinum (Air Embolus)
Broken Needle
Sterilization
Medication Errors
Posttrauma Therapy
Millennium Management of Endodontic Advances
Statistical Research
Other Clinicians' Substandard Treatment
Peer Review
Human Immunodeficiency Virus and Endodontics
Summary

ENDODONTIC RECORD EXCELLENCE

Importance

Endodontic therapy records serve as an important map to document and guide the clinician's journey down the correct diagnostic and treatment path. Documentation is essential to attaining endodontic excellence.

The dental record must contain sufficient information to identify the patient, support the diagnosis, justify the treatment, and document the course and result of treatment, and it should be designed to protect the patient's welfare. Records also are fundamental means of communication among health care professionals.

Content

Endodontic treatment records should include the following information:

1. Name of patient
2. Date of visit
3. Medical (periodically updated) and dental history
4. Allergies and adverse drug reactions
5. Chief complaints
6. Radiographs of diagnostic quality
7. Pulpal and periodontal diagnostic tests performed
8. Clinical examination findings
9. Differential diagnoses and final diagnosis
10. Treatment plan
11. Prognosis
12. Referrals, including patient refusals (if any)
13. Communications with other health care providers
14. Progress notes (including complications)
15. Completion notes
16. Canceled or missed appointments and stated reasons
17. Emergency treatment
18. Patient concerns and dissatisfactions
19. Planned follow-ups
20. Drug and laboratory prescriptions
21. Patient noncompliance
22. Consent and informed refusal forms
23. Accounting ledger
24. Recall notifications
25. Name or initials of author for each treatment entry
26. E-mail address of patient
27. Emergency contact persons, addresses, and phone numbers
28. Patient phone numbers including home, work, and cell
29. Log of records and radiographs received or sent

Function

Dental records should document the following information:

1. Course of therapy by recorded diagnosis including differential diagnoses, informed consent or refusal, treatment, and prognosis
2. Communications among the treating clinician, other health care providers, consultants, subsequent treating practitioners, and third-party carriers
3. Necessity and reasonableness of diagnosis and treatment capable of peer review and dental insurance carrier evaluation
4. Standard of care compliance
5. Adverse reactions or events with explanation
6. Follow-up comments to medical history–positive responses

Patient Information Form

A patient information form provides essential data for patient identification and office communication, which should be updated periodically (Fig. 29-1). The patient's name; home, business, and e-mail addresses; and telephone and fax numbers are needed to contact the patient for scheduling purposes and inquire about postoperative sequelae.[178] Location information about the patient's spouse, relative, or a close friend who can be notified in an emergency is also suggested. In the event the patient is a minor, the responsible parent or guardian should provide the information. Questions about dental insurance and financial responsibility are included on the form to avoid any later misunderstandings and help fulfill federal requirements of the Truth in Lending law, applicable if four or more

PATIENT ACCOUNT INFORMATION

Name: _____ Social Security No.: _____ Today's Date: _____

Birth date: _____ Responsible Parent or Guardian's Name: _____

Marital Status: ___ Single ___ Married ___ Widowed ___ Divorced

Address: _____ City: _____ State: _____ Zip Code: _____

E-mail Address: _____ Phone: _____ Fax: _____

REFERRED BY: _____ Patient Driver's License No.: _____

Occupation: _____ Employer: _____ How long: _____

Business Address: _____ City: _____ State: _____ Zip Code: _____

E-mail Address: _____ Phone: _____ Fax: _____

Spouse's Name: _____

Occupation: _____ Employer: _____ How long: _____

Business Address: _____ City: _____ State: _____ Zip Code: _____

E-mail Address: _____ Phone: _____ Fax: _____

PERSON FINANCIALLY RESPONSIBLE FOR ACCOUNT: _____

Address (if different from patient): _____

City: _____ State: _____ Zip Code: _____ Phone: _____

Relationship to patient: _____

DENTAL INSURANCE CARRIERS:

_____ Group No.: _____ Local No.: _____

Name of Insured Person: _____ Social Security No.: _____

Relationship to patient: _____

SECOND DENTAL INSURANCE CARRIER (if dual coverage):

_____ Group No.: _____ Local No.: _____

Name of Insured Person: _____ Social Security No.: _____

Relationship to patient: _____

Date: _____ Signature: _____

FIG. 29-1 Patient information form.

installment payments are arranged (whether or not there are interest or late-payment charges).[104]

Medical Health History

The clinician should thoroughly review the patient's past and present health status before proceeding so that dental treatment can be safely initiated. Health questionnaires open avenues for discussion about problems of major organ systems and important biochemical mechanisms, such as blood coagulation, allergy, immunocompromised status, need for antibiotic prophylaxis, and disease susceptibility. The clinician may request that the patient be examined by a physician or undergo laboratory testing under medical supervision to determine whether a suspected medical problem may require attention before endodontic therapy proceeds or if drug sensitivity or an allergy mandates treatment modifications.[43,165,188] Knowledge about current medications, medical therapy, and the name and address of the treating physician is essential.

Medical histories must be updated periodically (or at least annually or sooner as the need arises). The patient should be asked to review the previous and current medical history (Fig. 29-2). If no changes are necessary, the patient should date and sign the history form. If any changes occurred, the patient should identify each updated medical change, the date, and sign the form where indicated for medical update information. Periodically the patient should provide an entirely new updated form rather than changing data on the old form. Earlier medical histories should be retained in the chart for future reference. If physician communication for treatment occurs, the clinician should record these contacts. In addition, the clinician should

TELL US ABOUT YOUR MEDICAL HISTORY

First Name: _____ Last Name: _____

How would you describe your health? Please circle one. Excellent Good Fair Poor

When did you have your last physical examination? _____

Are you currently being treated for any illness or medical condition? Yes ____ No ____

If yes, please describe: _____

_____

Who is treating you for this condition? _____

Have you ever had any kind of surgery? Yes ____ No ____

When did you have this surgery? _____

What type of surgery did you have? _____

Have you ever had any trouble with prolonged bleeding after surgery? Yes ____ No ____
Do you wear a pacemaker or any other kind of prosthetic device? Yes ____ No ____
Are you taking any medication or drugs at this time? Yes ____ No ____
Have you ever taken Fen-phen and/or Redux (diet drugs)? Yes ____ No ____
What medications, drugs, or herbs are you taking?

_____

Why are you taking these medications? _____

_____

Have you ever had a reaction or complication to a local anesthetic or drug (like penicillin)? Yes ____ No ____

If yes, please explain: _____

Please circle any present or past illness you now have or had in the past:

Alcoholism	Blood pressure	Epilepsy	Hepatitis	Kidney or liver	Rheumatic fever
Allergies	Cancer	Glaucoma	Herpes	Mental	Sinusitis
Anemia	Diabetes	Head/neck injuries	Immunodeficiency (HIV)	Migraine	Ulcers
Asthma	Drug dependency	Heart/valve disease	Infectious diseases	Respiratory	Venereal disease

Are you allergic to Latex or any other substances or materials? Yes ____ No ____

If so, please explain. _____

If female, are you pregnant? Yes ____ No ____

Is there any other information that should be known about your health? _____

_____

Signature of Patient (or Parent) _____ Date _____

FIG. 29-2 Medical history form.

verify physician approvals for treatment by fax, with verification of receipt or letter or preferably both, and retain copies in the chart.

Updating the medical history requires the practitioner to be apprised of changes in the patient's medical condition and any new medications the patient is taking, including over-the-counter or herbal medications or supplements.[257] A patient untrained in medical science may not appreciate the fact that new medications may suggest new diseases or changes in existing disease status. For instance, certain regurgitating valvular heart diseases may require antibiotic prophylaxis.[334] New medications may also cause a synergistic effect with other medications the patient is using or another treating clinician is prescribing.

Clinicians who pass the buck by claiming, "Oh it was my secretary or my new assistant—I could do nothing about it," are nonetheless legally liable for their staff's actions or inactions.[335] A clinician exclusively relying on staff members to obtain medical histories in a waiting room full of patients is making a mistake. Instead, the clinician must check and then follow up regarding the accuracy of the histories. Training and monitoring staff are duties clinicians cannot afford to ignore. President Harry Truman's sage advice, "The buck stops here," applies to treating clinicians who overdelegate to nonclinician staff members who lack the requisite dental education for adequate medical history follow-up.

Dental History

The dental history should include past dental difficulties, name and address of current or most recent treating clinician, chief complaint (including onset, duration, frequency, type, and

TELL US ABOUT YOUR DENTAL SYMPTOMS

First Name: _____ Middle Initial: _____ Last Name: _____

1. Are you feeling any pain at this time? If not, please go to Question 5. Yes ____ No ____
2. If yes, where is the pain located? _____ Any pus? Yes ____ No ____
3. When did you first notice any symptoms? _____
4. Did symptoms occur suddenly or gradually? _____
5. Please check the intensity, frequency, and quality of the discomfort, which most closely describes your pain:

INTENSITY	FREQUENCY	QUALITY
(On a scale of 1 to 10) 1 = Mild, 10 = Severe		
1 __ 2 __ 3 __ 4 __ 5 __ 6 __ 7 __ 8 __ 9 __ 10 __	__ Constant __ Intermittent __ Momentary __ Occasional	__ Sharp __ Dull __ Throbbing

6. Is there anything you can do to relieve the pain? Yes ____ No ____
   If yes, what? _____
7. Is there anything that provokes pain or that you do to cause the pain to increase? Yes ____ No ____
   If yes, what? _____
8. When eating or drinking, is your tooth sensitive to: Heat ____ Cold ____ Sweets ____
9. Does your tooth hurt when you bite down or chew? Yes ____ No ____
10. Does it hurt if you press the gum tissue around this tooth? Yes ____ No ____
11. Does a change in posture (lying down or bending over) cause your tooth to hurt? Yes ____ No ____
12. Do you grind or clench your teeth? Yes ____ No ____
    If so, do you wear a splint or night guard? Yes ____ No ____
13. Has a restoration (filling or crown) been placed on this tooth recently? Yes ____ No ____
14. Prior to this appointment, has root canal therapy been started on this tooth? Yes ____ No ____
15. Any recent or past trauma or injury to this tooth? Yes ____ No ____
    If the answer to the preceding question is yes, describe recent or past trauma and occurrence date.
    _____
16. Is there anything else we should know about your teeth, gums, or sinuses that would assist us in our diagnosis or treatment? _____

Signature of Patient (or Parent) _____ Date _____

FIG. 29-3 Dental history record.

intensity of any pain or numbness), relevant prior dental treatment, and attitude regarding teeth retention. Positive responses suggest further patient consultation and consideration for obtaining records (by written release) for elucidation from the patient's previous or present treating clinician.[227] After receiving positive responses, the clinician may also wish to obtain prior and concurrent treaters' radiographs for current comparison with any progressive changes (Fig. 29-3).

Diagnostic and Progress Records

Diagnostic and progress records often combine fill-in and check-off types of forms. Fill-in or essay forms allow greater latitude of response to a question, resulting in a more detailed description. However, one drawback is that these forms are open to oversights unless a clinician is conscientious in following up with further clinical information.

Using only an essay-type health history is insufficient. Often a patient may not appreciate the significance of important symptoms. A check-off format is efficient and more practical. Forms with questions that reveal pertinent data alert the clinician to medical or dental conditions that warrant further evaluation or consultation.[43] Moreover, such records can document any missing medical information the patient failed to provide orally. At the end of the check-off portion of the medical history, an essay question allows the patient to provide any omitted pertinent medical information or elaborate on check-marked items.[273,286]

Electronic Records

Electronic records represent current methodology for recording patient information (see Chapter 30 for details).[307] Several safeguards are essential for their use. First, patient

confidentiality and security must be protected. Consider using AES encryption with at least 2048 bit key. Be certain that only the clinician and authorized staff view patient data in conformance with the Health Insurance Portability and Accountability Act (HIPAA) security standards, effective April 2005.[149] Second, data backup of patient records, including digital radiographs and account ledgers, should be stored off site. The entire hard drive should be periodically backed up for additional protection, which can be accomplished with cloud technology autosave backup systems.[94] Multiple copies of the backup data should be stored in more than one geographic location, and fault protection such as RAID level 1 or 5 should be installed to prevent data loss.

Radiographs

Radiographs are essential for diagnosis and also serve as additional documentation of the patient's pretreatment condition. A panographic radiograph is not diagnostically accurate for endodontics and therefore should be used only as a screening device (see Chapter 2).[143,271,344] Diagnostic-quality periapical plain film or digital radiographs are essential aids for diagnosis, for working films (e.g., measuring the length of root canals, fitting gutta-percha cones), to verify the final fill, and for follow-up comparisons at recall examinations. Therefore, the clinician should retake any radiographs that lack diagnostic quality. Retain all radiographs including working films.

Digital radiography is recommended because it increases endodontic efficiency at the chair. Also images may be forwarded via e-mail to either the patient or another clinician. No developing time is needed, so procedural radiographs can be viewed instantly and approved or retaken with little time in between (no downtime is required to wait for a film to exit the film processor). Newer digital x-rays with improved sensor technology use high-resolution charged coupling device (CCD) chips, rather than complementary metal-oxide semiconductor (CMOS) technology, and provide close-up zooming with improved image quality as described in Chapters 3 and 30.

Three-dimensional (3D) cone-beam computerized tomography (CBCT) imaging has greater information compared with conventional radiography. Conventional periapical radiographs do not facilitate accurate predictions in the preoperative assessment for the potential perforation of the maxillary sinus. CBCT has greatly aided the diagnosis of pain sources caused by root fractures or incompletely filled, overfilled, or missed extra canals that two-dimensional (2D) periapical imaging may obscure.*

According to the U.S. Food and Drug Administration (FDA), switching from D to E speed results in a 30% to 40% reduction in radiation exposure. F-speed film reduces exposure 20% to 50% compared to E-speed film, without compromising diagnostic quality.[19] Digital imaging reduces radiation dose by 40% to 60%.[5,124]

A rectangular collimator decreases the radiation dose by up to fivefold compared with a circular one.[126] Radiographic equipment should provide rectangular collimation for exposure of periapical and bitewing radiographs.

Evaluation and Differential Diagnosis

Diagnosis includes evaluating pertinent history of the current problem, clinical examination, pulpal testing, periodontal probe charting, and recorded radiographic results. If therapy is indicated, the reasons should be discussed with the patient in an organized way. When other factors affect the prognosis (e.g., strategic importance, restorability of the tooth, or proximity to vital structures), the clinician should consider further consultation with the referring clinician or specialists, including a prosthodontist, periodontist, or radiologist with CBCT imaging, before initiating endodontic treatment.

Diagnosis is essential in treatment decisions. Recommending extractions for undiagnosed pain etiology is generally contraindicated. Chronic idiopathic orofacial pain warrants referrals and consultation with other health care professionals, lest wholesale extractions worsen the patient's dental state and not relieve pain (see Chapter 17).[7] For instance, in a long-term follow-up study of patients with chronic facial pain and without a recognizable odontologic pathosis, some patients were subsequently diagnosed with maladies ranging from cracked-tooth syndrome to medication-mediated illnesses.[7] Accordingly, referral to other specialists is essential, lest a patient be misdiagnosed with idiopathic or atypical facial pain when instead the pain etiology was misdiagnosed.[238,255]

About 5.3% of patients have postendodontic pain 6 months or more after nonsurgical and surgical endodontic treatment. Nonodontogenic pain may account for about 57% to 64% of the total causes of postendodontic pain that persists after 6 months. Therefore, of patients being treated with endodontic procedures, about 3.4% (64% of 5.3%) will have posttreatment pain that arises from a nonodontogenic cause.[41,237]

Fig. 29-4, *A* and *B*, demonstrates the advantage of CBCT 3D imaging compared to 2D imaging. In *A*, the nail appears inside the cranium, contrasted with *B*, which shows the nail taped outside the skull.

Diagnostic Tests

Sound endodontics begins with a proper diagnosis. Otherwise, unnecessary or risky treatment with a compromised prognosis follows. Generally, as described in greater detail in Chapter 1, the following endodontic tests should be performed to arrive at a correct and accurate endodontic diagnosis:

1. Percussion
2. Thermal testing
3. Electrical testing (optional)
4. Palpation
5. Mobility
6. Periodontal assessment (pockets, mobility, furcations, gingival recession)

Both positive and negative pulpal testing results should be recorded. Juries, peer-review committees, and insurance consultants often disbelieve unrecorded test results. Uncharted test findings may be regarded as never having been done. Consequently, reasonable clinicians should record all testing results, both positive and negative. An evaluation of the temporomandibular apparatus is necessary to diagnose or rule out temporomandibular disorders (TMDs).

Treatment Plan

Treatment records should contain a written plan that considers all aspects of the patient's oral health. Treatment plans should be coordinated, preferably in writing, with other concurrent or jointly treating clinicians. For instance, endodontic treatment will probably fail if underlying periodontal pathosis is ignored or untreated. Thus, the clinician should assess the patient's

*References 20, 75, 97, 157, 169, 201, 204.

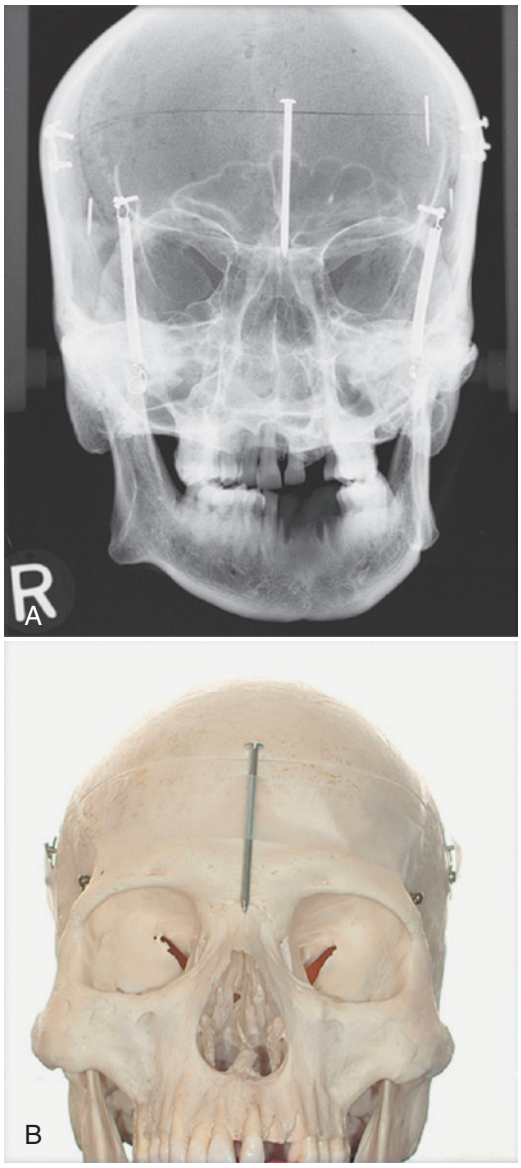


FIG. 29-4 A, Nail inside cranium? B, Nail in same patient as in (A), showing nail taped to outside of skull. Alas, poor Yorick!

entire dentition and not just a single root canal system. The clinician should also recommend a periodontal consultation to both the patient and the referring clinician if periodontal therapy is required. The patient should approve the treatment plan. Remember the maxim: If you fail to plan, plan to fail.

Examination

After gathering the dental and medical history, findings obtained from the various phases of the clinical and radiographic examinations are recorded as shown in Figure 29-5. Lists in each category afford the clinician a systematic format for recording details pertinent to an accurate diagnosis. Appropriate descriptions are circled, followed by the necessary notations in the accompanying spaces. Tabular arrangement allows easier recording and comparison of diagnostic test data acquired from one tooth on different dates or from different teeth on the same visit. A pain intensity index (i.e., 0 to 10) or pain classification (i.e., mild +, moderate ++, severe +++) should be used to differentiate diagnostic test results.

Diagnosis

Careful analysis of accumulated examination data should result in the determination of an accurate pulpal and periapical diagnosis. Clinical conditions and the probable etiologic factors for the presenting problem are circled. Alternative modalities of therapy are considered and analyzed. The recommended treatment plan is circled, followed by a prognostic assessment of the intended therapeutic course.

Patient Consultation

Patients should be advised of each diagnosis and should consent to the treatment plan before therapy is instituted. Consultation should include an explanation of reasonable alternative treatment approaches and rationales and treatment consequences of each alternative therapy, including risks from nontreatment or delayed treatment that may affect the outcome of intended therapy. This discussion is documented by completing the checklist items.

Treatment

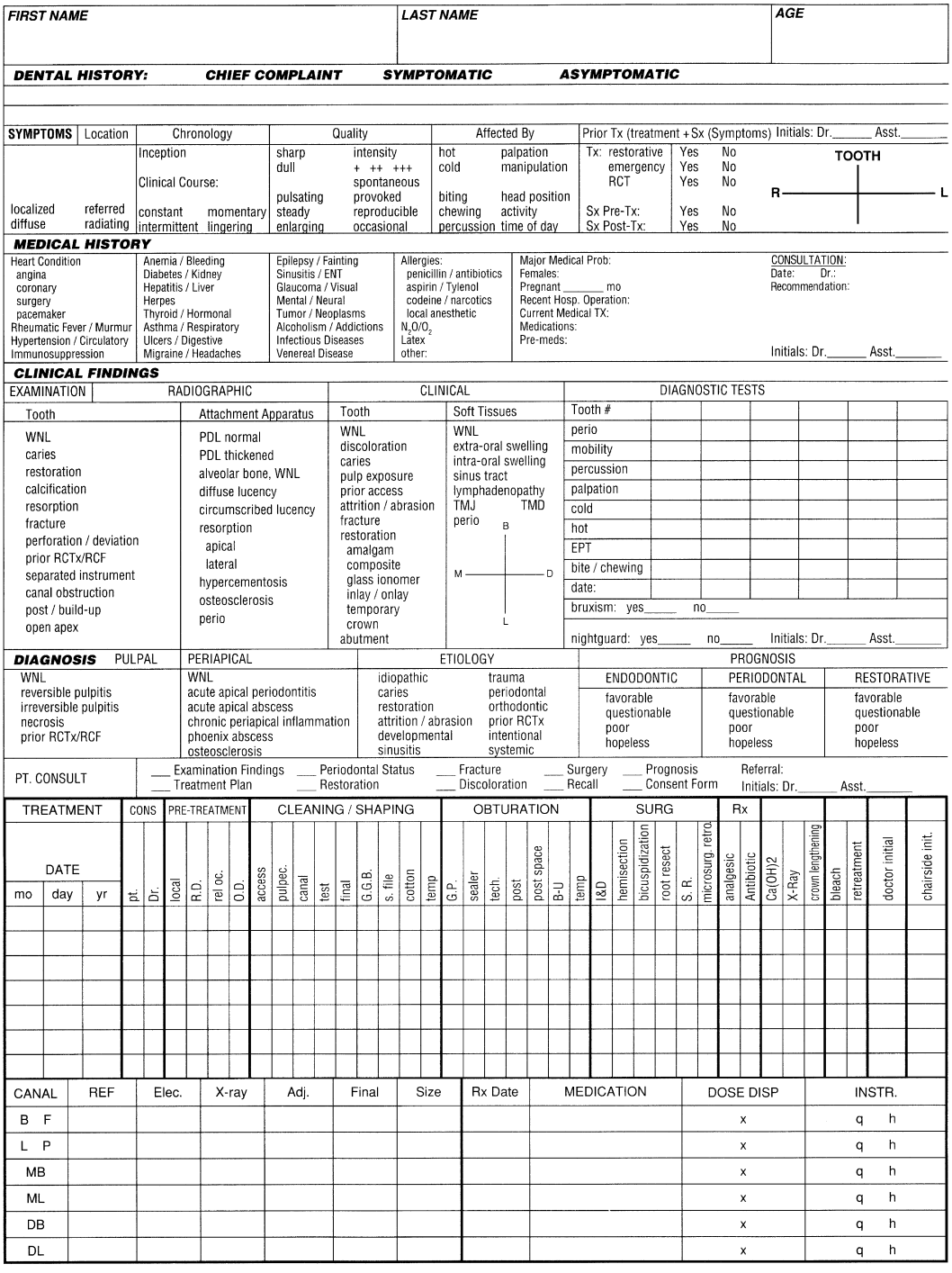
All treatment provided on a given date is documented by placing a checkmark (✓) within the designated procedural category. Only the most frequent retreatment procedures are included for tabulation. Descriptions of occasional procedures or explanatory treatment remarks should be entered in writing. A separate dated entry should be made for each patient visit, phone call, e-mail message, and fax communication (e.g., consultations with the patient or other clinicians) or correspondence (e.g., biopsy report, treatment completion letters).

Individual root canal lengths are recorded by (1) circling the corresponding anatomic designation and the method of length determination (e.g., radiograph or electronic measuring device), (2) writing the measurement (in millimeters), and (3) indicating the reference point. For any medication prescribed, refilled, or dispensed, the treatment record should show the date and type of drug (including dose, quantity, and instructions for use) in the treatment table under the heading "Rx." Figure 29-6 also shows a sample medication log. Periodic recall intervals, dates, and findings are entered in the spaces provided.

If the scope of the examination or treatment is intentionally limited, such as a screening examination or emergency endodontic therapy, the limited scope of the visit should be recorded. Otherwise, the chart appears as if the examination were superficial and treatment incomplete. If a suspicious apical lesion requires subsequent reevaluation, the clinician should record the future reevaluation date and differential diagnosis—for example, "Small apical lesion on #28. Reevaluate for changes in 2 months. Also, check for any root fractures." If this is not done, it appears (on the chart) as if the clinician ignored potential pathosis, such as suspected root fracture. A soft-tissue examination (including cancer check) is a standard part of any complete dental examination. E-mail, prescriptions, and correspondence should be documented with hard copies in the chart unless virtually all treatment records are stored in electronic format.[191,307]

Informed Consent Form

After endodontic diagnosis, the benefits, risks, treatment plan, and alternatives to endodontic treatment—including any patient refusal of recommended treatment and consequences of refused treatment—should be presented to the patient or the



FIRST NAME	LAST NAME	AGE

DENTAL HISTORY: **CHIEF COMPLAINT** **SYMPTOMATIC** **ASYMPTOMATIC**

SYMPTOMS	Location	Chronology	Quality	Affected By	Prior Tx (treatment + Sx (Symptoms) Initials: Dr._____ Asst._____
		Inception	sharp intensity	hot palpation	Tx: restorative Yes No
			dull + ++ +++	cold manipulation	emergency Yes No
		Clinical Course:	spontaneous		RCT Yes No
	localized referred		pulsating provoked	biting head position	
	diffuse radiating	constant momentary	steady reproducible	chewing activity	Sx Pre-Tx: Yes No
		intermittent lingering	enlarging occasional	percussion time of day	Sx Post-Tx: Yes No

TOOTH R L

MEDICAL HISTORY

Heart Condition angina coronary surgery pacemaker Rheumatic Fever / Murmur Hypertension / Circulatory Immunosuppression	Anemia / Bleeding Diabetes / Kidney Hepatitis / Liver Herpes Thyroid / Hormonal Asthma / Respiratory Ulcers / Digestive Migraine / Headaches	Epilepsy / Fainting Sinusitis / ENT Glaucoma / Visual Mental / Neural Tumor / Neoplasms Alcoholism / Addictions Infectious Diseases Venereal Disease	Allergies: penicillin / antibiotics aspirin / Tylenol codeine / narcotics local anesthetic N_2O/O_2 Latex other:	Major Medical Prob: Females: Pregnant _____ mo Recent Hosp. Operation: Current Medical TX: Medications: Pre-meds:	CONSULTATION: Date: Dr.: Recommendation: Initials: Dr._____ Asst._____

CLINICAL FINDINGS

EXAMINATION	RADIOGRAPHIC		CLINICAL		DIAGNOSTIC TESTS
	Tooth	Attachment Apparatus	Tooth	Soft Tissues	Tooth #
	WNL caries restoration calcification resorption fracture perforation / deviation prior RCTx/RCF separated instrument canal obstruction post / build-up open apex	PDL normal PDL thickened alveolar bone, WNL diffuse lucency circumscribed lucency resorption apical lateral hypercementosis osteosclerosis perio	WNL discoloration caries pulp exposure prior access attrition / abrasion fracture restoration amalgam composite glass ionomer inlay / onlay temporary crown abutment	WNL extra-oral swelling intra-oral swelling sinus tract lymphadenopathy TMJ TMD perio B M D L	perio mobility percussion palpation cold hot EPT bite / chewing date: bruxism: yes_____ no_____ nightguard: yes_____ no_____ Initials: Dr._____ Asst._____

DIAGNOSIS PULPAL	PERIAPICAL	ETIOLOGY		PROGNOSIS		
				ENDODONTIC	PERIODONTAL	RESTORATIVE
WNL reversible pulpitis irreversible pulpitis necrosis prior RCTx/RCF	WNL acute apical periodontitis acute apical abscess chronic periapical inflammation phoenix abscess osteosclerosis	idiopathic caries restoration attrition / abrasion developmental sinusitis	trauma periodontal orthodontic prior RCTx intentional systemic	favorable questionable poor hopeless	favorable questionable poor hopeless	favorable questionable poor hopeless

PT. CONSULT ___ Examination Findings ___ Periodontal Status ___ Fracture ___ Surgery ___ Prognosis Referral:
___ Treatment Plan ___ Restoration ___ Discoloration ___ Recall ___ Consent Form Initials: Dr._____ Asst._____

TREATMENT			CONS		PRE-TREATMENT					CLEANING / SHAPING										OBTURATION										SURG						Rx								
DATE																																									
mo	day	yr	pt.	Dr.	local	R.D.	rel oc.	O.D.	access	pulpec.	canal	test	final	G.G.B.	s. file	cotton	temp	G.P.	sealer	tech.	post	post space	B-U	temp	I&D	hemisection	bicuspidization	root resect	S. R.	microsurg. retro	analgesic	Antibiotic	Ca(OH)2	X-Ray	crown lengthening	bleach	retreatment	doctor initial	chairside init.

CANAL	REF	Elec.	X-ray	Adj.	Final	Size	Rx Date	MEDICATION	DOSE DISP	INSTR.
B F									x	q h
L P									x	q h
MB									x	q h
ML									x	q h
DB									x	q h
DL									x	q h

FIG. 29-5 Diagnosis and treatment form.

patient's guardian. This will document acceptance or rejection of treatment recommendations. The patient or guardian, along with a witness (who can be a staff member), should sign and date the consent form. Any informed-consent video should similarly be noted, if viewed. Subsequent changes in the proposed treatment plan should also be discussed and initialed by the patient to indicate continued acceptance and acknowledge the patient's understanding of any newly disclosed risks, alternatives, or referrals.

Despite a patient's confirmed signature on an informed consent form, a jury is free to decide that the patient never read the informed consent form's content before signing. For instance, the patient may claim it was impossible to have read the consent form, because he or she did not have reading glasses when signing and no clinician ever explained the form contents. Another frequent factual scenario is when the patient claims the staff stated it was a standard consent form that was a mere procedural formality requiring patient signature, rather than a detailed informed consent form. Consequently, numerous legal cases have been lost at trial on the issue of inadequate informed consent despite a signed patient consent form.

MEDICATION LOG: P. Ulp (Patient name) (415) 391-5353 (Phone number)

Medication allergies Sulfa drugs

Date	Medication	No. Tabs/ Caps/ Gel	Dosage/ quantity	Refill? (Y/N)	Rx Written-W Dispen.-D Phone-P	Phoned Rx? (pharmacy & phone #)	Staff Init	Dr. Init
6/19/15								
6/29/15								

FIG. 29-6 Medication log.

To obviate a patient's claim that no explanation ever occurred, a patient questionnaire can additionally be used. Patients can be instructed that unless they score 100%, proposed procedures will not be done (because patient understanding is imperative and essential for cooperation, including postoperative care). To be effective, the patient questionnaire should be relatively short and simple (Fig. 29-7). Also, the patient should be given an opportunity to correct any incorrect answers after the clinician reviews with the patient the reasons why the patient answered incorrectly. Note that the majority of answers are false. This precludes a patient from guessing the correct answer by assuming that all test questions are necessarily true.

Treatment Record: Endodontic Chart

A suggested chart is presented in Figure 29-8 to facilitate recording of information pertinent to the diagnosis, recommendations, and treatment of the endodontic patient. Systematic acquisition and arrangement of data from the patient questionnaire, along with clinical and radiographic examinations and careful recording of treatment performed, expedites accurate diagnosis and maximizes clinician efficiency. Suggested chart format and use are described in the sections that follow.

General Patient Data

Patient name, address, phone numbers, referring clinician, and chief complaint are printed or typed in the corresponding space at the patient's initial office visit. The patient's appointment and financial record are divided into two parts as follows:

1. The treating clinician or staff completes the first portion after the diagnosis and treatment plan have been formulated and presented to the patient. Tooth number and quoted fee are posted. The treatment plan is recorded by circling the appropriate description. Under "Special Instructions," specific treatment requests by the referring clinician are circled. Details of planned adjunctive procedures (e.g., hemisection, root resection) may be written in the adjacent space, along with information from the patient data section. The clinician can then use this information for general reference during future treatment. The secretary or treatment coordinator can also use this information when scheduling appointments and establishing financial arrangements.
2. Business personnel complete the second portion. Financial agreements, third-party coverage, account status, and appointment data (including the day, date, and scheduled procedure) are recorded.

ENDODONTIC INFORMED CONSENT QUESTIONNAIRE

In order to help your understanding of endodontic benefits, risks, and alternatives, please answer the following questions by checking the true box if the statement is correct and the false box if the statement is incorrect. In the event that your answers suggest the need for further explanation, Dr. Cohen will review root canal treatment (and options) with you and answer any questions.

Please feel free to ask questions so that you may feel comfortable with your understanding of the surgical endodontic procedure on tooth number 19 and your consent to endodontic surgery.

	True	False
1. Endodontic surgical success is guaranteed.	☐	☐
2. Since endodontic surgery is carefully done, there is no chance of any infection occurring after surgery.	☐	☐
3. As with any surgical procedure involving the human body, there are associated risks. One of these risks is numbness of the lower lip or chin on the side that the surgery is done. Any numbness is usually temporary but, on rare occasions, it can be permanent.	☐	☐
4. Daily oral hygiene care after endodontic surgery is unnecessary.	☐	☐
5. Dr. Cohen is unavailable for emergency calls after business hours.	☐	☐

I understand that I may ask Dr. Cohen any question I may have regarding endodontics at any time. Dr. Cohen has answered all of my questions, if any, to my satisfaction.

Date: _____ Patient's signature _____ Witness

FIG. 29-7 True/false endodontic informed consent form.

Either the clinician or the assistant may complete portions of the diagnosis and treatment sections, but *the clinician should review and approve all entries*.

Dental History

Chief complaint should include whether the patient is symptomatic at the time of examination or in the recent past and what medications the patient is currently taking for pain relief. Narrative facts regarding the presenting problem are then recorded. Additional details of the chief complaint obtained during successive questioning are recorded by circling the applicable word within each symptom parameter. The pain intensity index (i.e., 0 to 10) or pain classification (i.e., mild +, moderate ++, severe +++) should be registered alongside the appropriate description. For accurate assessment of the effects of prior dental treatment pertaining to the examination site, a summarized account of these procedures should be documented. In addition, all pretreatment signs and symptoms should be described. It is essential that the patient comprehend all communications with the clinician and staff. If there is any language comprehension difficulty, the clinician should ask the patient whether he or she understands completely the language the clinician is speaking. To verify patient comprehension, the clinician should ask the patient to repeat important information that has been conveyed. A clinician- or patient-provided interpreter may be necessary if language problems exist.

Medical History

More medical history information is obtained from a patient-administered questionnaire than from the clinician obtaining

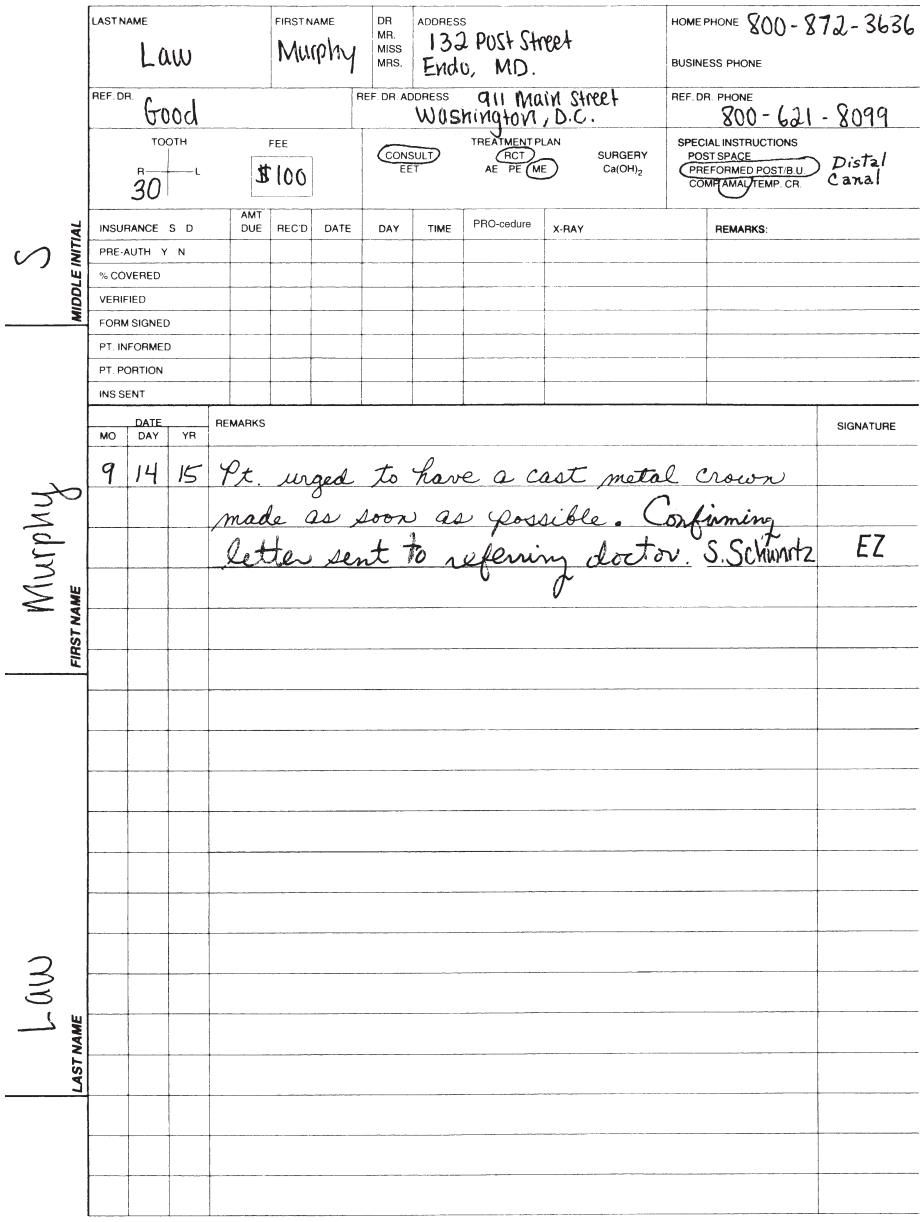
an oral history solely from the patient.[43,286] Maximum information is obtained if the clinician reviews with the patient the written health history form the patient has completed.

Information (e.g., personal physician's name, address, and phone number; patient's age; date of last medical examination) is recorded for reference. The clinician can obtain a detailed medical history by completing a survey of common diseases and disorders along with a comprehensive review of corresponding organ systems and related pathosis. Specific entities that have affected the patient are circled. Essential remarks regarding these entries (e.g., details of consultations with the patient's physician) should be documented on an attached blank sheet with dated treatment notes attached to the back of the chart (see Fig. 29-2). A review of the patient's medical status (including recent or current conditions, treatments, and medications) completes the medical history. Updated medical histories that the patient signs or initials should be done at least annually and at reevaluation visits.

Excessive alcohol consumption can contribute to a higher incidence of restoration corrosion from chronic regurgitation and vomiting from gastritis associated with alcohol abuse.[139] Corrosion can lead to coronal marginal leakage predisposing to endodontic failure.[305] Accordingly, medical histories should include histories of gastric reflux disease, bulimia with associated habitual vomiting, gastritis, and excessive alcohol intake.

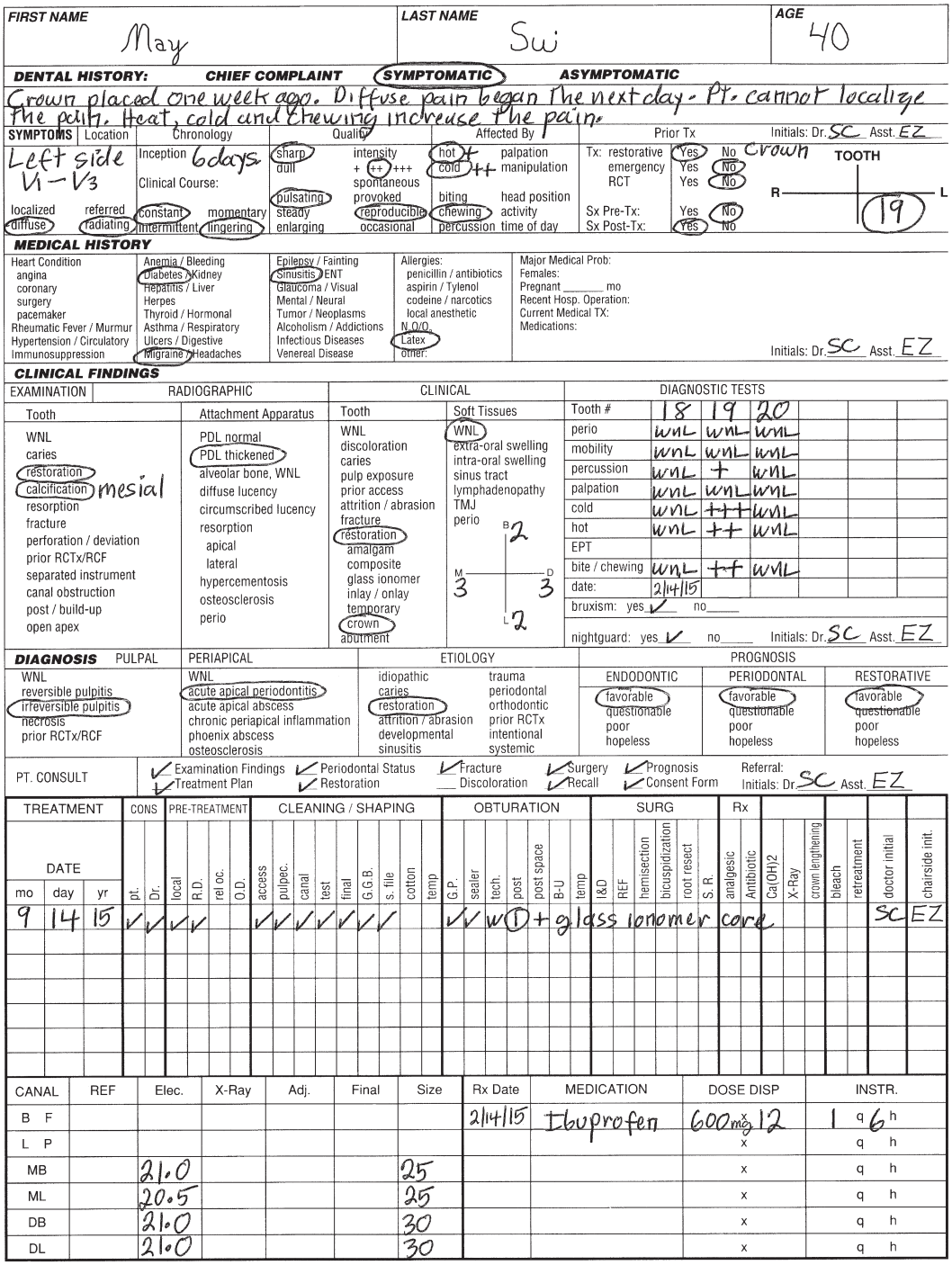
Periodontal Disease

Nearly half of American adults older than age 30 years have periodontal disease.[241] Periodontal disease was higher in men



A

FIG. 29-8 A, Progress notes.


FIRST NAME May
LAST NAME Su
AGE 40
DENTAL HISTORY: CHIEF COMPLAINT SYMPTOMATIC ASYMPTOMATIC
Crown placed one week ago. Diffuse pain began the next day - Pt. cannot localize the pain. Heat, cold and chewing increase the pain.
SYMPTOMS Location Chronology Quality Affected By Prior Tx
Initials: Dr. SC Asst. EZ
Left side #1-#3
Inception 6 days
Clinical Course:
localized referred
diffuse radiating
constant momentary
intermittent lingering
sharp dull
intensity + ++ +++
spontaneous provoked
pulsating steady
enlarging reproducible
occasional
hot + cold ++
palpation manipulation
biting head position
chewing activity
percussion time of day
Tx: restorative Yes No
emergency Yes No
RCT Yes No
Sx Pre-Tx: Yes No
Sx Post-Tx: Yes No
Crown
TOOTH
R L
19
MEDICAL HISTORY
Heart Condition
angina
coronary
surgery
pacemaker
Rheumatic Fever / Murmur
Hypertension / Circulatory
Immunosuppression
Anemia / Bleeding
Diabetes / Kidney
Hepatitis / Liver
Herpes
Thyroid / Hormonal
Asthma / Respiratory
Ulcers / Digestive
Migraine / Headaches
Epilepsy / Fainting
Sinusitis / ENT
Glaucoma / Visual
Mental / Neural
Tumor / Neoplasms
Alcoholism / Addictions
Infectious Diseases
Venereal Disease
Allergies:
penicillin / antibiotics
aspirin / Tylenol
codeine / narcotics
local anesthetic
N2O/O2
Latex
Other:
Major Medical Prob:
Females:
Pregnant _____ mo
Recent Hosp. Operation:
Current Medical TX:
Medications:
Initials: Dr. SC Asst. EZ
CLINICAL FINDINGS
EXAMINATION RADIOGRAPHIC CLINICAL DIAGNOSTIC TESTS
Tooth
WNL
caries
restoration
calcification mesial
resorption
fracture
perforation / deviation
prior RCTx/RCF
separated instrument
canal obstruction
post / build-up
open apex
Attachment Apparatus
PDL normal
PDL thickened
alveolar bone, WNL
diffuse lucency
circumscribed lucency
resorption
apical
lateral
hypercementosis
osteosclerosis
perio
Tooth
WNL
discoloration
caries
pulp exposure
prior access
attrition / abrasion
fracture
restoration
amalgam
composite
glass ionomer
inlay / onlay
temporary
crown
abutment
Soft Tissues
WNL
extra-oral swelling
intra-oral swelling
sinus tract
lymphadenopathy
TMJ
perio
B 2
M 3 D 3
L 2
Tooth # 18 19 20
perio WNL WNL WNL
mobility WNL WNL WNL
percussion WNL + WNL
palpation WNL WNL WNL
cold WNL +++ WNL
hot WNL ++ WNL
EPT
bite / chewing WNL ++ WNL
date: 2/14/15
bruxism: yes ✓ no
nightguard: yes ✓ no Initials: Dr. SC Asst. EZ
DIAGNOSIS PULPAL PERIAPICAL ETIOLOGY PROGNOSIS
WNL
reversible pulpitis
irreversible pulpitis
necrosis
prior RCTx/RCF
WNL
acute apical periodontitis
acute apical abscess
chronic periapical inflammation
phoenix abscess
osteosclerosis
idiopathic
caries
restoration
attrition / abrasion
developmental
sinusitis
trauma
periodontal
orthodontic
prior RCTx
intentional
systemic
ENDODONTIC favorable questionable poor hopeless
PERIODONTAL favorable questionable poor hopeless
RESTORATIVE favorable questionable poor hopeless
PT. CONSULT
✓ Examination Findings ✓ Treatment Plan
✓ Periodontal Status ✓ Restoration
✓ Fracture ___ Discoloration
✓ Surgery ✓ Recall
✓ Prognosis ✓ Consent Form
Referral:
Initials: Dr. SC Asst. EZ
TREATMENT DATE mo day yr
CONS pt. Dr.
PRE-TREATMENT local R.D. rel oc. O.D.
CLEANING / SHAPING access pulpec. canal test final G.G.B. s. file cotton temp
OBTURATION G.P. sealer tech. post post space B-U temp
SURG I&D REF hemisection bicuspidization root resect S. R.
Rx analgesic Antibiotic
Ca(OH)2 X-Ray crown lengthening
bleach retreatment doctor initial chairside init.
9 14 15 ✓ ✓ ✓

Each tooth undergoing endodontic therapy (and adjacent teeth) should be probed with a calibrated periodontal instrument to obtain six measurements per tooth. Pockets of 4 mm or greater should be recorded on the patient's chart. If no pockets exist, WNL (within normal limits) or a similar abbreviation should be noted. Mobility should also be charted and classified class I, II, or III. Gingival recession, furcations, and mucogingival deficiencies should also be recorded.[9,10]

A clinician who treats with endodontic success but ignores loss of periodontal attachment may misdiagnose or fail to appreciate the risk of failure because of poor periodontal prognosis. It bears repeating that the endodontic treating clinician should not assume an adequate periodontal evaluation has been performed by another clinician or the referring clinician. Instead, an independent periodontal evaluation should be done with a calibrated periodontal probe and results recorded before initiating endodontics, including noting absence of pockets or within normal limits.

If clinically significant periodontal disease is present, the endodontic treating clinician should consult with the restorative clinician to determine whether the periodontal disease will be properly treated or referred to a periodontist in conjunction with endodontic treatment. A patient should be advised of any compromise of the endodontically treated tooth's periodontal status to comply with required informed consent disclosure.

Drug History

A current drug history, both written and oral, is necessary to alert the clinician to any potential for interaction between any newly prescribed drugs and any other medication, supplements, or over-the-counter medication the patient is already taking.

In patients aged 57 through 85, 40% potentially are at risk of a major drug reaction—half involving various prescription medications and the other half involving anticoagulant drug reactions. About half of prescription users also concurrently take over-the-counter medications plus dietary supplements.[190,235]

Risks of drug interactions or side effects incidence may not be discovered or appreciated in premarket research-controlled drug trials for newly marketed drugs, despite FDA approval. For example, rofecoxib (Vioxx) was withdrawn from the market in September 2004 after increased incidences of myocardial infarcts and strokes manifested in a postmarket research study of 2600 patients. Merck's voluntary recall followed an FDA researcher's claim that the FDA tried to ostracize and intimidate him after he raised safety concerns over Vioxx.[33] Currently, pharmaceutical manufacturers budget more for marketing than research and development.[121] Some drug manufacturers have suppressed publication of adverse research.[55,208,337] The Patient Protection and Affordable Care Act of 2010 (Section 6002) requires drug companies to disclose payments to medical/dental professionals who tout their drugs in seminars or publications and receive compensation greater than $500.[245]

For newly marketed drugs, the small number of premarket patients studied may lack sufficient statistical power to expose serious side effects that occur infrequently but are nonetheless life threatening.[337] For instance, mibefradil (Posicor) was withdrawn from the market after 1 year because it increased plasma concentrations of 25 other co-administered drugs, including erythromycin.[235,239] In another example, the popular prescription nighttime heartburn drug Propulsid (cisapride) was linked to 70 deaths and more than 270 significant negative reactions.[328] Other examples include the risk of esophageal cancer with bisphosphonate[337,341] and bisphosphonate-associated osteonecrosis.[92,287] In 2008, the FDA added a black box warning label for Levaquin-associated risks of tendonitis (www.levaquin.com/levaquin/medication).

Preoperative and Postoperative Radiographs

Pretreatment, midtreatment, and posttreatment radiographs or digital images are essential for endodontic diagnosis and treatment:

1. A current, pretreatment, interpretable periapical radiograph of adequate diagnostic quality is mandatory.
2. Measurement films or digital images are necessary to verify canal length (which can be supplemented with an electronic canal-measuring device) and the apical extent of the gutta-percha fill to the radiographic apex.
3. Posttreatment radiographs are essential for determining the adequacy of the endodontic seal or if further treatment is necessary (see Chapter 8).

Digital Radiography

Digital radiographic endodontic applications are ever increasing in accuracy.[142] The standard of care does not currently require digital imaging, because traditional silver halide radiographic film is a reasonable alternative. When there is more than one reasonably acceptable practice modality, a clinician who chooses either modality meets the standard of care. Figure 29-9, *A*, represents a distal open margin on tooth #30 (shown digitally) that is not evident in part *B* with plain-film radiography.

Advertised claims of 80% reduction in radiation with direct digital radiography (rather than film) assume the following[53,142]:

1. Ultraspeed D-speed film is used. (Ektaspeed E-speed film and the newer F-speed film reduce radiation 50% and 60%, respectively, while producing images of comparable quality to D speed.)[4]
2. No rectangular collimation is used, which reduces exposure another 30%.
3. No extra radiographs are taken to compensate for a smaller area. For instance, a 2 charged-coupling device (CCD) sensor has an active area smaller than size 1 film and may require an extra image with additional radiation exposure.

A digital x-ray image should never be modified to "enhance" the radiographic appearance of the original image. X-ray modification can be detected. In litigation, it can prove devastating to the clinician's defense because the clinician's credibility of events or recorded entries are not believable, particularly if the radiograph is Photoshopped.

Periapical (PA) radiography is limited to only two dimensions. Newer 3D imaging systems for dental radiology use x-ray beams that are cone shaped. Cone-beam computed tomography can provide a more nearly accurate diagnosis.[75,122,157] In Figure 29-10, *A* to *C*, the PA does not show the transported canal and extent of short fill, which is more evident in the CBCT. Each CBCT scan produces three image views: axial, coronal, and sagittal.

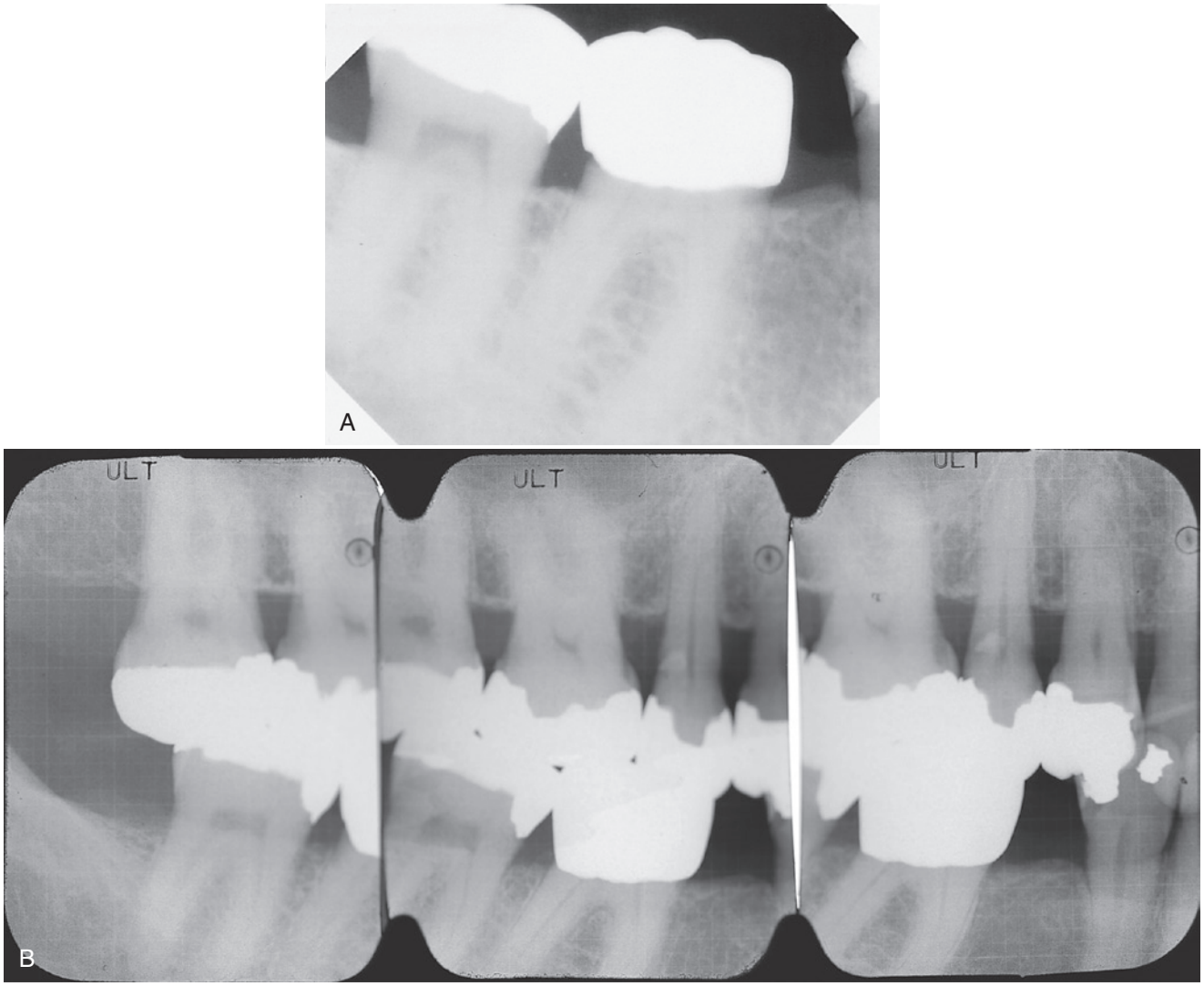


FIG. 29-9 **A,** Distal open margin on tooth #30 (shown digitally). **B,** Distal open margin on tooth #30 not evident with plain-film radiography.

Radiation Safety

The American Dental Association (ADA) Council on Scientific Affairs recommends the dentist recognize the importance of keeping radiation exposure as low as reasonably achievable (ALARA). Accordingly, CBCT imaging should not be performed for screening purposes but rather for specific diagnostic and treatment usage.[20]

The Food and Drug Administration provides recommendations for dental cone-beam computed tomography.[99] The American Academy of Oral and Maxillofacial Radiology provides indicated use of the CBCT for implant placement.[317] In the future, magnetic resonance imaging (MRI) may supplant or augment CBCT because its imaging quality equals and in certain instances surpasses that of CBCT.[189]

Children have greater radiosensitivity because of developing cells in their young bodies. Thus, CBCT imaging radiation dosage should be considered for young patients in assessing the need for a CBCT.

Computed tomography (CT) scans enable evaluation of the true extent of lesions and their spatial relationship to important anatomic landmarks.[97] Figure 29-11, *A* to *C*, shows a bifid inferior alveolar nerve canal (IANC) that is not apparent on Panorexes. CT scans may provide information important for retreatment decisions, especially apical surgery. CBCT[75,201,204] showed significantly more lesions than PA radiography, including expansion of lesions into the maxillary sinus, sinus membrane thickening, and missed canals; PA radiography missed 34% of these lesions.[201,204] Artificial bone defects in the antral surface were not detected with PA radiography. Because of overlapping roots, only 1 out of 14 furcation defects in upper molars were seen on PA radiography, but CT scans were able to identify all furcal defects. Second molars proved to be the most difficult for detecting lesions with PA radiography alone. Twenty-three teeth with lesions expanding into the maxillary sinus were detected by CBCT, of which only two teeth were seen with PA radiography. Thirty-five teeth with membrane thickening were identified with CBCT, of which 16 teeth were also detected with PA radiography. Fifteen teeth with missing canals were detected with CBCT, of which only four teeth were identified with PA radiography. In a study that compared the efficacy of PA radiography and CBCT in detecting periapical lesions in

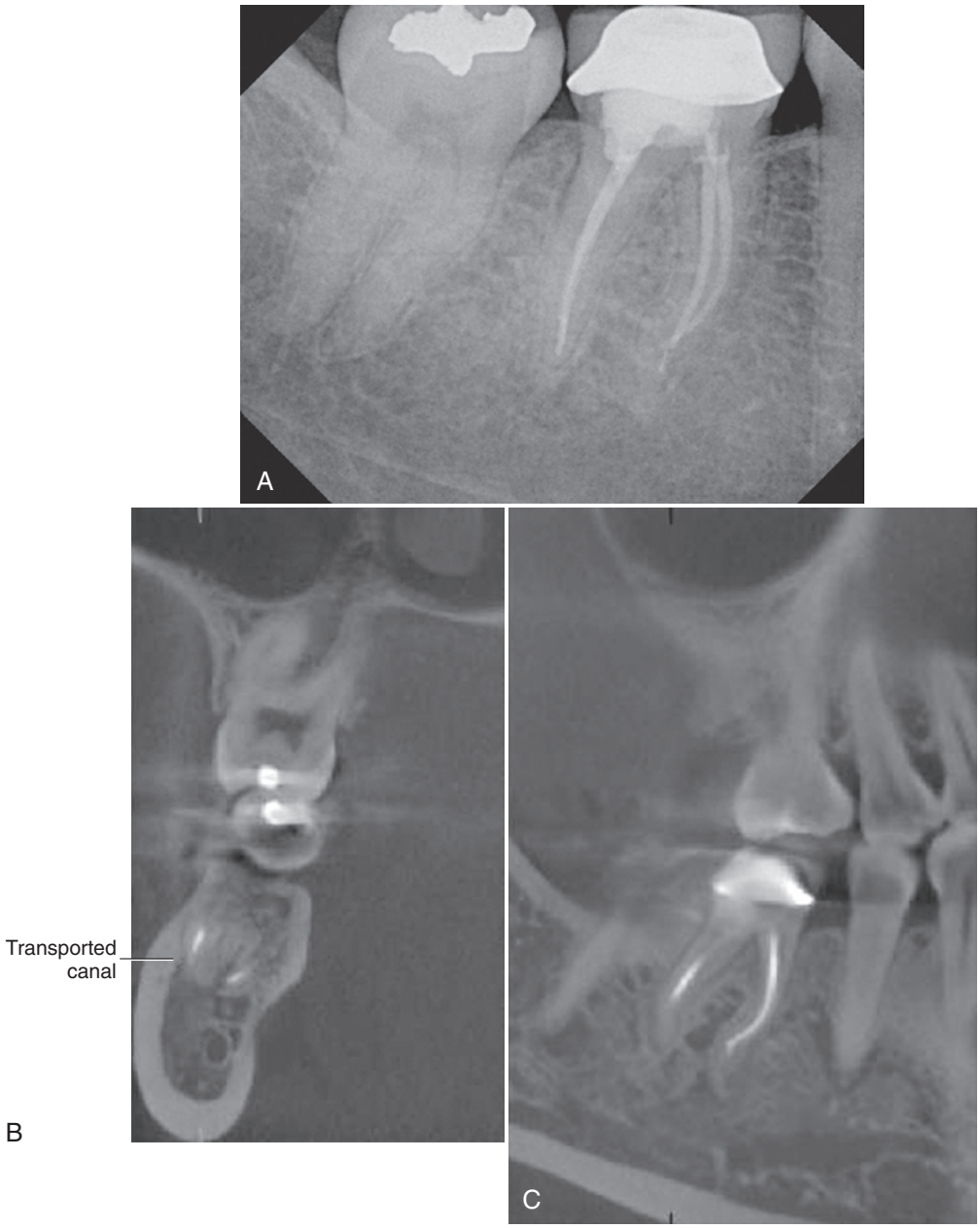


FIG. 29-10 A, Periapical (PA) view, which does not show full extent of transported and short-filled canal. Both deficiencies evident in B and C with cone-beam computed tomography (CBCT). B, CBCT Accuitomo of transported canal. C, CBCT Accuitomo of short-filled canal.

maxillary premolars and molars for apical surgery,[204] Lofthag-Hansen showed that 38% of maxillary posterior teeth lesions were undetected by PA radiography, despite the fact that an additional different angulated PA radiograph was taken.[201] Lesions associated with apices near the sinus floor had a higher probability of being missed with PA than lesions associated with apices located away from or overlapping the sinus floor. Expansion of the lesion into the maxillary sinus, thickening of the sinus membrane, missed canals, and presence of apicomarginal communications were more frequently detected with CBCT than with PA radiography.[201]

Apicomarginal communication is an important predictor for the success rate of apical surgery, including evidence of undetected vertical fractures necessitating extraction; 83% of apicomarginal communications were not seen with PA radiography in the Lofthag-Hansen study.

If the radiograph shows a small distance between the periapical lesion and the sinus floor when the bony wall is thin, there is a high probability that an oral antral communication (OAC) can result, unless skillful care avoids sinus penetration during apical surgery. CBCT also allows the clinician to study the topography of the bony defect and assess whether the use of guided tissue membranes would be beneficial. Precise morphometric assessment of osseous relationships to the sinus is often inadequate with periapical radiography. Thus, CBCT is invaluable in treatment planning for apical surgery, as 70% of cases studied revealed clinically relevant information not found in PA radiography.[204]

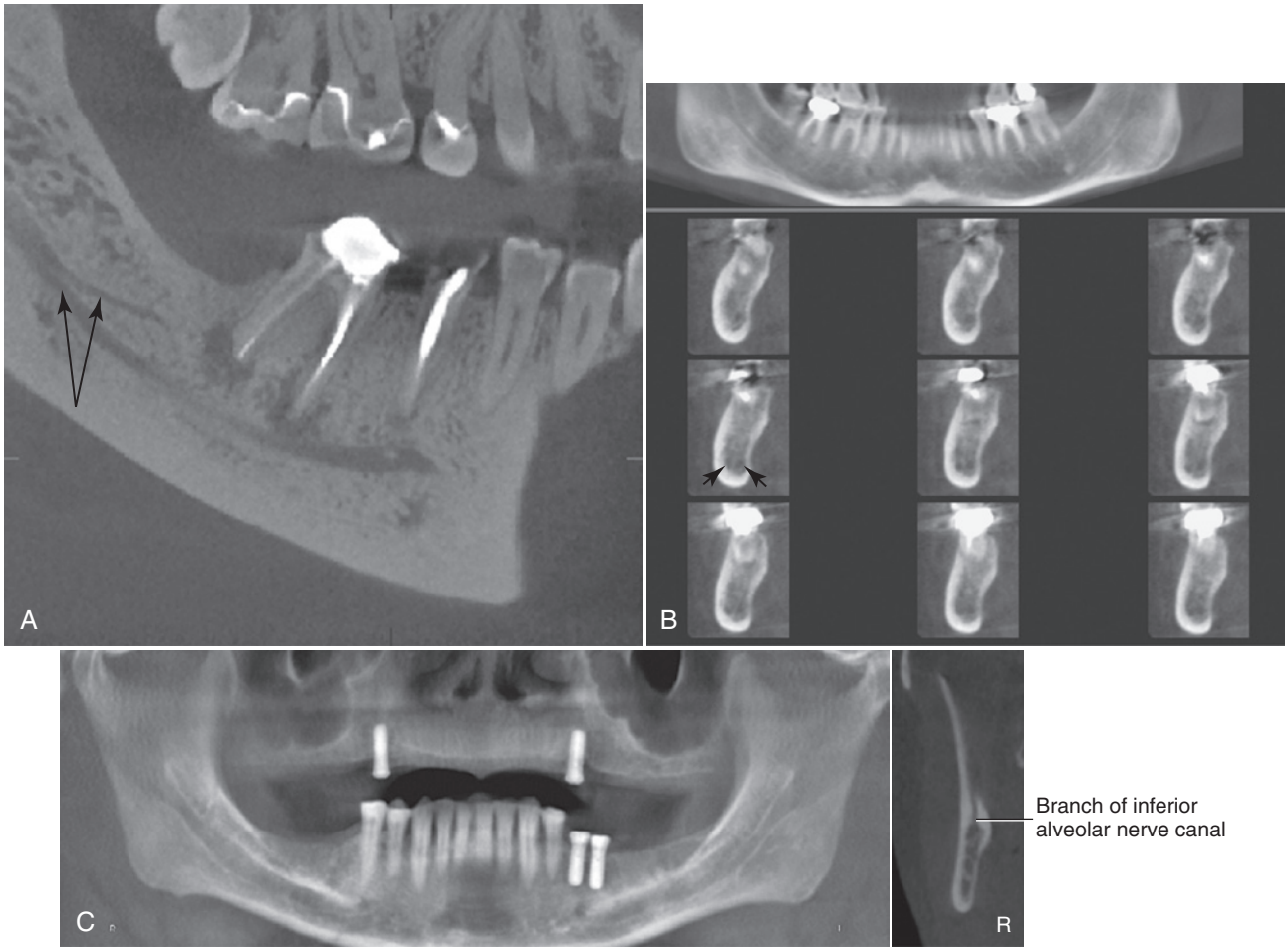


FIG. 29-11 A, Bifid inferior alveolar nerve canal (IANC) visible with cone-beam computed tomography (CBCT). B, Bifid canal visible with CBCT but not shown on Panorex. C, Branch of IANC apparent on CBCT but not shown on Panorex. (Courtesy Gurminder Sidhu, DDS, MS.)

The probability of detecting lesions with PA alone was limited for teeth with apices in close contact with the floor of the maxillary sinus, for molars (in particular second molars), and when bone thickness between lesion and sinus was less than or equal to 1 mm. Findings of lesion expansion into the sinus, sinus membrane thickening, missed canals, and presence of apicomarginal defects are more frequently diagnosed with CBCT than PA.[75,204]

ENDODONTICS AND HEART DISEASE

Associations between chronic infections such as periodontal diseases and coronary heart disease (CHD) have been suggested.[280] Conversely, supporting evidence linking root canal–filled teeth or teeth with periapical disease to CHD is lacking.[119]

If a patient's medical history shows valvular heart defects with regurgitation, the clinician should consult with the patient's physician to determine the necessity of antibiotic prophylaxis against infective endocarditis in accordance with current American Heart Association (AHA) guidelines.[334] If the physician does not appear knowledgeable about those guidelines, the clinician should provide a copy of the guidelines to the physician. If the physician advises deviation from AHA guidelines, the clinician should inquire why the physician feels that this is appropriate. The clinician should also record any discussions with the physician in the patient's chart and confirm the discussion and the physician's recommendation in writing (by fax and letter to the physician).

Communications with the physician should be specific, because dental treatment risks, such as overextension of files or filling material sealants that occur nonsurgically and may enter the bloodstream, may not be fully understood or appreciated. A sample format is shown in Figure 29-12.

Abbreviations

Abbreviated records can be frustrating if the clinician is unable to decipher one's own or other clinicians' handwritten entries, so the clinician should use standard or easily understood abbreviations. Pencil entries are legally valid, but ink entries are less vulnerable to a plaintiff's claim of erasure or alteration. A short pencil is better than a long memory; records remember, but patients and clinicians may forget.

A sample of a completed endodontic chart (Fig. 29-13) illustrates its proper use. Box 29-1 lists a key to the abbreviations.

Computerized Treatment Records

Clinicians are increasingly using computerized record storage as described in this chapter.[282] To avoid a claim of record falsification, whatever computer system is used should be able to

Dear Physician:

Your patient requires potentially invasive dental treatment that will likely result in transient bacteremia. Does the patient have a heart valve defect that increases the risk of infective endocarditis? If so, please advise of the diagnosed defect and recommended prophylactic antibiotics and dose for endodontic treatment or surgery with potential to invade the bloodstream. If you require the patient to have a current cardiac evaluation or echocardiogram to provide an answer, please advise both me and the patient, preferably in writing. Please also advise if your recommendation is in accordance with the enclosed American Heart Association current guidelines concerning bacterial endocarditis relating to dental treatment.

Thank you for your anticipated cooperation.

(Enclosure: current AHA guidelines)

FIG. 29-12 Sample of written physician recommendations.

Clinician and Staff Initials Log

Clinician	Staff	Initials	Date
Edwin J. Zinman		EZ	6-01-15
	Alyssa Malfatti	AM	6-01-15
Stephen Cohen		SC	6/1/15
	Kelly Fishman	KF	7/1/15

FIG. 29-13 Clinician and staff initials log.

demonstrate that records indicating earlier treatment were not recently falsified. Software technology, such as the "write only read memory" (WORM) system, used to identify computer data tampering, is not foolproof because it cannot detect tampering where an entire disk of recent origin has been substituted for an earlier version. Periodically, a hard copy of computer data should be printed out, hand initialed, dated in ink, and stored in the chart to provide written verification of the computer records if the office is a virtual paperless office.[307]

Health Information Technology for Economic and Clinical Health (HITECH) Act Pitfalls

Affected patients must be notified if there is a breach in the electronic record storage system.[148] Electronic records have the following pitfalls:

- Data entry in the wrong patient record
- Incorrect data entry in the patient record

Incorrectly entered information or information that was not entered at all was reported as the cause of the majority of errors. The absence of an undecipherable scrawl is designed to reduce errors. Whether the result of fat fingers or providers pushed for time, mistakes will still occasionally occur. Some of the things that make paperless electronic records desirable—transparency, the ability of more people to view the same information, and more immediate accessibility—may cause any electronic record mistakes risk to be amplified.

Health Insurance Portability and Accountability Act (HIPAA)

The Department of Health and Human Services' Security and Electronic Signature Standards rule is part of the Health Insurance Portability and Accountability Act (HIPAA) of 1996. This act emphasizes the safeguarding of clinicians' health care information and permits a patient to obtain a copy of the requesting patient's records. Most states have authorized digital electronic signatures as binding for most contracts and orders. Digital signatures with electronic transactions are authorized by the Third Millennium Electronic Commerce Act (also called the Electronic Signature in Global and National Commerce Act).[323]

Pertinent HIPAA requirements follow:

1. Original records and radiographs should not be released but only copies provided to the patient. A reasonable copying charge may be requested before or after release of the copies.
2. A patient may amend health information in the chart that the patient believes to be incorrect or incomplete. The clinician may respond to the patient's proposed chart amendments.
3. Notice of office privacy practices should be provided to the patient, and the patient should acknowledge receipt in writing.
4. If privacy rights are violated, the patient can file a grievance with the secretary of the Department of Health and Human Services. Patients cannot be penalized for complaining.
5. Confidential dental and medical information may be shared with other clinicians, based on a need to know.
6. All e-mail communications between clinician and patient must use a secure network and be stored on a secure server.[282]

Record Size

Although there is little harm in recording too much information, there is great danger in recording too little. Standard $8\frac{1}{2} \times 11$ inch or larger clinical records possess the advantage of providing the treating clinician adequate space for clinical notes.

Identity of Entry Author

Either a clinician or an auxiliary can chart clinical entries unless state law indicates otherwise. Some states require clinicians and assistants to sign or initial each treatment entry.[58] What is important is that the correct clinical information is recorded. Each person who makes a clinical chart entry should record the date and initial the entry. Otherwise, the author's identity may be forgotten if the individual who recorded the entry is needed in a legal proceeding. For instance, initialing the entry makes it easier to identify the particular clinician or auxiliary who, after the original recording, is now employed elsewhere. A separate log of clinician and staff initials should be created and stored for later comparison to identify the entrant's initials (see Fig. 29-13).

BOX 29-1

Standard Abbreviation Key

Ab	=	Antibiotic
ABS	=	Abscess
Access	=	Access cavity
AG or AmAL	=	Amalgam
AE	=	Air embolus
analg.	=	Analgesic
Apico	=	Apicoectomy
B-U	=	Buildup of tooth
Bisp	=	Bisphosphonate
BW	=	Bitewing
Canal	=	Identify canal that has been cleaned and shaped
CBCT	=	Cone-beam computed tomography
Comp	=	Composite
Cotton	=	Placed in pulp chamber between treatments
CRN	=	Crown
Dx	=	Diagnosis
ENDO	=	Endodontics
EPT	=	Electric pulp test
Epin	=	Epinephrine
Final	=	Final file
Fx	=	Fracture
G.G.B.	=	Gates-Glidden bur
G.P.	=	Gutta-percha
I & D	=	Incision and drainage
I.P.	=	Irreversible pulpitis
L.A. or local	=	Local anesthetic
Mm	=	Millimeters
O+D	=	Open and drain
P.A.	=	Periapical
Perio	=	Periodontal
Post	=	Preformed, custom, or transilluminated post
Pt	=	Patient
Pulpec	=	Pulpectomy
R.D.	=	Rubber dam
Rel occ	=	Relieved occlusion
resorp.	=	Resorption
Retro	=	Retrograde procedure
S/R	=	Suture removal
s.file	=	Serial filing
S/D	=	Single insurance or dual coverage
Sealer	=	Sealer type used
Sepr	=	Separated
Sutr	=	Suture
SX	=	Symptom
tech.	=	Technique for canal obturation
Temp	=	Temporary restoration
Test	=	Test file
Tx	=	Treatment
WNL	=	Within normal limits

Patient Record Request

Patient requests for record transfers or copies must be honored. It is unethical to refuse to transfer patient records, on the patient's request, to another treating clinician.[18] Moreover, refusing to provide patient records is illegal in some states, subjecting the clinician to discipline and fines should the records not be provided to the patient upon written request, even if the balance is outstanding.[62]

Patient education pamphlets may be used in litigation as evidence that a patient was properly informed and given endodontic alternatives but instead chose extraction. Such pamphlets include the American Dental Association's (ADA's) *Your Teeth Can Be Saved by Endodontic Root Canal Treatment* and the American Association of Endodontists' (AAE's) *Your Guide to Endodontic Treatment, Your Guide to Endodontic Retreatment,* and *Your Guide to Endodontic Surgery*. The clinician should indicate in the patient's chart which particular pamphlets the patient was provided or which video was shown, because this may prove useful should the patient later deny being informed of endodontic alternatives.

Patient Education Materials

The use of educational materials may improve health behavior, enhance patient satisfaction, and facilitate the consultation processes. Consequently, the patient is better informed if patient questions are answered after prompting with informative educational materials.

Postoperative Instructions

It is unlikely a patient will remember oral postoperative instructions unless accompanied with written instructions. After endodontic procedures, the patient may be sedated or affected by analgesic drugs. Accordingly, written postoperative instructions are beneficial. Emergency phone numbers to contact the treating clinician should be included on the form. Written instructions reduce postoperative morbidity and pain and improve patient compliance.[6] Document that both written and oral postoperative instructions were provided. If treating in close proximity to the IANC, place a precaution, such as seen in Figure 29-14, in the postoperative instruction sheet.

> If numbness persists the next morning or suddenly within three days following root canal treatment, immediately telephone our office so we may treat or refer to another specialist to surgically remove any root canal filling material overfill impinging upon the sensory jaw nerve, causing altered sensation to the lip and/or chin.

FIG. 29-14 Postoperative instruction sheet.

Recording Referrals

Every clinician, including the endodontic specialist, has a duty to refer under appropriate circumstances. If consultations with additional experts or specialists become necessary, referrals should be recorded lest they be forgotten or refused. Carbonless, two-part referral cards allow the clinician to provide an original referral slip to the patient while retaining a copy for the patient's chart. The clinician or staff member should document the fact that the original referral card was given or mailed to the patient and record the name of the person who provided the referral card and the date on which it was provided. A copy of the referral card should also be sent to the referred doctor. If the patient fails to keep the referral appointment, this copy will provide proof that a referral was made. The clinician should request that the patient and the referred clinician report back if the referral appointment is canceled. Staff should calendar to verify that the referral consultation occurred.

Dental Fees Related to Quality Care

The link between health care fees and the quality of health care remains unclear.[156]

Record Correction

Records must be complete, accurate, legible, and dated. All diagnoses, treatments, and referrals should be recorded. Chart additions may expand, correct, define, modify, or clarify (as long as they are currently dated to indicate a belated entry).

To correct an entry, the clinician should draw a line through (but not erase or obscure) the erroneous entry. The correction should then be written on the next available line and dated. Handwriting and ink experts use ink chemical tags, age dating, and infrared technology to prove falsified additions, deletions, or substitutions. If records are proved to be falsified, the clinician may be subject to punitive damages in civil litigation. In addition, the clinician may be subject to license revocation for intentional misconduct.[58] Professional liability insurance policies may defend but will usually not indemnify punitive damages if the clinician is found to have committed fraud or deceit.[56,57,61,77] In addition, insurance carriers may deny renewal of professional liability coverage to a clinician who fraudulently alters dental records.

If an erroneous entry occurs, the clinician should add another entry dated as a late entry to demonstrate later corrected information. Figure 29-15 gives an example of the proper method of belated record correction.

When patient records have been requested or subpoenaed, it is wise to refrain from examining in detail prior to copying to avoid the temptation to clarify or belatedly add an entry. Alteration of clinical records is a cause of large settlements because the clinician's credibility is suspect. Also the jury may be similarly instructed. For instance, California Jury Instruction 204 regarding "Willful Suppression of Evidence" states as follows:

You may consider whether one party intentionally concealed or destroyed evidence. If you decide that a party did so, you may decide that the evidence would have been unfavorable to that party.

- 10-13-15-Corrected entry for 10-12-15. (Entry should have recorded): Hot, cold, and percussion tests plus one. Recommend re-evaluation in 24 hours and repeat pulpal tests. Next visit scheduled 10/13/15 at 9 a.m.

FIG. 29-15 Belated record correction.

Spoliation

Spoliation is the destruction or significant alteration of evidence, or the failure to preserve property for another's use as evidence, in pending or reasonably foreseeable litigation under Fed. R. Civ. P. 37(b)(2)(C).[103]

Spoliation occurs when the wrongdoer alters, changes, or substitutes dental records in an attempt to defeat a civil lawsuit.[64,322] It is far easier to defend a dental negligence lawsuit with poor records compared to attempting to justifiably defend altered (falsified) records. A jury and/or a judge will likely conclude the clinician acted with consciousness of guilt for falsifying records compared with excusing staff clerical oversight if records are poorly maintained. Dental records, like all business records, deserve accuracy and completeness.

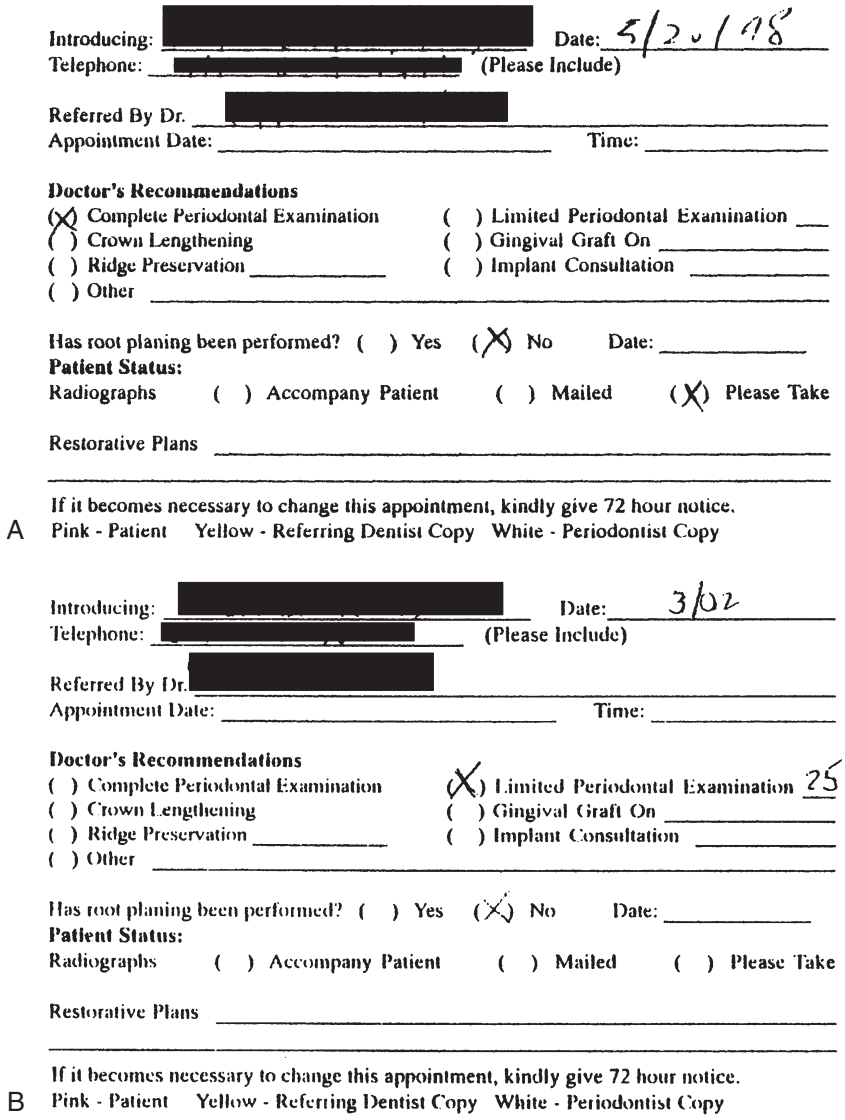
Questioned document experts utilize ink age dating, examine watermarks, or apply infrared techniques to ascertain substituted pages and additions or deletions in records, including prior erasures, entries underneath whiteouts, and indentations made when one page is overwritten on another page. Infrared technique analyzes these patterns of indentations underneath the altered pages that detect belated second entries. In one case, the defendant clinician claimed a written referral was made to a specialist in 1998, but the patient refused the referral. During litigation, the patient denied the referral and subpoenaed records of the print shop where the specialist's referral forms were printed. These subpoenaed records proved the carbon copy of the form used for the purported 1998 referral was instead first printed in 2000, which was 2 years after the alleged specialist referral was made (Fig. 29-16, *A* and *B*).

In another example, altered records were detected in nine different entries in one medical negligence case involving an undiagnosed malignancy. The physician's carrier subsequently settled for $1 million. Also, the defendant personally paid a separate fine to the court. As part of the settlement, the defendant authored an article for the Washington Medical Association detailing why records falsification is wrong.[29]

Records are subjected to (1) audits by insurance carriers for documentation that treatment was performed, (2) review by peer review committees, and (3) subpoena by state licensing boards or agencies for disciplinary proceedings. Accordingly, falsified records expose the clinician not only to civil liability for professional negligence but also to criminal penalties for criminal offenses, such as insurance fraud.[64,313,318,322,323]

Deliberate record alteration with intent to deceive may also subject the clinician to licensing[58] and ethical discipline as well as punitive damages for deceitful misconduct. Insurance carriers may defend but not indemnify a verdict for intentional material alteration of dental records done with intent to deceive.

Digital radiography may have dental advantages, but because the digital images originally may be computer manipulated, they may be legally suspect as altered.[168,313] Therefore, hard copies of the digital images may also be printed and dated in ink to show the informational baseline upon which the clinician based diagnostic or therapeutic decisions. This also protects against computer glitches, such as disk drive crashes, electrical power surges, computer viruses, and operator delete errors.



Introducing: [redacted] Date: 5/2/98
Telephone: [redacted] (Please Include)

Referred By Dr. [redacted]
Appointment Date: _____ Time: _____

Doctor's Recommendations
(X) Complete Periodontal Examination ( ) Limited Periodontal Examination ___
( ) Crown Lengthening ( ) Gingival Graft On _____
( ) Ridge Preservation _____ ( ) Implant Consultation _____
( ) Other _____

Has root planing been performed? ( ) Yes (X) No Date: _____
Patient Status:
Radiographs ( ) Accompany Patient ( ) Mailed (X) Please Take

Restorative Plans _____

If it becomes necessary to change this appointment, kindly give 72 hour notice.
Pink - Patient Yellow - Referring Dentist Copy White - Periodontist Copy

A

Introducing: [redacted] Date: 3/02
Telephone: [redacted] (Please Include)

Referred By Dr. [redacted]
Appointment Date: _____ Time: _____

Doctor's Recommendations
( ) Complete Periodontal Examination (X) Limited Periodontal Examination 25
( ) Crown Lengthening ( ) Gingival Graft On _____
( ) Ridge Preservation _____ ( ) Implant Consultation _____
( ) Other _____

Has root planing been performed? ( ) Yes (X) No Date: _____
Patient Status:
Radiographs ( ) Accompany Patient ( ) Mailed ( ) Please Take

Restorative Plans _____

If it becomes necessary to change this appointment, kindly give 72 hour notice.
Pink - Patient Yellow - Referring Dentist Copy White - Periodontist Copy

B

FIG. 29-16 A, Falsified records with belated entry of year of referral. B, Falsified records.

In one instance, a judgment of $641,541.65 was awarded for negligently designed and prepared veneers and crowns. The dentist claimed she spilled Diet Coke on her records, which entailed inspection with a questioned document expert. Also the claim that a flood of muddy water caused the dentist to discard her study models was refuted by showing that the models could have been easily washed and preserved.[324]

False Claims

Performing or billing for unnecessary endodontic therapy, such as prophylactic endodontic therapy with every crown preparation, subjects the clinician to fraud. In non–pulpal exposure crown preparation, the likely incidence of subsequent endodontic therapy is approximately 3%. Therefore, performing prophylactic endodontics on the other 97% of patients represents unnecessary and therefore fraudulent treatment. Excessive unnecessary treatment also ethically violates the Hippocratic oath of "primum non nocere" ("first, do no harm").

The Federal False Claims Act carries both civil and general penalties of treble damages, fines, and attorneys' fees for fraudulently billing government programs, such as the Civilian Health and Medical Program of the Uniformed Services (CHAMPUS) or Medicare. Fines between $5000 and $10,000 per claim form apply if the U.S. mail was used for a false-claim submission. If any portion of the claimed treatment was fraudulently misrepresented, even if a small minority, a violation results.[322] Fraudulent intent need not be conclusively proved. Reckless disregard for the accuracy of submitted data is all that is necessary to obtain a federal criminal conviction or prove civil wrongdoing.[314]

LEGAL RESPONSIBILITIES

Malpractice Claims

More than 42% of physicians have been sued over the course of their careers. Before reaching the age of 40, more than half of obstetricians/gynecologists have already been sued. About 90% of general surgeons age 55 and over have been sued.[22]

Incidence of Negligence

Medical errors or adverse events are estimated to occur in almost one out of four patient visits to a family practitioner.[270]

A U.S. government study of the Institute of Medicine found that 152,000 avoidable deaths occur annually because of medical negligence.[118,120] In this government study, injury from medical care occurred in 3.7% of hospital admissions. Some 58% of these injuries were concluded to be preventable, and 13.6% were fatal injuries. The actual fatality numbers may be underestimated, because calculations were based solely on hospital chart review.

Malpractice Prophylaxis: Importance of Records

Good clinicians keep good records. Records represent the single most critical evidence a clinician can present in court as confirmation that an accurate diagnosis, proper planned treatment, and informed consent were provided.

Prevention is the goal of modern dental care. Competent endodontic treatment performed within the requisite standard of care not only saves endodontically treated teeth but also helps prevent a lawsuit for professional negligence. Thus, sound and carefully applied endodontic principles protect both patient and clinician. Prudent practices reduce avoidable and unreasonable risks associated with imprudent or careless endodontic diagnosis or therapy.

Not all examples of negligent endodontic treatment are included in this chapter, because the myriad of malpractice incidents far exceeds its scope and length. Rather, only select examples are elucidated for educational purposes.

Standard of Care

Good endodontic practice, as defined by the courts, is the standard of reasonable care that reasonably careful clinicians legally are required to comply. The standard of care does not require perfection. Instead, the legal standard is the reasonable degree of skill, knowledge, and care ordinarily possessed and exercised by reasonably careful clinicians under similar circumstances.[172]

Although the standard of care is flexible to accommodate individual variations in treatment, it is objectively tested based on what a reasonable clinician would do. Reasonable conduct represents a minimum standard required for legal due care. Reasonable care is care based on reasoning supported by sound science such as evidence-based peer-reviewed literature. Additional precautionary steps that rise above this minimal floor of reasonableness and ascend to achieve ideal care are laudable but not legally mandated. Again, the standard of care does not require perfection; nor does it require ideal endodontics. Nevertheless, prudent clinicians should always strive to achieve a higher level of care than barely or minimally adequate care. Excellent clinicians always strive to do their best and practice endodontics at the highest level. Some have criticized this concept by contending that average care is below average, because 49% of clinicians represent a minority compared to the majority of clinicians. Careful clinicians should strive to avoid the mediocrity of minimally acceptable care and instead zealously pursue a goal of endodontic excellence. Endodontists set the standard of care in endodontics. Accordingly, a general dentist and endodontist must each meet the same standard of care.

The written policies and procedures of a clinician, institution, or organization may be used as evidence of standard of care. An expert may consider these the appropriate standard of care in testifying about alleged breaches of standard of care on that issue. For instance, an expert may rely upon AAE's 1998 position paper on paraformaldehyde-containing root canal filling materials as substandard practice, as well as the fact that all U.S. dental schools teach that toxic paraformaldehyde substances should not be used for adult patients as a root canal obturant.

Negligent errors are unintended acts, either of omission or commission. Some errors do not lead to patient injury if intercepted. Failure to mitigate factors that contribute to errors or near misses may combine with human fallibilities of mood, inattention, carelessness, and forgetfulness to cause error repetition or a future more serious injury.

In deep-sea oil exploration (e.g., Deepwater Horizon) and nuclear power (e.g., the Fukushima Daiichi Power Plant) obtained oil and nuclear power presumed safely but paid a great human and environmental cost because of inadequate safety precautions. (The difference in dental care is that dental disasters are usually episodes of patient suffering or a single death rather than a single episode with many injuries and multiple deaths.)

We all make mistakes. The biggest mistake we make is not to learn from these errors. Our responsibility is to recognize our error, discuss the adverse event with the injured patient and colleagues, and follow up with prevention strategies.[187]

An example of error prevention through shared learning from the mistakes of others is Radiology Events and Discrepancies (READ). READ is structured as a confidential, educational resource to encourage learning from events and discrepancies with the goal of promoting improved patient safety.[86]

Useful lessons are thus learned from incidents/discrepancies that result in adverse consequences. As clinicians, we learn from the mistakes of others. By encouraging a reflective approach to adverse events, radiologists are encouraged to become keenly critical self-observers. For example, a "miss" is noted when an abnormality is radiographically visible but not detected at the time of reporting.

The READ submission form is accessible online via the READ website. Radiologists are encouraged to submit reports and safety-related events that have occurred in their practice. Submitted reports must be anonymized to assure the reporter's identity remains confidential. The READ panel reviews all reports and learning points identified. To ensure patient confidentiality, the event is "disidentified" by changing age and sex along with other alterations to preserve confidentiality. After publication, all material relating to the case is destroyed. A story is thus created based on a real event, which is published for shared learning. A "lessons learned" conclusion provides instructions to prevent future risks.

Standards of Care: Generalist versus Endodontist

A general practitioner performing treatment ordinarily performed exclusively by specialists—such as apical endodontic surgery, periodontal surgical grafting, or full bony impaction surgery—will be held to the specialist's standard of care.[131,132] To avoid performing treatment that is below the specialist's standard, a generalist should refer to a specialist rather than perform procedures that are beyond the general practitioner's training or competency. The three levels of clinical skill are (1) competency or beginning level, (2) proficiency, and (3) mastery.

To err is human. Even specialists may not always know what they believe they know.[98,179] Approximately 80% of general practitioners in the United States provide some endodontic therapy. Endodontic expansion into the realm of the generalist can be linked to (1) refinements in root canal preparation and improved obturation (i.e., packing) techniques currently taught in dental schools, (2) continuing education courses, and (3) significant improvements in the armamentarium of instruments, equipment, and materials available to all clinicians to optimize efficiencies in endodontics.

The AAE's Guidelines Case Difficulty Assessment Form and Guidelines provide a road map for when a generalist should treat or refer to an endodontist. Levels of difficulty range from minimal to moderate and high difficulty (Fig. 29-17). General dentists are competent to treat cases of minimal difficulty. Experienced general dentists may treat the moderate-difficulty cases but should refer high-difficulty patients.

Ethical Guidelines

The ADA's Code of Ethics guides conduct that distinguishes the dental profession from a trade by placing patient service paramount and profit secondary.[122] Section 5 of the ADA Code of Ethics regarding veracity ("truthfulness") states, "Professionals have a duty to be honest and trustworthy in their dealings with people." Accordingly, both ethically and legally,[159] clinicians are obligated to communicate truthfully and without deception to patients. Announcements to the public must be truthful so that the average person is not materially misled. Truth in dentistry is the rule and not the exception.[122]

Section 5.I of the ADA Code of Ethics provides the following:

> General Practitioner Announcement … general clinicians who wish to announce the services available in their practices are permitted to announce the availability of those services so long as they avoid any communications that express or imply specialization.

Thus, clinicians should not misrepresent or overstate the facts of their training and competence in any way that would be false or misleading in any material or significant manner to the public or their peers. General clinicians should avoid false and misleading self-representation regarding endodontic "superiority." Implying credentials that the generalist lacks misrepresents a general clinician's endodontic skill and knowledge as equivalent to a board-eligible or board-certified endodontist. A generalist's 30-year experience may be little more than 1 year's experience repeated 30 times. Every state has specific laws governing advertising. Consult your state's law, because each state deals differently with specialty announcements.

Telephone directory listings are organized into categories based on types of procedure, such as endodontics. Generalists must also state in such ads that endodontic services are being provided by general clinicians. The public is entitled to be informed of the distinction between an endodontist specialist and a generalist, although each provides endodontics. Only an ADA-recognized board-eligible or board-certified clinician is entitled to represent him or herself as an endodontist. With this distinction in mind, the ADA-recognized endodontic specialty organization is titled the American Association of Endodontists, not the American Association of Endodontics.

Standard of Care for Endodontics

Endodontists set the standard for routine endodontics. Therefore, if the endodontist's standard of care cannot be met, such as the need for microscopy or apical surgery, the generalist should refer the patient to an endodontist.[76,131,132,183]

Endodontists should not forget their general dentist training. Even though a patient may be referred for a specific procedure or undertaking, the endodontist should not overlook sound biologic principles inherent in the overall treatment. A specialist may also be held liable for relying solely on the information referral card or radiographs of the referring clinician if the diagnosis or therapeutic recommendations prove incorrect, unnecessary, or the referral card lists the wrong tooth for treatment. Therefore, conduct a "time-out" with patient and staff to verify which tooth is to be treated.[167]

Figure 29-18 demonstrates a general clinician's radiograph showing apparent complete endodontic fill within the canal space, whereas Figure 29-19 demonstrates an endodontist's radiograph showing a transported canal. This difference is explained by radiographic quality and angulation. Taking radiographs at different angles improves radiographic accuracy.[201,204]

Endodontists should not provide rubber-stamp treatment to whatever the clinician refers or recommends. Without performing an independent examination, the endodontist risks misdiagnosis and resulting incorrect treatment. Prevention of misdiagnosis or incorrect treatment requires an accurate medical and dental history preceding a clinical examination (not only of the specific tooth or teeth involved but also of the general oral condition). Obvious problems such as oral lesions, periodontitis, or gross decay should be noted in the chart and the patient advised regarding a referral for further examination, testing, or another specialist's consultation.

Radiographs from the referring clinician should be reviewed for completeness, clarity, and diagnostic accuracy. An endodontist should expose a new radiograph to verify current status before treatment and only use the referring clinician's radiograph for historical comparison. Unfortunately, the referring clinician may surreptitiously forward a pretreatment film with the referred patient, rather than the referring clinician's own posttreatment film depicting a perforation or broken file that necessitated the referral. This may occur whenever an errant clinician attempts to fraudulently conceal negligently performed endodontic treatment in an attempt to shift blame for bungled treatment to the endodontist. In such cases, a valuable lesson is learned when the endodontist exposes independent pretreatment radiographs, rather than relying exclusively on the referring clinician's radiographs to determine current status of endodontic treatment.

Poor oral hygiene may contribute to periodontal disease. In such cases, endodontic treatment may be compromised unless the associated periodontal condition, along with the tooth being treated endodontically, is brought under periodontal disease control, including any crown-lengthening needs (see Chapter 25 for more information). Referral to a periodontist may be necessary before or contemporaneous with completion of endodontic treatment.

In summary, it is necessary for the endodontist to do the following:

1. Be alert to any contributory medical or dental condition within the operative area of endodontic treatment that can affect treatment.

AAE Endodontic Case Difficulty Assessment Form and Guidelines

PATIENT INFORMATION

Name_____

Address_____

City/State/Zip_____

Phone_____

DISPOSITION

Treat in Office: Yes ☐ No ☐

Refer Patient to:

_____

Date:_____

Guidelines for Using the AAE Endodontic Case Difficulty Assessment Form

The AAE designed the Endodontic Case Difficulty Assessment Form for use in endodontic curricula. The Assessment Form makes case selection more efficient, more consistent and easier to document. Dentists may also choose to use the Assessment Form to help with referral decision making and record keeping.

Conditions listed in this form should be considered potential risk factors that may complicate treatment and adversely affect the outcome. Levels of difficulty are sets of conditions that may not be controllable by the dentist. Risk factors can influence the ability to provide care at a consistently predictable level and impact the appropriate provision of care and quality assurance.

The Assessment Form enables a practitioner to assign a level of difficulty to a particular case.

LEVELS OF DIFFICULTY

MINIMAL DIFFICULTY	Preoperative condition indicates routine complexity (uncomplicated). These types of cases would exhibit only those factors listed in the MINIMAL DIFFICULTY category. Achieving a predictable treatment outcome should be attainable by a competent practitioner with limited experience.
MODERATE DIFFICULTY	Preoperative condition is complicated, exhibiting one or more patient or treatment factors listed in the MODERATE DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for a competent, experienced practitioner.
HIGH DIFFICULTY	Preoperative condition is exceptionally complicated, exhibiting several factors listed in the MODERATE DIFFICULTY category or at least one in the HIGH DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for even the most experienced practitioner with an extensive history of favorable outcomes.

Review your assessment of each case to determine the level of difficulty. If the level of difficulty exceeds your experience and comfort, you might consider referral to an endodontist.

The contribution of the Canadian Academy of Endodontics and others to the development of this form is gratefully acknowledged.

The AAE Endodontic Case Difficulty Assessment Form is designed to aid the practitioner in determining appropriate case disposition. The American Association of Endodontists neither expressly nor implicitly warrants any positive results associated with the use of this form. This form may be reproduced but may not be amended or altered in any way.

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FIG. 29-17 American Association of Endodontists (AAE) Endodontic Case Difficulty Assessment Form and Guidelines.

AAE Endodontic Case Difficulty Assessment Form

Criteria and Subcriteria	Minimal Difficulty	Moderate Difficulty	High Difficulty
A. PATIENT CONSIDERATIONS			
Medical history	☐ No medical problem (ASA Class 1*)	☐ One or more medical problems (ASA Class 2*)	☐ Complex medical history/serious illness/disability (ASA Classes 3-5*)
Anesthesia	☐ No history of anesthesia problems	☐ Vasoconstrictor intolerance	☐ Difficulty achieving anesthesia
Patient disposition	☐ Cooperative and compliant	☐ Anxious but cooperative	☐ Uncooperative
Ability to open mouth	☐ No limitation	☐ Slight limitation in opening	☐ Significant limitation in opening
Gag reflex	☐ None	☐ Gags occasionally with radiographs/treatment	☐ Extreme gag reflex which has compromised past dental care
Emergency condition	☐ Minimum pain or swelling	☐ Moderate pain or swelling	☐ Severe pain or swelling
B. DIAGNOSTIC AND TREATMENT CONSIDERATIONS			
Diagnosis	☐ Signs and symptoms consistent with recognized pulpal and periapical conditions	☐ Extensive differential diagnosis of usual signs and symptoms required	☐ Confusing and complex signs and symptoms: difficult diagnosis ☐ History of chronic oral/facial pain
Radiographic difficulties	☐ Minimal difficulty obtaining/interpreting radiographs	☐ Moderate difficulty obtaining/interpreting radiographs (*e.g.*, high floor of mouth, narrow or low palatal vault, presence of tori)	☐ Extreme difficulty obtaining/interpreting radiographs (*e.g.*, superimposed anatomical structures)
Position in the arch	☐ Anterior/premolar ☐ Slight inclination (<10°) ☐ Slight rotation (<10°)	☐ 1st molar ☐ Moderate inclination (10-30°) ☐ Moderate rotation (10-30°)	☐ 2nd or 3rd molar ☐ Extreme inclination (>30°) ☐ Extreme rotation (>30°)
Tooth isolation	☐ Routine rubber dam placement	☐ Simple pretreatment modification required for rubber dam isolation	☐ Extensive pretreatment modification required for rubber dam isolation
Crown morphology	☐ Normal original crown morphology	☐ Full coverage restoration ☐ Porcelain restoration ☐ Bridge abutment ☐ Moderate deviation from normal tooth/root form (*e.g.*, taurodontism, microdens) ☐ Teeth with extensive coronal destruction	☐ Restoration does not reflect original anatomy/alignment ☐ Significant deviation from normal tooth/root form (*e.g.*, fusion, dens in dente)
Canal and root morphology	☐ Slight or no curvature (<10°) ☐ Closed apex (<1 mm in diameter)	☐ Moderate curvature (10-30°) ☐ Crown axis differs moderately from root axis. Apical opening 1-1.5 mm in diameter	☐ Extreme curvature (>30°) or S-shaped curve ☐ Mandibular premolar or anterior with 2 roots ☐ Maxillary premolar with 3 roots ☐ Canal divides in the middle or apical third ☐ Very long tooth (>25 mm) ☐ Open apex (>1.5 mm in diameter)
Radiographic appearance of canal(s)	☐ Canal(s) visible and not reduced in size	☐ Canal(s) and chamber visible but reduced in size ☐ Pulp stones	☐ Indistinct canal path ☐ Canal(s) not visible
Resorption	☐ No resorption evident	☐ Minimal apical resorption	☐ Extensive apical resorption ☐ Internal resorption ☐ External resorption
C. ADDITIONAL CONSIDERATIONS			
Trauma history	☐ Uncomplicated crown fracture of mature or immature teeth	☐ Complicated crown fracture of mature teeth ☐ Subluxation	☐ Complicated crown fracture of immature teeth ☐ Horizontal root fracture ☐ Alveolar fracture ☐ Intrusive, extrusive or lateral luxation ☐ Avulsion
Endodontic treatment history	☐ No previous treatment	☐ Previous access without complications	☐ Previous access with complications (*e.g.*, perforation, non-negotiated canal, ledge, separated instrument) ☐ Previous surgical or nonsurgical endodontic treatment completed
Periodontal-endodontic condition	☐ None or mild periodontal disease	☐ Concurrent moderate periodontal disease	☐ Concurrent severe periodontal disease ☐ Cracked teeth with periodontal complications ☐ Combined endodontic/periodontic lesion ☐ Root amputation prior to endodontic treatment

*American Society of Anesthesiologists (ASA) Classification System

Class 1: No systemic illness. Patient healthy.
Class 2: Patient with mild degree of systemic illness, but without functional restrictions, *e.g.*, well-controlled hypertension.
Class 3: Patient with severe degree of systemic illness which limits activities, but does not immobilize the patient.
Class 4: Patient with severe systemic illness that immobilizes and is sometimes life threatening.
Class 5: Patient will not survive more than 24 hours whether or not surgical intervention takes place.

www.asahq.org/clinical/physicalstatus.htm

FIG. 29-17, cont'd

FIG. 29-18 A general clinician's radiograph that ostensibly shows a complete endodontic fill within the root canal space.

FIG. 29-19 Endodontist's radiograph demonstrating a transported canal.

2. Undertake an independent diagnostic and radiographic examination of the treatment area and treatment plan rather than relying solely on the referring clinician.
3. Perform a general dental examination (at least a screening) to diagnose any hard- and soft-tissue pathosis.
4. Evaluate status and prognosis of adjacent and opposing teeth.
5. Advise the referring clinician and patient of pertinent findings.

Ordinary Care Equals Prudent Care

Ordinary is commonly understood (outside its legal context) to mean "lacking in excellence" or "being of poor or mediocre quality."[217] As expressed in the context of actions for negligence, however, ordinary care has assumed a technical legal definition that differs somewhat from its common meaning. The eighth edition of *Black's Law Dictionary* describes ordinary care as "that degree which persons of ordinary care and prudence are accustomed to use or employ … that is, reasonable care."[46]

FIG. 29-20 Implant "harpooned" inferior alveolar nerve canal.

In adopting this distinction, the courts have defined *ordinary care* as "that degree of care which people ordinarily prudent could be reasonably expected to exercise under circumstances of a given case."[108] It has been equated with the reasonable care and prudence exercised by ordinarily prudent clinicians under similar circumstances.[136,265] It is not extraordinary or ideal care. Thus, ordinary care is not average care but instead equates with prudently careful care. Stated otherwise, average care may be below the average of what all reasonable clinicians should practice.

Although the standard required of a professional cannot be only that of the most highly skilled practitioner, neither can it be limited to the arithmetic-average member of the profession, because those who possess somewhat less than median skill may still be competent and qualified to treat.[265] By such an illogical definition of average care, half of all clinicians would automatically fall short of the mark and be negligent as a matter of law. As one court explained, "We are not permitted to aggregate into a common class the quacks, the young men who have not practiced, the old ones who have dropped out of practice, the good, and the very best, and then strike an average between them."[281] Thus, the standard of care equates with reasonable care and not average care.

Customary Practice versus Negligence

Customary practice may constitute evidence of the standard of care, but it is not the only determinant. Moreover, if the customary practice constitutes negligence, it is not considered reasonable (although or even if customarily practiced by a majority of clinicians).[38] Rather, the reasonably careful clinician is the standard of care and not an average or mediocre practitioner. For instance, a majority of clinicians did not perform biologic testing of the dental unit water lines despite ADA recommendations to do so.[220,222] However, careful clinicians did follow the ADA's recommendation (Box 29-2 and Fig. 29-20).

Merely because a majority of clinicians in a community practice a particular method does not establish it as the standard of care if the practice is unreasonable or imprudent.[38] Ultimately, the courts determine what constitutes reasonable practice by considering available dental knowledge and

BOX 29-2

Typical Examples of Negligent Customs

- Failing to probe and record periodontal pockets
- Failing to take diagnostic-quality radiographs
- Failing to refer patients for complicated procedures
- Failing to use aseptic practices such as gloves, facemasks, and sterile fluids for surgery
- Failing to use rubber dams for root canal therapy
- Failing to install or periodically check valves in dental units to prevent water retraction, suck-back, and cross-contamination
- Failing to do thermal pulp testing as an aid to diagnosing pulpal disease (see Chapter 1)
- Failing to discontinue quaternary ammonium chloride–based products for precleaning or disinfection of environmental surfaces
- Failing to use a microscope for fractured file retrieval and periradicular surgery
- Failure to include CAMBRA in a comprehensive new patient exam
- Failure to use CBCT technology to guide implant placement when placement is in close proximity to the IANC (see Fig. 29-21 for implant "harpooning" of IANC)
- Not informing CT radiation risks to patient[171]

CAMBRA, caries management by risk assessment; *CBCT,* cone-beam computed tomography; *IANC,* inferior alveolar nerve canal.

Data from American Academy of Orthopaedic Surgeons: Advisory statement, antibiotic prophylaxis for dental patients with total joint replacements, *J Am Dent Assoc* 134:895, 2003.
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evaluating the relative risks versus benefits of a particular procedure.

Again, the law does not require dental perfection.[311] Instead, the legal yardstick by which prudent conduct is measured is what a reasonably prudent clinician should do under the same or similar circumstances, regardless of how many or how few clinicians conform to this standard.

In one case, it was not customary practice in the state of Washington for ophthalmologists to test patients under 40 years old for glaucoma, because the incidence was only 1 in 25,000 patients. Nevertheless, the Supreme Court of Washington state held that the defendant ophthalmologist was negligent as a matter of law, irrespective of customary medical practice, as a negligent customary practice is no defense.[150,213]

Little excuse exists for failing to routinely probe and chart periodontal pockets before rendering endodontic therapy, no matter how many other clinicians in the community may fail to do so. The benefit of probing for periodontal disease substantially outweighs the virtually nonexistent risk of conducting this valuable diagnostic procedure. A legal defense likely to invoke a jury's wrath is to claim that a necessary diagnostic or prophylactic procedure is "too time consuming," when dental and medical patient health are placed at risk if not done. If the benefit outweighs the risk, a reasonable clinician complying with the standard of care should opt for the beneficial diagnostic test or treatment. For instance, the Accuitomo and Kodak 9000 CBCT may prove an invaluable benefit for diagnosing the etiology and source of endodontic pain when compared to the relatively low radiation risk. Thus, the CBCT diagnostic benefit outweighs the radiation risk if a differential diagnosis cannot accurately rule in or rule out a final diagnosis.[203]

Another example of negligent customary practice includes a majority of general dentists not using a rubber dam for all root canal therapy despite virtually all endodontists who do so. The AAE's 2010 Position Statement mandates that "tooth isolation using the dental dam is the standard of care; it is integral and essential for any nonsurgical endodontic treatment."

A further instance of a customary but nevertheless negligent practice concerns the majority of dentists who did not adhere to CDC guidelines for infection control.

Health Maintenance Organization Care versus Standard of Care

Reasonably careful or prudent practitioners (not insurance carriers) set the standard of care. Third-party payers may limit reimbursement but should not limit access to quality care. Clinicians have an affirmative duty on behalf of patients to appeal insurance carrier care denial decisions and, in some states, are protected against retaliation.[332] The clinician who complies (without protest) with restrictive limitations or denials imposed by a third-party payer when sound judgment dictates otherwise cannot avoid ultimate responsibility for the patient's care.[332]

Although dental insurance companies do not set the standard of care, insurance carriers may contractually limit dental benefits. Therefore, a clinician is obligated to inform patients of their dental needs, regardless of carrier reimbursement. Patients may then elect to pay out of pocket for noncovered treatment or decline uninsured services. Informed choice is uninformed if the clinician fails to provide patients with all reasonable options and alternatives.[209] Follow the adage to not x-ray the patient's pocket book or wallet to determine what options the patient should be presented. Instead offer the patient all reasonable treatment options.

Notwithstanding that an insurance carrier denies endodontic therapy or limits endodontics to only certain clinical conditions, a prudent clinician must nevertheless, both legally and ethically, provide an informed consent option to the patient that a tooth may be endodontically treated and retained rather than extracted.[71,220,288,303,346] The California Dental Association's Dental Patient Bill of Rights advises patients that "You have the right to ask your clinician to explain all treatment options regardless of coverage or cost."[60]

Clinicians may agree to a discounted fee with a health maintenance organization (HMO) carrier but must never discount the quality of care provided. Peer reviews and the courts

recognize only one standard of care. A lower, double, or different standard of care for reduced-fee HMO plans is not legally recognized. Expediency should not be exercised at the expense of quality patient care. Section 3 of the ADA Code of Ethics affirms, "Managed care contract obligations do not excuse clinicians from their ethical duty to put the patient's welfare first."

An Illinois appellate court held that a clinician may be sued for injuries a patient experiences as a result of the clinician's failure to disclose a contractual arrangement with the patient's HMO that creates financial incentives to minimize diagnostic tests and referrals to specialists. The court recognized a distinct cause of action for breach of fiduciary duty for failure to disclose these types of financial incentives.[232] In reaching its conclusion, the court cited an American Medical Association (AMA) ethics opinion that stated that a clinician must ensure that a patient is aware of financial incentives through which health insurers limit diagnostic tests and treatment options.

Managed care organizations use a variety of strategies to influence the practice styles of providers. One of the most controversial of these methods is the use of financial incentives designed to limit referrals to specialists. Such financial incentives usually take the form of bonus payments drawn from surpluses in risk pools funded by "withholds." These funds are deducted from the primary care provider's base payments or otherwise reserved under contracts in which the care provider bears financial risk. Such bonuses are based on referral limitations.[140] Conversely, the federal Patient Protection and Affordable Care Act compensates based on the quality of care rather than the quantity.[244]

Plans that delay treatment approval, resulting in endodontic complications or tooth nontreatability, may be subject to liability.[233,303] HMO carriers have not always succeeded in arguing that the 1974 Employee Retirement Income Security Act (ERISA) preempts state law for dental negligence claims against entities who administrate health care benefits to an ERISA plan and shift blame to the clinician.[248]

Capitation systems have a built-in incentive to undertreat, delay, or discourage treatment and access to care. For the HMO-paid clinician, the patient may be perceived as a threat to profits. Thus, capitation creates incentives that can transform clinicians from the patient's advocate to the patient's adversary.

Although clinicians seem like double agents serving two masters (i.e., managed care carriers and patients), the law is clear that the clinician must always act in the patient's best interest, because the clinician owes a fiduciary obligation to the patient. Should the carrier deny requested care, the clinician is legally obligated to appeal the decision to protect the patient's dental health.[332] A patient's best interest is always paramount, both legally and ethically, to a clinician's financial interest.[122,333]

Although profitable to insurance carriers, the success of denying or limiting benefits has created an era of rightfully indignant patients and frustrated clinicians. Consequently, the clinician must guard against patient disappointment with limited insurance benefits by explaining that although insurance carriers determine coverage under their patients' insurance policy, carriers do not set the standard of care. Regardless of how much or how little is covered by the patient's insurance carrier, prudent and reasonably careful clinicians set the standard of care. Providing less than necessary care because of insurance carriers' dictates may explain resulting deficient endodontics but does not excuse such conduct. Moreover, the legal doctrine of informed consent mandates the patient be informed of alternative therapy choices that the patient may pay privately even though the HMO denies approval for alternative therapies. Follow the maxim: Do not x-ray a patient's pocketbook. Reasonable informed consent is premised on patient choice and not cost alone.

Employment Law

An employer may fire an employee for a bad reason or no reason at all, as long as termination was not done for discriminatory reasons.[236,326]

Dental Negligence Defined

Dental negligence is defined as a violation of the standard of care (i.e., an act or omission that a reasonably careful or prudent clinician under similar circumstances would not have done).[172] *Negligence* is equated with carelessness or inattentiveness.[179] *Malpractice* is a lay term for such professional negligence. Dental negligence occurs for two reasons:

1. A clinician does not possess a reasonable degree of education and training to act prudently.
2. Despite reasonable schooling, additional training, and continuing education, the clinician acts with unreasonable carelessness or imprudently fails to act as a reasonably careful clinician would act.

One simple test to determine if an adverse outcome results from negligence is to ask the following question: Was the treatment result reasonably avoidable? If the answer is yes, it is probably malpractice. If the answer is no, it is instead an unfortunate maloccurrence that resulted despite reasonable care attempts.

Locality Rule

The locality rule, which provides for a different standard of care in different communities, is rapidly becoming outdated. Originating in the 19th century, the rule was designed to acknowledge differences in facilities, training, and equipment between rural and urban communities.[198,347]

The trend across the United States is to move from a locally based standard to a statewide standard, at least for generalists. For endodontists, a national standard of care is applied, as the AAE Board is national in scope.[290] Because of nationally published endodontic literature, advances in Internet communication, continuing education courses, and reasonably available transportation for patients, no disparity generally exists between rural and urban endodontic standards. A current exception may be the limited geographic availability of specific cone-beam CT technology such as the Accuitomo or Kodak 9000. Their higher resolution provides superior radiographic endodontic diagnosis compared with 2D imaging and even other CBCT devices. As CBCT machine pricing is lowered and availability increases, the locality rule may be broadened beyond a local community for CBCT technology. On the other hand, time is critical during the first 48 hours of symptomatic numbness if an endodontic overfill enters the IANC.[254] In such instances, a referral to a distant CBCT facility may be required to assess the need for immediate microsurgical removal or instead obtain, at the very least, a medical CT in the local geographic area if a CBCT facility is unavailable.*

*References 75, 97, 157, 169, 201, 204.

A clinician should provide reasonably careful endodontic care to a patient regardless of treatment locality. Rather than focusing on different standards for different communities, more important considerations include knowledge of endodontic advances in the field gained with continuing education to utilize improved diagnostics, instrumentation, and therapeutic interventions.

The locality rule has two major drawbacks. In areas with small populations, clinicians may be reluctant to testify as expert witnesses against other local clinicians. Also, the locality rule allows a small group of clinicians in an area to establish a local standard of care inferior to what the law requires of larger urban areas. Peer-reviewed publications are available to all clinicians in print and online. Clinicians can easily travel great distances to attend continuing education courses or attend webcasts in their own office. Blaming technologic ignorance on a clinician's rural location is inexcusable with modern media, computer technology, continuing education courses, and travel ease.

Continuing Education

Continuing education should not add new skills at the expense of compromising or eliminating core clinical values. A prudent practitioner should be cautious adding a new methodology that is counterintuitive to basic biomechanical principles.[224] As a general maxim, don't be the first or the last to adopt new technology.

A clinician is legally obligated to maintain current knowledge in the field of endodontics. If not, the clinician may have only 1 year of knowledge (repeated 30 times) during the span of a 30-year career. By not maintaining continuing education knowledge and updating clinical skills, a clinician may unreasonably condemn otherwise salvageable teeth because of inadequate diagnosis or treatment.

Scientific Research Evaluation

In evaluating scientific research, the clinician should remember that epidemiologic data of risk factors are not always equivalent to etiology. Thus, *epidemiology* is not a synonym for *etiology*. The scientific community has never exclusively relied on epidemiology as the accepted method of evaluating cause-and-effect relationships in an effort to make clinical decisions. Accordingly, clinicians should not consider only one class of data to supply ultimate proof when conducting an evaluation. Instead, clinicians should consider the strength of any study to be related to the presence and contributing cause of other cofactors.[137,239,240,253] The unusual characteristics of a particular patient can place the patient at higher risk than the average patient.[153]

New products and procedures are often introduced faster than epidemiologic and toxicologic studies are implemented to evaluate their potential risks. Academic medical/dental centers are facing severe pressure on their reimbursement for health care services and postgraduate education. This creates the need to examine additional sources of revenue to fund their tripartite mission of education, research, and patient-care delivery. Reduced government spending for research has resulted in proprietary interests (with proprietary goals) funding more research.[147] Since 1984, the *New England Journal of Medicine*'s policy has been to refuse publication of research done by those with financial ties to drug makers. But in 1999, the journal admitted conflicts in nearly half of the drug studies published since 1997.[26] The next year, the retiring editor of the *New England Journal of Medicine* concluded that despite tax-supported privileges and extraordinary pharmaceutical industry profits, the best interests of society were not always served. A public trust more accountable to science was needed.[27]

Evidence-Based Endodontics

Although much emphasis is currently placed on evidence-based medicine and dentistry, most clinical practices are not based on data derived from randomized clinical trials.[240] It is virtually impossible to conduct a trial to test the validity of every possible patient-management option. Many therapeutic choices are so compelling that a test would be unethical; others are so trivial that a test might not be worth the time or effort. Clinicians do not always practice in conformity to evidence-based research, even when evidence is available from randomized clinical trials. This is because knowing the right answer is only the first step in the process of adopting a new treatment methodology. Many clinicians will not use a new drug simply because supporting research data suggest its efficacy. Instead, identification of the probable mechanism of action is often a prerequisite before adopting a new treatment. Moreover, the test of time may eventually reveal an adverse effect of a particular drug or device (e.g., defects that were not identified before marketing). Even if the new product's research proves beneficial, comparative effectiveness studies with other time-proven and tested products are needed to determine a new product's relative worthiness.[137,196,253,295]

Clinicians should not avoid using certain drugs if the data supporting their use are overwhelming. When the results of a clinical trial conflict with a widely held mechanistic model, many clinicians doubt research-based evidence.[226] However, when the results from well-researched clinical trials become so convincing that the evidence can no longer be ignored, a prudent clinician embraces the new paradigm and discards previously held concepts. Endodontic science advances as disproved older concepts retreat. For example, delaying extractions until after several days of antibiotic use to reduce infection is no longer the accepted practice. Rather, immediate extraction, along with any necessary antibiotics, is the preferred current therapy.[6] Another example is one-visit endodontics, which has been proved to be as effective as two-visit endodontics.[109,225,250]

New Products

New devices that lack definitive research studies should be used cautiously. For instance, high-intensity, wireless, fast-curing lights may generate heat at the wand tip and cause pulpal pathosis. First-generation halogen bulbs require longer composite curing time to achieve polymerization but generate only 400 to 800 mW/cm. Plasma arc bulbs reduce curing time but generate 2000 mW/cm.

Hydron was marketed without long-term clinical testing. Its purported biocompatibility and reduced inflammation portended an improved endodontic drug for obturation. Post-marketing failures resulted from inadequate premarket testing that lacked long-term research. Thus, Hydron's inability to obturate canals with a durable filling material resulted in both clinicians and patients being harmed as postmarket guinea pigs. Hydron's in vitro research did not match in vivo patients' experiences.

Other examples include Endocal 10 (formerly Biocalex 6/9), which proved ineffective. Arsenic to promote devitalization of the pulp resulted in local osteonecrosis. Thermafil metal carriers proved difficult to remove and were replaced with plastic carriers. Advance cement was touted as an improved luting cement for retaining permanent restorations. The high incidence of marginal leakage with Advance resulted in its withdrawal from the market in 2000. A multimillion-dollar class action settlement resulted against the manufacturer on behalf of California clinicians with unsealed margins of crowns cemented with the Advance luting agent.[128]

Older devices, over time and with sufficient patient experience, may manifest adverse events. Overheating with ultrasonic tips for post removal has resulted in teeth and tissue loss (Fig. 29-21). Accordingly, despite insufficient label warnings on some ultrasonic devices, copious water coolant, rest periods, and avoidance of prolonged ultrasonic device use are needed for safe post removal.[90,95,130]

Today's clinician exploring ways to improve the quality and success of endodontic therapy is constantly presented with new dental products and techniques. For prescribing and using drugs or other agents, the ADA's Principles of Ethics, Section 5D, provide this guideline[18]:

> Except for formal investigative studies, dentists shall be obliged to prescribe, dispense, or promote only those devices, drugs and other agents whose complete formulae are available to the dental profession. Dentists shall have the further obligation of not holding out as exclusive any device, agent, method or technique if that representation would be false or misleading in any material respect.

Ethical clinicians should not indiscriminately adopt every new product. Instead, the supporting research should be reviewed, rather than risk the patient's welfare with inadequately tested products or researched products only tested in vitro. Again, don't be the first or the last to adopt new technology.

FDA Approval

Despite FDA approval, some drugs are marketed with fraudulent concealment of the number and nature of adverse event incidents.[10]

FDA Drug Approval

Until 1962, drug companies were allowed to promote their products for any use so long as they were shown to be "safe" for one use. Manufacturers were marketing drugs with serious side effects for minor conditions and to vulnerable populations, resulting in many injuries and deaths. Ineffective drugs were the rule rather than the exception. When a retrospective review of all drugs was conducted after 1962, the National Academy of Sciences found that fully 80% of the uses for which drugs were being promoted could not be shown to be effective. Since 1962, the FDA has required prescription drug manufacturers to demonstrate safety and efficacy with animal and randomly controlled research trials conducted with FDA-approved protocols. FDA "approval" to market new drugs for specific uses is termed *premarket approval* (PMA).

In a new drug application (NDA) to the FDA, findings from many clinical studies are done to assess a new drug's efficacy and safety and to gain the FDA's PMA to market the drug. An NDA judges whether the proposed new drug (1) causes more good than harm (benefit outweighs the risk), (2) is comparatively more effective than already existing marketed drugs, or (3) has unreasonably frequent or severe side effects (risk outweighs the benefit).[35,321]

In 2008, $58 billion in privately funded drug research was approximately twice the basic federal medical research budget and encompassed an estimated 50,000 clinical trials among 2.3 million patients. Medicare drug spending is forecast to grow from $2 billion in 2000 to $153 billion in 2016, a 7550% increase.[247] In 2007, one in seven U.S. residents younger than age 65 skipped prescribed medications because of rising prescription cost.[106,152]

The Pharmaceutical Research and Manufacturers Association of America has created an online database that summarizes

FIG. 29-21 Results of overheating a post with an ultrasonic device during post removal.

clinical study results involving hundreds of prescription medicines. Notwithstanding, the sheer volume of clinical testing has overwhelmed the FDA's ability to independently assess commercial trials and make all test results public. Deceptive marketing, unreported side effects, and hidden payments to medical/dental researchers highlight the gap between the number of clinical trials conducted and the number published.*

Comprehensive clinical data filed with the FDA for gaining market approval of a new drug (PMA) form the basis for safety information that accompanies every prescription label.[113,114] For the first time, under a new federal law[115] effective in 2009, researchers in PMA trials will have to post their basic research results publicly on a federal online registry maintained by the National Library of Medicine. However, new registry regulation will only cover new tests, not tests with poor results done prior to the effective date of the new law. Moreover, findings are not independently FDA verified. Instead, the FDA relies on the manufacturer's submitted trial data research.

Until September 2007, researchers only had to report the start of a clinical trial and were under no federal legal obligation to report the outcome in the FDA's registry or in a peer-reviewed journal. Consequently, clinical data on health risks could be downplayed or benefits extolled selectively in any one of 5200 peer-reviewed biomedical journals.[316,338]

By January 31, 2009, as a voluntary patient guide, the federal registry (www.clinicaltrials.gov) logged 67,000 studies. The results of all drug trials that were ongoing on or after September 27, 2007, must be reported at ClinicalTrials.gov if the products under study have FDA approval. The results for drugs not yet receiving FDA approval do not have to be posted. A deficiency of the current law is that the results of older trials of drugs that were FDA approved before September 27, 2007, and that were no longer the subject of ongoing trials on or after September 27, 2007, do not need to be posted. Such drugs constitute the majority of current prescription drugs. The FDA considers much of the data on clinical trials it receives from sponsors—and its own analyses of these data—to be "confidential commercial information." FDA drug-trial information released to the public is frequently heavily redacted. Redacted disclosure occurs in response to one or multiple requests made under the Freedom of Information Act, but usually only after a prolonged delay. The FDA Amendments Act of 2003, Section 916, institutionalizes this limitation by mandating that the FDA publish on its website key information related to approval of an application for a drug that contains a previously approved active ingredient only after it has received three Freedom of Information Act requests.

In a 2008 University of California, San Francisco (UCSF), study of 164 clinical trials testing 33 different drugs submitted for FDA approval, approximately one in four had yet to be published.[269] Virtually all of the unpublished findings were negative. The UCSF study also reported that in 900 FDA filings involving 90 new drugs, more than half the clinical trials were still unpublished 5 years after the drug had FDA approval. UCSF researchers who reviewed the FDA's regulatory paperwork for dozens of recently FDA-approved (PMA) drugs found that in some clinical trials previously reported to the FDA, when subsequently submitted for publication, conclusions were changed, statistics revised, and outcomes altered to make treatments appear more effective. Among 43 outcomes reported in the FDA filings that did not favor a drug, 20 were never published. In four out of five instances in which the statistical significance of findings were changed from the FDA filing, the published version was more favorable. Positive effects of a new drug were more likely to be submitted for publication than those reporting negative findings. Basic principles of evidence-based practice require the analysis of all data. Publishing only some results but not others undermines the ability to adequately assess a product's safety and efficacy, which is FDA's standard for FDA approval of drugs or devices.

Journals want to sell journals. Companies wish to increase profits. Researchers promote career advancements and seek research funding. Consequently, potential conflicts are pervasive. To offset these temptations, failure to timely report basic data about clinical test results in the federal public registry can subject researchers to fines of $10,000 a day and/or loss of their federal research funding (Public Health Service Act Section 302[J]).[114,338]

In 2009, the Government Accounting Office (GAO) concluded in the GAO's 2009 High-Risk Series, *FDA Oversight of Medical Products*[65]:

> New Laws, the complexity of items submitted to FDA for approval, and the globalization of the medical products industry are challenging FDA's ability to guarantee the safety and effectiveness of drugs, biologic, and medical devices. As a result, the American consumer may not be adequately protected from unsafe and ineffective medical products. FDA needs to improve the data it uses to manage the foreign drug inspection program, do more inspections of foreign establishments that manufacture drugs or medical devices, more systemically review the claims made in drug advertising and promotional material, and ensure that drug sponsors accurately report clinical trial results.

Globalization of clinical research is a relatively recent phenomenon. The number of countries serving as trial sites outside the United States more than doubled in 10 years. Conversely, the proportion of trials conducted in the United States and Western Europe decreased.[127] Clinical testing in developing countries sidesteps stringent FDA regulatory barriers for FDA drug approval.[28]

Wide disparities in education, economic and social standing, and health care systems may jeopardize the rights of research participants in foreign countries. Foreign participants may lack understanding of the investigational nature of therapeutic products or the use of placebo research participant groups. In some places, financial compensation for research participation may exceed participants' annual wages. Standards of health care in developing countries may also allow ethically problematic study designs or clinical research trials that would not be allowed in wealthier countries. In one study, only 56% of the 670 researchers surveyed in developing countries reported that their research had been reviewed by a local institutional review board or health ministry.[158] In another study, 90% of published clinical trials conducted in China in 2004 did not report ethical review of the protocol, and only 18% adequately discussed informed consent.[345]

Developing countries will also not realize the benefits of clinical research trials if the drugs being evaluated do not become readily available for host-country research participants after FDA approval is obtained.[28] The Declaration of Helsinki

*References 27, 181, 196, 216, 256, 269, 274, 284, 295, 316, 337.

includes an expectation that every patient enrolled in a clinical trial should, at the end of the trial, be assured access to the best proven therapy identified in the research study.

Geographically distinct populations can have different genetic profiles; these differences have been shown to be related to the safety and effectiveness of drugs and medical devices. For example, a study of 42 genetic variants associated with pharmacologic response in drug studies showed that more than two thirds had significant differences in frequency between persons of African ancestry and those of European ancestry.[133] Genetic diversity is often not considered in study design and interpretation and in the reporting of trial results.

From fiscal 2002 through 2007, the FDA issued 15 warning letters to foreign companies with serious deficiencies, but the agency only reinspected four of the companies, and then only 2 to 5 years later. Decades ago, most pills consumed in the United States were made here. But like other manufacturing operations, drug plants have been moving to Asia where labor, construction, regulatory, and environmental costs are lower. The world's growing dependency on Chinese drug manufacturers became apparent in the heparin scare. In 2007, Baxter International and APP Pharmaceuticals split the domestic market for heparin, an anticlotting drug needed for surgery and dialysis. When federal drug regulators discovered that Baxter's product had been contaminated by Chinese suppliers, the FDA banned Baxter's product and turned almost exclusively to the one from APP. However, APP also obtained its heparin product from China. Of the 1154 pharmaceutical plants with generic drug application to the FDA in 2007, only 13% were in the United States; 43% were in China, and 39% were in India.[65] Drug labels often claim that the pills are manufactured in the United States, but the listed U.S. plants are often the sites where foreign-made drug powders are pounded into pills and packaged.

Under the Prescription Drug User Fee Act, known as PDUFA, pharmaceutical companies pay the FDA to facilitate their PMA applications. Congress provided PDUFA with added power to impose restrictions on the sale of drugs and enforce regulations requiring that postmarketing surveillance studies be completed in a timely manner rather than delayed or evaded. Notwithstanding PDUFA, in 2008 the FDA was still missing target dates to act on NDAs because the FDA lacks sufficiently adequate staffing to handle PMA applications.[259]

FDA Clearance

The applicant must receive FDA approval of its PMA application prior to marketing the device as FDA approved. PMA approval is based on an FDA determination that the PMA contains sufficient valid scientific evidence to assure that the device is safe and effective for its intended uses.

The FDA lists certain products that are exempt from premarket notification requirements under the Food and Drug Administration Modernization Act of 1997 (the Modernization act),[116] which include the following endodontic products:

- Root canal posts
- Gutta-percha

Adverse Drug Events (ADEs)

Adverse drug events (ADEs) include "injury due to medications."[39] In 1999, the Institute of Medicine identified medication errors as a significant and preventable source of ADEs. Warning labels represent a quick reminder to highlight the most important instructions for the safe and effective use of prescribed medications. In one study, 50% of participants looked directly at the warning labels; 22% did not look at any. The recommendation was to move all of the warnings from the colored stickers to the main, white label.[304] More than 30% of those 65 and older take 10 medications daily.[304]

Primary Medication Nonadherence: Unfilled Prescriptions

Primary medication nonadherence occurs when a noncompliant patient fails to fill a prescription. Up to 25% of children discharged from a hospital with a prescription do not have those prescriptions filled.[348] Other data from emergency departments suggest that the frequency of unfilled prescriptions is between 7% and 35%.[348] Electronic prescriptions are more likely to be filled. When a prescription is electronically prescribed, the patient does not have an opportunity to lose or misplace it. Data suggest that clinicians should explore whether patients actually filled the medication prescribed or were taking it properly when a patient does not appear to be responding adequately to prescribed treatment.[199]

FDA MedWatch

Newly marketed drugs may not list all potential adverse drug reactions or events because of the limited number of patients in research trials before marketing. Therefore, the FDA's MedWatch program encourages clinicians to report known or suspected drug reactions. MedWatch encourages reporting of serious unexpected adverse drug reactions whose nature and severity are inconsistent with or absent from the drug's labeled warnings.[111-113,115,235] Clinician reporting to the FDA is confidential and voluntary. The patient's identity need not be disclosed. Clinicians should contact the FDA by telephone at (800) FDA-1088 or fax at (800) FDA-0178 to obtain the FDA Medical Products Reporting Program (MedWatch) form (FDA form #3500). The back portion of the *Physicians' Desk Reference* (PDR) contains a MedWatch form. Clinicians can also download the MedWatch reporting form at www.fda.gov/medwatch/getforms.htm; request the form by mail via MedWatch, 5600 Fishers Lane, Rockville, Maryland 20852-9787; and report suspected drug interactions, reactions, or adverse product events at www.fda.gov/medwatch/report.htm. Other electronic drug databases are available to report and access important drug interaction information.[340] For FDA-approved safety-related drug labeling online, see www.fda.gov/MedWatch.

Adverse drug reactions are injuries occurring when drugs are administered at usual doses. These reactions represent the primary focus of regulatory agencies and postmarketing surveillance. An adverse drug event suggests medication prescribing or dispensing errors that compromise patient safety. Unfortunately, clinicians report only between 1% and 5% of adverse drug incidents to the FDA, because clinician reporting is voluntary.[184] On the other hand, drug manufacturers must report known adverse drug incidents to the FDA.[113]

Clinicians should document adverse drug reactions not only in the progress notes but also in the allergy section of the chart in order to assess whether future use of the same drug is contraindicated and to prevent a harmful recurrence. Predictable adverse drug reactions that manifest should also be recorded as consideration for future disuse of the involved particular drug.

The Weber effect is a known phenomenon in the epidemiology literature in which the number of spontaneously reported adverse events for a drug tends to peak around the second year after a drug's initial marketing.[80] The influence of the Weber effect suggests that an increase in adverse events may be attributed to epidemiologic reporting bias, which may not reflect a true difference in adverse events.[80]

Compounding Pharmacies

More than 500 cases of fungal meningitis including 44 fatalities were attributed to injectable steroid solutions prepared by the New England Compounding Center (NECC), which subsequently filed for bankruptcy.[297]

Contamination is only one of five categories of risk associated with compounding pharmacies. The other four are subpotency, superpotency, overmedication, and medication replacement.[47]

Many patients received sterile injections for back and joint pain for procedures that lack high-quality evidence-based efficacy.[299] FDA-approved methylprednisolone acetate is sold by Pfizer and two generics companies. NECC's version did not contain preservatives, which avoided the FDA's regulatory process.

Compounding involves the preparation of medications on prescription to meet unique patient health care needs that cannot be met with commercially manufactured and marketed drug products. Compounding includes providing different strengths of a drug with different nonactive excipients for allergy patients or creating dosage forms that are more palatable for a patient. Compounding pharmacies and pharmacists work directly with the prescribing dentist to create customized personalized medication solutions for patients in doses and formulations that the pharmaceutical industry does not manufacture.

The Food and Drug Administration Modernization Act (FDAMA), enacted November 21, 1997, amended the Federal Food, Drug, and Cosmetic Act relating to the regulation of food, drugs, devices, and biologic products. FDAMA exempted individualized compounded drug products from the rigid federal current goods manufacturing practices. Under federal law, drugs that are not manufactured in conformity with federal current goods manufacturing practices are deemed to be "adulterated" under 21 U.S.C. 351(a)(2)(B) and subject to federal sanction. FDAMA, 21 U.S.C. 351(a)(2)(B) does not apply to a drug product compounded by a licensed pharmacist in a state-licensed pharmacy for an identified individual patient with a valid prescription order—or in limited quantities before receipt of the valid prescription order.[299]

Federal law prohibits compounding of drugs previously removed from the market because such drug products, or one or more of their components, have been found to be unsafe or ineffective. Compounding drugs that are copies of a commercially available drug product is also prohibited.

After the 1937 sulfanilamide disaster that killed more than 100 people, Congress passed the Food, Drug, and Cosmetic Act (FDCA), requiring drugs to be safe and properly labeled. In 1962, a requirement was introduced for proof of drug efficacy through "adequate and well-controlled investigations," in response to thalidomide's fatal side effects causing impaired limb development.

Traditional compounding pharmacies are not registered with the FDA as drug manufacturers. The FDA does not approve compounding pharmacy prescriptions before marketing. Nor does the FDA require compounding pharmacies to report adverse events. Instead, state law generally controls recordkeeping, certifications, and licensing for compounding pharmacies.

Dietary Supplements

The Dietary Supplements Health and Education (DSHE) Act allows dietary supplements to be sold directly to consumers without any oversight or FDA regulation. In response to intense lobbying by the multibillion-dollar dietary supplement industry, Congress in 1994 exempted these products from FDA regulation.[190,205,342] Products may contain amounts claimed, but they need not, and nothing can prevent their sale if they do not. For example, an analysis of ginseng[205] products showed no ginseng at all in some products. The DSHE Act places the burden of proof for dietary supplements' safety on the FDA and not the manufacturer. As a result, consumers of herbal supplements must depend on self-regulation within the industry for assurance of product quality, consistency, potency, and purity.[164,342] An understaffed and overworked FDA can hardly be expected to be a vigorous enforcer.

An herbal product label can state the way the product is intended to affect "the structure or function" of the body but cannot claim its use for a specific disease. Manufacturers thus use creative language that complies technically with the statute but generally is confusing or deceptive.[205]

A current medication history form should include over-the-counter, herbal, and illicit drugs, many of which have the potential for synergistic or antagonistic interaction with clinician-prescribed drugs.[342] Ma-huang, or ephedra, a major component of many weight loss supplements, has been associated with more than 800 adverse health effects, including the death of major league baseball player Steve Bechler. Ephedra is an amphetamine-like compound with the potential for overstimulating the central nervous system and heart. Ephedra/ma-huang has been associated with 54 deaths, primarily involving cranial hemorrhage or stroke.[42,146] A Los Angeles police officer who experienced a crippling stroke after taking an ephedra-based energy supplement, Dymetradrine Xtreme, was awarded $4.1 million.[32] The retail storeowner had read but disregarded 30 journal articles warning of strokes, heart attacks, seizures, and other ailments associated with the drug. The stroke occurred before the FDA banned ephedra from sale in April 2004. Nonetheless, Internet sales proliferated.[300]

Echinacea increases potential for liver damage when used with steroids. Gingko biloba and feverfew interfere with anticoagulants such as warfarin (Coumadin).[74] Dong quai root, willow bark, goldenseal, guarana, horse chestnut, and bilberry tablets/supplements also have antiplatelet properties.[300] Ginseng may cause rapid heartbeat or high blood pressure in some individuals, as well as coagulation disruption. Vitamin E has antiplatelet properties and inhibits vital clot formation. Gingko and selenium are powerful anticoagulants, three times stronger than vitamin E. In 1998, California investigators found about one third of 260 imported Asian drugs were either spiked with unlisted drugs or contained mercury, lead, or arsenic. In 2009, the FDA warned of 70 weight-loss pills containing potentially harmful drugs unlisted on their labels.

The formulations for herbal supplements—or *botanicals*, as they are correctly called—such as St. John's wort and

Echinacea are often complex, highly variable, and impure. Many are toxic, carcinogenic, or otherwise dangerous. Side effects include blood-clotting abnormalities, high blood pressure, life-threatening allergic reactions, abnormal heart rhythms, exacerbation of autoimmune diseases, and interference with life-saving prescription drugs.

The American Society of Anesthesiologists has warned patients to stop taking herbal supplements at least 2 weeks before surgery to avoid dangerous interactions with anesthesia.

Congress exempted supplements from oversight under a 1994 law that prevents federal regulators from requiring manufacturer's proof that botanicals are safe or effective, or even that dosage information on the label is correct. In 1999, the FDA allowed manufacturers to make dubious health claims, such as for products to treat conditions such as premenstrual syndrome and acne, although only a few botanicals have proved to be efficacious for anything.

Negligence Per Se

Violation of a health safety statute constitutes negligence per se if the patient was in the class of persons the statute was designed to protect.[292] Compliance with a health safety statute does not conclusively establish due care, because regulations require only minimal care and not (necessarily) prudent care or what the law regards as due care.[79,308]

Prescription Drugs

Clinicians should exercise extreme caution when administering or prescribing dangerous drugs. For sedative or narcotic drugs, cautionary directions should be written on prescriptions, and the pharmacist should place these directions on the prescription container as a patient reminder. For example, for the appropriate drugs, the clinician should prepare a prescription rubber stamp or obtain preprinted prescriptions that state the following:

> Do not drive or operate dangerous machinery after taking medication because drowsiness is likely to occur. Alcohol, sedative, or tranquilizing drugs will cause drowsiness if taken in combination with this prescribed drug.

The ADA and American Medical Association (AMA) provide prescription drug warning pads. Clinicians should document each drug information form provided with the prescription in the patient's chart.

Overuse of antibiotics risks resistant-strain development and side effects. Studies demonstrate generally no increased therapeutic efficiency of endodontic therapy with antibiotics when performed in the absence of facial swelling.[327] Unless persistent infections occur or compelling systemic reasons exist (e.g., uncontrolled diabetes, antibiotic prophylaxis necessary because of mitral valve regurgitation), antibiotics should not be prescribed prophylactically.[163,227] Neither pain nor localized swelling alone justifies antibiotics. However, extraoral swelling, cellulitis, or lymphadenopathy may require surgical drainage, antibiotics, or both. The U.S. Centers for Disease Control and Prevention (CDC) estimates about one third of all antibiotic outpatient prescriptions are unnecessary.[12]

Product liability may result when the manufacturer provides inadequate hazard warnings for safe use of its product. Product contraindications should be identified (Fig. 29-22).

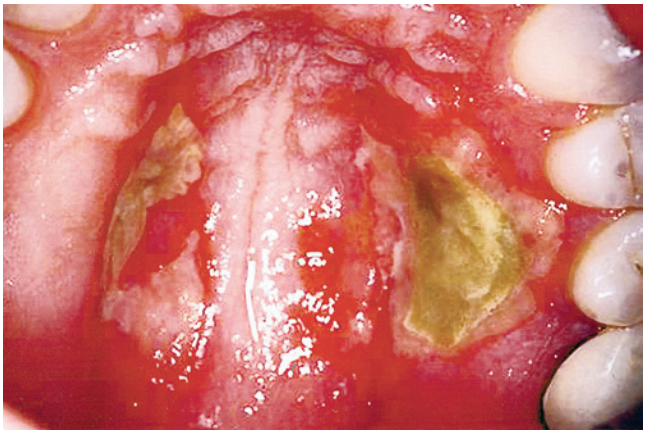


FIG. 29-22 Excessive local anesthetic resulting in palatal tissue sloughing with AMSA (wand) injection.

Ability to Foresee Unreasonable Risk

Each endodontic procedure has a variable degree of inherent risk. The standard of care requires that the clinician avoid unreasonable risks that may harm the patient. Treatment is deemed negligent when a reasonably careful clinician would have foreseen some unreasonable risk of harm to the patient. Failure to follow the dictates of sound endodontic practice increases the risk of negligently induced deleterious results. Accordingly, prophylactic endodontic practice is designed to prevent foreseeable or reasonably avoidable injury risks. Foreseeability connotes predictability.

It is not necessary that the exact injuries that occur be foreseeable. Nor is it necessary to foresee the precise manner or circumstances under which injuries are inflicted. It is enough that a reasonably prudent clinician would foresee that injuries of the same general type would likely result in the absence of adequate safeguards.[265]

Consent

Simple consent occurs when the clinician makes the treatment decision unilaterally and obtains the patient's consent without offering the patient a choice. Conversely, informed consent is a joint decisional process whether to proceed with treatment or not. Unconsented treatment constitutes a battery in legal parlance.

Patients are legally empowered to decide for themselves when substantial competing risks are involved with each separate treatment choice. A patient's therapeutic choice is particularly apt when there is no professional consensus for the best treatment. For instance, different implant systems exist with arguably different risks for each. A decision to proceed with an immediate implant versus waiting for osseointegration before initiating restorations requires that a risk-versus-benefit analysis be provided to the patient. Such decisions should include comparative peer-reviewed longevity research results compared with newer short-term immediate implant research. The benefits of immediate implant placement are attractive to both patient and clinician. To fairly compare researched implant survivability studies, the variables should similarly be controlled for each study, such as whether the implants are placed in native bone, socket size, and shape.

Emergency endodontic treatment often requires that the patient be provided pain relief. Acute pain clouds the patient's

decisional process; palliative treatment provides immediate pain relief so that on a return visit, the patient can rationally decide among treatment choices. With ample time, the patient can rationally decide and compare choices such as completion of endodontics and a crown versus extraction followed by an implant with crown. Also, an extraction that may result in progressive alveolar ridge bone loss and the need for bone grafting are considerations the clinician should explain to the patient.

Patient choices may be influenced by cost, convenience, time off work, travel needs, aesthetics, or a myriad of other reasons. The clinician plants the seeds of the decisional tree, but the patient decides which branch of the tree should be followed.

Informed Consent Principles

The legal doctrine of informed consent requires that the patient be advised of reasonably foreseeable material risks of endodontic therapy, the nature of the treatment, reasonable alternatives, and the consequences of nontreatment.[71,173,221] This doctrine is based on the legal principle that, as individuals, patients have the right to do with their own bodies as they see fit, including the right to prematurely lose teeth regardless of recommended dental treatment. Once the clinician has informed the patient of the diagnosis, treatment risks, prognosis, and nontreatment risks, as well as recommendations for corrective treatment or alternative therapy, the patient has the right to decide how, when, or if to proceed. An adult of sound mind is entitled to elect to do nothing about existing endodontic disease. Rather than elect corrective treatment, the patient may elect to suffer nontreatment consequences, including present or future tooth loss or apical abscess.

To be legally effective, a patient's consent to treatment involving potentially serious injury complications must include informed consent. Accordingly, a clinician has a fiduciary duty to disclose all material or essential information necessary for the patient to make a decision.[71,173,221] The scope of a clinician's duty to disclose information is measured by the amount of knowledge a reasonable patient requires to make an informed choice. Material information is the disclosure a clinician knows (or should know) would be regarded as significant by a reasonable person in the patient's position who must decide whether to accept or reject a recommended endodontic procedure.

If a clinician fails to reasonably disclose information that would make a reasonable person in the patient's position decline the procedure, the clinician may be liable should an undisclosed risk manifest. Beyond the foregoing minimal disclosure, a clinician must also reveal such additional information as a skilled practitioner of good standing would provide under similar circumstances.

The personal bond between clinician and patient has long been considered an essential element of the therapeutic environment. Information is empowering. Laws requiring informed consent place patients on a closer footing with their clinicians. In addition to the transfer of information in both directions, bonding takes place when the clinician and patient engage in a conversation of length and substance. Canned disclosure displaces this human interaction. One of the surest safeguards clinicians can have against malpractice litigation is the bond of personal relationship forged with their patients.[273]

Informed Consent

Adequate informed consent requires that a patient be informed of the ABC's—the alternatives, benefits, and complications—of a proposed treatment, along with the pros and cons of the alternative therapies or options.[71,173,221] A patient has a right to be informed of these ABC's from the treating clinician and not solely from an informed consent form or by a front office staff person who lacks a dental license. Informed consent must be presented in layperson's language, so that the patient can appreciate and understand what the treatment risks are.

A patient cannot legally consent to negligent care.[315] Accordingly, informed consent applies only to treatment that complies within the standard of care. If the clinician is reasonably careful during treatment but an untoward result occurs, the clinician is not liable if the patient was forewarned of the potentially unavoidable risk prior to treatment.

A reasonably unavoidable risk that manifests during treatment represents a maloccurrence and not malpractice. For example, a small percentage of endodontic failures occur for non-negligent reasons. The clinician should be sensitive to the patient's feelings of disappointment. When endodontics fails, regardless of cause, it is a 100% failure rate for the patient. On the other hand, if the risk was reasonably avoidable, then informed consent does not apply to reasonably avoidable risks.

Patient treatment refusals should be documented. Some states require informed refusal disclosures advising the patient about the consequences of refusing treatment or diagnostic modalities.

Informed Consent Application

Informed consent is a flexible standard that considers reasonably foreseeable consequences, depending on the clinical situation present both before and during treatment. For instance, a fractured or separated endodontic instrument left in the root canal creates the possibility of root canal failure or impaired success (depending on whether the fracture occurred in the coronal, middle [least favorable], or apical third of the root canal). The clinician must advise the patient of the relative risk of future failure and suggest treatment alternatives to correct the problem. An adequately informed patient can better make an intelligent choice among apicoectomy, referral to an endodontist utilizing microscopy for attempted retrieval, or periodic radiographic observation at recall visits.[76,183,197] In Figure 29-23, the clinician overinstrumented and perforated the inferior alveolar nerve (IAN), causing both a permanent dysesthesia and paresthesia. The clinician should have immediately referred the patient to an endodontist for attempted gutta-percha retrieval before the endodontic sealant containing eugenol set, which caused more deleterious chemical injury to the IAN. If retrieval was unsuccessful, the patient should have then been referred to a microsurgeon within 48 hours from occurrence for optimal opportunity to prevent chemical cytotoxic injury to the IAN.[262]

Adequate disclosure includes clinical judgment and experience, which assesses current research and applies it to the clinical needs of each patient. Today's advance may be tomorrow's retreat if materials, devices, or instruments lacking adequate long-term study of safety and efficacy predictably fail. For instance, the one-component bonding agent is more technique sensitive than its two-component predecessor. This is because the one-component agent contains acetone that desiccates the tooth surface and requires a moist surface to

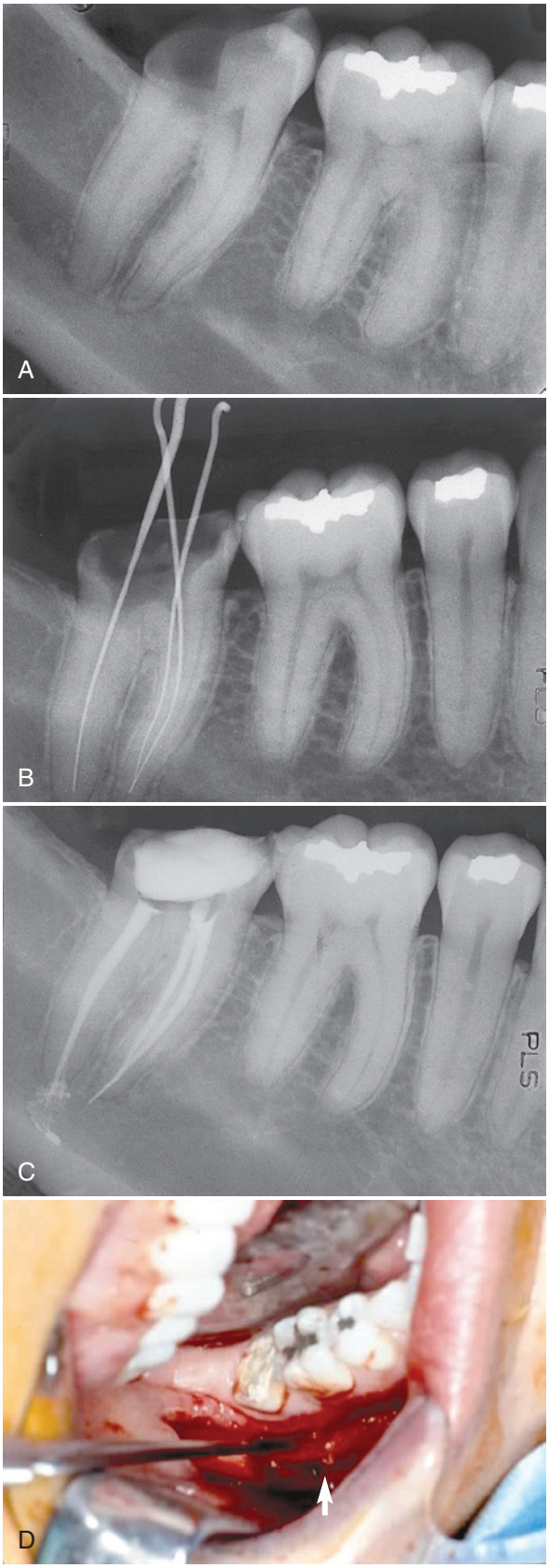


FIG. 29-23 A, Preoperative radiograph. B, Measuring film without a rubber dam. C, Postoperative radiograph. D, Decompression microsurgery showing protruding "dagger" of bone to right of retractor at inferior border of inferior alveolar nerve, resulting from prior perforation of the mandibular canal. This is probably due to file overinstrumentation. (Courtesy Anthony Pogrel, DDS, MD.)

avoid postoperative sensitivity. Long-term testing before marketing would have revealed that the two-step system is more reliable and forgiving.[312] Reasonable clinicians do not sacrifice patient safety for speed and profit and then compensate with desensitizing agents. Clinicians should be sensitive to limited research on new products or devices lest the patient be the injured guinea pig patient with products that were inadequately tested before marketing.

Material disclosure concerns whether the patient was provided sufficient information for a reasonable patient to achieve a general understanding of the proposed treatment or procedure. This disclosure includes information concerning any dentally acceptable alternatives, any predictable risks of serious injury, and any likely consequences should the patient refuse the proposed therapy. The standard to be applied is whether a reasonable person in the patient's position would have consented to the procedure or treatment in question if adequately informed of all significant perils.[66] Informed consent applies only to inherent risks of non-negligent treatment, because a patient's consent to negligent treatment is voidable as being contrary to public policy.[315] Accordingly, a patient who refuses necessary diagnostic radiographs should be refused treatment.

If warnings are limited to some risks but not others, some courts have held that the patient can recover for all surgical injuries including warned risks.[160]

Endodontic Informed Consent

If the practitioner's own statistical experience varies significantly from national statistics, using statistics presented in national literature regarding success rates for endodontic procedures is considered insufficient disclosure and does not fulfill the legal requirements of informed consent (Fig. 29-24).[145,291]

Among specialists, the reported incidence of treatment complications in endodontics is relatively low. Based on a Southwest Endodontic Society retrospective study, a reasonable endodontist or a practitioner with similar abilities should disclose the following facts to patients[288]:

1. Endodontic therapy cannot be guaranteed.
2. Although endodontic therapy is usually successful, a small percentage of teeth are lost due to complications or treatment failure, despite competent endodontic care.
3. Slight overextending or underfilling of root canals occurs in 2% to 4% of cases, which may contribute to treatment failure.
4. Slight to moderate transient postoperative pain may occur. Severe postoperative pain rarely occurs.
5. Irreparable damage to the existing crown or restoration secondary to endodontic treatment is uncommon after the access opening is restored.

Video-Informed Consent

Animated video-informed consent shown to the patient is a dynamic method of providing informed consent. Because the

INFORMED CONSENT

We are concerned not only about your dental health and endodontic treatment needs, but also about your right as a patient to make the treatment decision that you feel is best for you. Our commitment to you is to provide you with detailed and complete information about your dental needs as we diagnose them. We will share our diagnostic processes with you, and we invite and welcome all of your questions regarding our treatment.

Towards this aim of a mutual sharing of information, we feel it is important to advise you of the reasonably foreseeable risks of endodontic therapy. The following is important information you should consider to aid your treatment decision.

- Root canal therapy is a procedure designed to retain a tooth that may otherwise require extraction. Root canal therapy has a very high degree of success. However, it is a biological procedure, and results cannot therefore be guaranteed.
- Approximately 5% to 10% of teeth that have undergone nonsurgical root canal therapy may require retreatment or root-end surgery.
- Despite our best efforts, approximately 5% of endodontically treated teeth may fail and require extraction.
- Final restoration (crown) of the tooth that has undergone root canal therapy is essential to root canal success and retention of the tooth. A final restoration should be completed within 30 days of root canal therapy. This restoration should be done by your restorative dentist.

Signature of Patient (or Parent) _____ Date _____

Witness _____ Date _____

FIG. 29-24 Informed consent form.

video-informed consent is considered part of the clinician's records, in the event the patient disputes having ever been advised of (1) the nature of endodontic disease, (2) the availability of endodontic specialists, or (3) the relative indications for nonsurgical versus surgical endodontic care, the videotape can be played back to the jury as proof that the patient was informed. It is doubtful a jury would believe a forgetful patient who admits having previously viewed the videotape, because the videotape refreshes the stream of memories that may have otherwise faded into the unconscious.

A patient viewing the videotape is more likely to understand the informed consent disclosure of a technical procedure, particularly if the video contains illustrated animation. After viewing the videotape and discussing its contents with the clinician, the patient should sign a video consent form to verify that the video was viewed and all the patient's questions were answered.

Alternative Technique Choices

Currently taught methodologies include filling the root canal with the lateral compaction method or using vertical warm gutta-percha carrier delivery systems, such as heat-transfer units for warm gutta-percha.[197] Each technique has zealous advocates. Both methods are reasonable and acceptable choices, and each method conforms to the standard of care. The choice of techniques changes as new products are introduced and scientific research is conducted. The vertical warm gutta-percha technique promises to deliver improved flow to fill lateral canals and is likely to predominate in the 21st century as the preferred methodology. Nonetheless, lateral

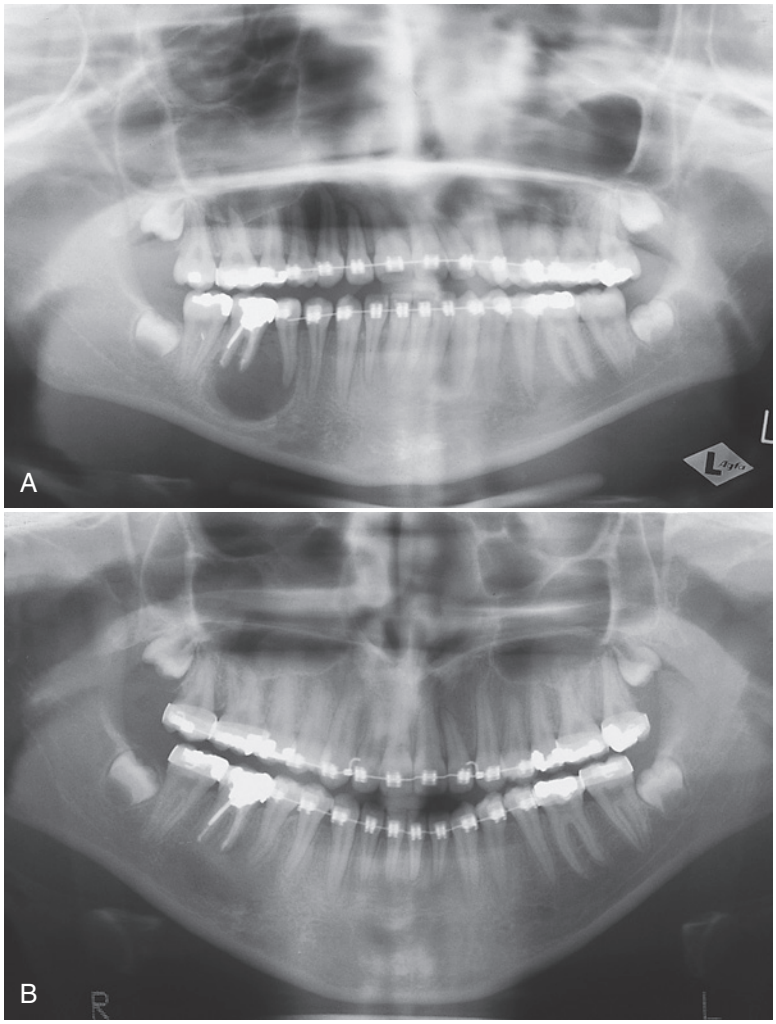


FIG. 29-25 A, Preoperative panoramic radiograph showing a successful apicoectomy and retrograde seal. B, Postoperative panoramic radiograph showing a successful apicoectomy and retrograde seal. (Courtesy Edmond Bedrossian, DDS.)

compaction has a long-term proven track record, particularly with less-experienced practitioners, and is less technique sensitive. Regenerative endodontics will have greater utilization as this scientific application increases.[16]

Restorative Informed Consent

Patient choices for restorations may discount longevity and elect instead aesthetics. In one study, amalgams lasted 15 years, composites 6 years, and conventional glass isomers 7 years.[116] Shorter-term restorations' longevity increases the frequency of restoration replacement and increased potential for endodontic therapy with each replacement. Nevertheless, it is the patient's treatment choice after the clinician first explains relative risks, benefits, and alternatives.

Different Schools of Thought

After the clinician advises treatment, if other reasonable clinicians would disagree or if there are other respectable schools of thought on the correct treatment, this material information should be disclosed to the patient.[325,346] For example, there may be two schools of thought concerning the optimal treatment for retrofilling and apicoectomy. One school posits that a retrograde with mineral trioxide aggregate (MTA) or Biodentine is the appropriate procedure, whereas another school asserts that retrograde with Super EBA is a reasonable choice.[343] One can assume further that an explanation of these two different methods of treatment constitutes material information for the purposes of informed consent. The mere fact that there is a disagreement within the relevant endodontic community does not establish that the selection of one procedure as opposed to the other constitutes negligent endodontic therapy. Because competent endodontists regularly use both procedures, a patient would have a difficult time proving dental negligence (i.e., that the endodontist failed "to have the knowledge and skill ordinarily possessed, and to use the care and skill ordinarily used, by reputable specialists practicing in the same field and in the same or a similar locality and under similar circumstances"). Moreover, neither school of thought possesses long-term clinical research results to prognosticate apical sealant insolubility rates in an 18-year-old patient with an additional 60-year life expectancy.[293] Long-term durability depends on an insoluble seal to reduce the risk of bacterial leakage.[66,228] Figure 29-25 represents a preoperative and postoperative radiograph of a successful apicoectomy and retrograde in a 14-year-old.

On the other hand, the specialist would have a duty under the previous hypothetical circumstances to disclose the two recognized schools of treatment so that the patient could be sufficiently informed to make the final personal decision. An endodontist, being the expert, appreciates the risks inherent

in the procedure prescribed, the risks of a decision not to undergo the treatment, and the probability of a successful outcome of the treatment. Once this information has been disclosed, this aspect of the endodontist's expert function has been performed. The weighing of these risks against the individual subjective fears and hopes of the patient is not an expert skill. This evaluation and decision constitute a nondental judgment reserved for the patient alone.[71,173] In this hypothetical situation, failure to disclose such material information would deprive the patient of the opportunity to weigh the risks versus the benefit. Consequently, the clinician would have failed in the duty of disclosure, which the doctrine of informed consent requires.

When improved technology offers clearly superior results, it no longer becomes a patient choice issue but rather a requisite requirement to fulfill the standard of care. Apical microsurgery with ultrasonic tips for retrofilling exemplifies improved technology and the current standard of practice. Likewise, apical retrogrades should be done with MTA and not with amalgam.[66] Finally, microscopic endodontics represents the current standard of care for apical surgery or fractured instrument retrieval.[76,86,166,183,197]

Avoiding Patient Claims

If a clinician fails to obtain adequate informed consent, a plaintiff can recover damages (even in the absence of any negligent treatment) should the patient testify that performed treatment would have been refused if the clinician had provided information concerning possible risks. Therefore, discussions of treatment risks with the patient should be documented. Informed consent forms are very helpful, although not legally mandated. A jury may believe that the patient was informed orally although the patient's attorney may argue that if it is not documented it did not happen. Equally, if not more important than consent forms, is a chart notation indicating that the clinician discussed informed-consent risks and alternatives (and that the patient understood and accepted this information).

Patients may mentally block out frightening information. Trauma and a potent anesthetic agent can create retrograde amnesia. Therefore, clinicians must document (in the patient's chart) any risks, benefits, alternatives, and consequences of nontreatment provided to the alert patient before sedation.

Clinicians should follow only the patient-authorized and consented treatment plan. If an emergency precludes advising treatment risks to the minor patient, lack of informed consent may be defensible as implied consent (because no reasonable parent would refuse necessary emergency treatment).

The following example demonstrates how a clinician should record any recommended treatment the patient has refused and the reason for refusal:

> Patient refused endodontic referral for consultation with endodontist (Dr. Goodguy) because husband was laid off work last month and cannot afford treatment. After Dr. G. provided explanation, patient states she understands detrimental delay risks but is willing to assume these risks.

Patients may initial any referral refusal on the chart. Although patient signature is not mandatory, it will enhance the clinician's credibility should the patient later dispute a referral was made.

Reasonable familiarity with a new product or technique is required before it is used. In addition, a patient is entitled to know the clinician's personal experience with a particular modality because the patient has a right to choose between reasonable alternatives or to seek care from a clinician who has more extensive experience with a particular modality or new product. A clinician who fails to obtain informed consent may be liable for injury caused by a product or instrument. The fact that the clinician followed the manufacturer's instructions is no defense if the clinician did not provide adequate information concerning a product or instrument risk (to permit the patient to intelligently weigh the disclosed information and choose among treatment options).

Referrals to Other Specialists

Every clinician, including specialists, will at some time need to refer a patient to another specialist for treatment to comply with the standard of care required of a reasonably careful clinician.[123]

Generally, if the referral takes place within the same dental practice, the legal doctrine of respondeat superior ("let the master answer") may be applicable. Under this doctrine, a clinician is liable for the dental negligence of a person acting as his or her agent, employee, or partner.[264] Liability is determined by whether the principal clinician controls the agent clinician's activities or methodology, regardless of whether such control is actually exercised.

If the referral is made (even within the same physical environment) to an "independent contractor" endodontist who does not diagnose or treat under the direction or supervision of the referring clinician, and that referring clinician has no right of control as to the mode of performing the treatment, the principle of agency and responsibility for the acts of another does not apply.[223] The legal test for agency is the principal's right to control, regardless if the control is exercised over the agent. However, if the independent contractor or referring clinician in the same practice does not provide adequate notice of this independent relationship to the patient, the independent contractor will still be liable under the legal doctrine of ostensible agency. Thus, despite an independent contractor agreement, a patient can legally infer the so-called independent clinician is instead an employee, because neither clinician in the group practice disclosed an independent contractor relationship to the patient.

To ensure that the referred clinician is not considered the agent of the referring clinician within the same facility, fees for the referred clinician should not be set by the referring clinician nor should the fees be divided equally or shared based on some other arrangement. Also, the referred clinician should bill separately and exercise independent diagnostic and therapeutic judgment. The patient should be advised that the referred clinician is independent of the referring clinician. Otherwise, the referred clinician or specialist may be regarded or inferred to be the ostensible agent of the referring clinician in the same office although not intended. The legal test for agency is how the facts or circumstances appear to a reasonable patient, regardless of the clinician's understanding or written contract intent that the other treating clinician is to be considered an independent contractor. Thus, agency may be actual in fact or, alternatively, ostensible (i.e., implied by circumstantial conduct).[174]

Surgical versus Nonsurgical Endodontics

Litigation to determine whether nonsurgical or surgical endodontics was the proper treatment choice will not be decided by any one clinician, the ADA, the AAE, or the ablest of judges. Rather, after considering all the evidence and the opinions of experts, a jury of the patient's peers will decide the disputed matter. Depending on the individual case, the jury may decide that either a combination of nonsurgical and surgical endodontic therapy, rather than one method exclusively, should have been attempted. The jury may also decide that the patient should have been advised of the availability of such alternative therapy. Similarly, the jury may determine that apical surgery should have been done microscopically rather than macroscopically, depending on expert testimony regarding the relative advantages of each method or upon presenting clinical circumstances (e.g., suspected calcification, viewing a separated instrument).

Microscopic Endodontics

Microscopic endodontics improves posture and reduces neck and back fatigue.[25] Identification of microfractures in teeth[76,183,197,230] and removal of diseased dental tissue are easier and more accurate when done microscopically.

> The operating microscope is an indispensable tool for state-of-the-art endodontic treatment. The specialty practice should not be without a microscope; this instrument is useful in all phases of endodontic treatment from diagnosis to placement of the final restoration.[166]

Microscope

A 2007 survey of 1091 endodontists indicated that 90% of endodontists have access to and use the OM in their practice, a dramatic increase from 52% in use in 1999.[15] The 2012 AAE Position Statement is that the microscope is an integral and important part of the performance of modern endodontic techniques.[15]

According to the AAE, microscope benefits are as follows:

- Locating hidden canals obstructed by calcifications or reduced in size
- Removing materials such as solid obturation materials (silver points and carrier-based materials), posts, or separated files
- Removing canal obstructions
- Assisting in access preparation to avoid unnecessary destruction of mineralized tissue
- Repairing perforations
- Locating cracks and fractures that not clinically visible or palpable with an endodontic explorer
- Facilitating all aspects of endodontic surgery, particularly in root-end resection and placement of retrofilling material
- Photographic documentation enhancement
- Ergonomic benefit[15]

Magnification

The modern approach to periradicular surgery (PRS) is through magnification, illumination, and microsurgery. Prognosis is greatly improved when these techniques are employed. Although loupes are helpful, improved illumination and magnification of the surgical operating microscope are essential for PRS. Microscopy provides a more detailed examination of the root apex anatomic features such as isthmuses, accessory canals, fracture cracks, and crazing. Failure to shape, clean, and fill the root canal system is acknowledged as the main reason for failure of root canal treatment. The same principle applies to apical surgery. A disadvantage of MTA for retrograde sealant is handling and placement. MTA is hydrophilic and requires moisture to set with up to 3 hours of setting time. Newer agents to replace MTA include Biodentine, containing tricalcium silicate.

Root canal systems have much greater complexity at the level of root resection than previously recognized. At least 3 mm of the root apex should be removed to ensure successful retrograde obturation of all portals of exit. Angle of resection should be close to horizontal.

Floor of the sinus may be only 1 or 2 mm above root apices of the posterior dentition. Accordingly, the risk of sinus perforation increases when apical pathosis is present. Care must be taken to prevent debris from entering the sinus during maxillary posterior root canal therapy.

Separated Instruments

The risk of broken files can be reduced if all hand and rotary nickel and titanium (NiTi) cleaning and shaping files are not resterilized and reused but are discarded after single-tooth use. Efficiency also increases, because approximately 50% of cutting efficiency is lost after initial use. Episodes of broken instruments will be dramatically reduced and nearly eliminated when files are used correctly and discarded after single-tooth use. Breakage increases sharply when hand or rotary shaping files are reused. Therefore, the clinician should discard rotary instruments after a single-use visit. Chair and staff time efficiency, along with improved safety, dictates single-visit use of files.[228]

The clinician should save broken or defective instruments (e.g., a file that was broken inside a canal) (Fig. 29-26). The instrument manufacturer may be liable because the product was defective, rather than the clinician being liable for dental negligence.[266] Electron microscopy spectrographic analysis can determine if manufacturing defects with contaminants caused the break, rather than the clinician excessively stressing the instrument.

Equipment and Supplies

Equipment should be kept in good repair by checking and following the manufacturer's recommended maintenance schedule. The clinician should carefully read the manufacturer's instruction warnings on instruments and inform staff of any important points. Infection control in operating dental equipment is mandatory, such as updating and maintaining dental units with check valves to prevent water retraction or suck-back.[70] The clinician should inspect check valves monthly and change clogged valves immediately.[287] Water retraction testers, at no charge to the clinician, are available from some manufacturers (A-dec [Newberg, OR], for instance). The clinician or a staff member should also disassemble the unit's handpiece, run water through the line for a few seconds, and then stop. If a bubble of water is visible at the end of the water hose holes, the check valve is operating properly. If a bubble of water is not visible, water may be sucked back because of an absent or clogged check valve. An absent or clogged check valve is a source of cross-contamination, so it is important for the clinician or staff to perform weekly spore testing of the autoclave and monthly bacteriologic testing of water lines.[17] The

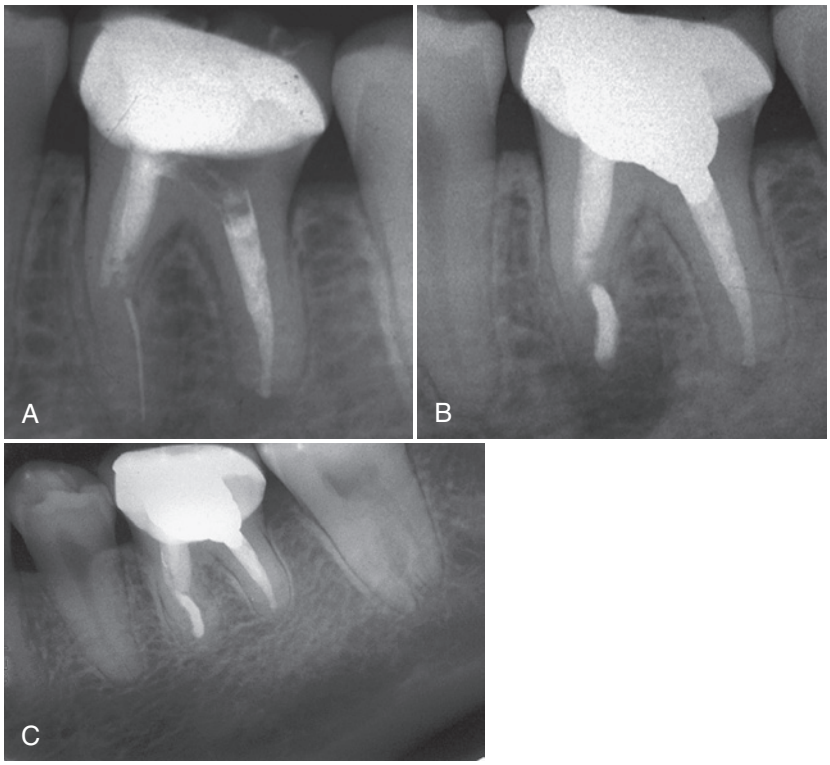


FIG. 29-26 A, Nickel-titanium file broken inside the mesiobuccal canal of the mandibular first molar. B, Due to patient discomfort, the segment was surgically removed and mineral trioxide aggregate (MTA) was used as root-filling material. C, Periapical radiograph 2½ years posttreatment shows complete healing. (From Torabinejad M, Walton RE: *Endodontics: principles and practice,* ed 4, St. Louis, 2009, Saunders/Elsevier.)

clinician or staff should consider using filters and should flush dental-unit water lines between patients and also daily with FDA-approved chemical disinfectants to reduce water tubing biofilm. Some chemical disinfectants claim improved cost effectiveness by increasing handpiece and bur longevity concomitant with water-line purging or with continual use during treatment. Currently available water line delivery systems, such as AquaSept, provide for safe water delivery or coolant to the handpiece or sonic scaler with autoclavable tubing. Figure 29-27 shows a student who was burned by an inadequately maintained handpiece that overheated. This case settled for $280,000.

Clinician's Liability for Staff's Acts or Omissions

A clinician is liable for the acts or omissions of the clinician's staff under the doctrine of respondeat superior ("let the master answer"). This is termed *vicarious liability,* which means that the clinician is responsible not because of personal wrongdoing but because the clinician assumes legal responsibility for the conduct of employees and agents who act in the course and scope of their employment.

The clinician should instruct the staff in advising patients regarding posttreatment complaints. For example, if the staff ignores signs of infection, such as difficulty in swallowing or breathing or elevated temperature, and dismisses the patient's complaints as normal postoperative swelling, the clinician may be held liable for injury to the patient, such as delayed cellulitis, diagnosis or treatment of Ludwig's angina, brain abscess, or other serious complications.

A clinician must be cautious when delegating responsibilities and give clear instructions to ensure that staff members properly represent the clinician and the chosen practice methods. Auxiliaries should not be allowed to practice beyond their competency level or license. For example, in states where assistant-placed restorations are legally permissible, the clinician should check any assistant-placed restoration before patient dismissal. Staff members should not make final diagnoses or handle patient clinical complaints without the clinician's involvement and consultation. Staff should be instructed to ask appropriate questions and relay the patient's answers to the clinician so the clinician can adequately determine what should be done.

Abandonment

Once endodontic treatment is initiated, the clinician is legally obligated to complete the treatment regardless of the patient's payment of any outstanding balance. This requirement is posited on the legal premise that any person who attempts to rescue another from harm must reasonably complete the rescue with beneficial intervention unless another rescuer (i.e., clinician) is willing to assume the undertaking.[69,195] Another view is that should a patient be placed in a position of danger unless further treatment is performed, the clinician must institute reasonable therapeutic measures to ensure that adverse consequences do not result.[212,296]

A clinician performing endodontic therapy should have reasonable means of communicating with patients after regular office hours to avoid a claim for abandonment. A recorded message is inadequate if the clinician fails to check for recorded messages frequently. Therefore, answering services, pagers, or cell phones is required by the standard of care.

If the clinician providing endodontic therapy is away from the office for an extended period, a substitute on-call clinician should be available for any endodontic emergency and to answer patients' emergency calls. The endodontic treating clinician should arrange in advance for emergency service with

FIG. 29-27 A, Before injury. B-D, A clarinetist music student was burned by an inadequately maintained handpiece that overheated. This lawsuit was settled for $280,000.

a covering clinician. Leaving a name on the office answering machine or with the answering service without first determining the availability of the covering clinician is a mistake.

To avoid an abandonment claim, several prophylactic measures apply:

1. No legal duty requires a clinician to accept all patients for treatment. A private practice clinician may legally refuse to treat a new patient, despite severe pain or infection, except on the grounds of race or disability.[49,329] If treatment is limited to emergency measures only, the clinician must advise the patient that only temporary emergency endodontic therapy is being provided and that endodontic treatment is incomplete. The clinician should record this information in the patient's chart. For example:

   > Emergency palliative treatment only. Patient advised endodontic treatment of tooth #8 needs to be completed, either here or with another clinician. Explained complications likely if not soon completed, including infection recurrence, tooth loss, or both.

   The patient should also be asked to acknowledge that treatment is limited to the existing emergency by endorsing an informed consent to emergency endodontics statement as follows:

   > I agree to emergency endodontic treatment of my tooth #8 and have been advised that (1) emergency treatment is for temporary relief of pain and (2) further root canal treatment of tooth #8 after emergency treatment is necessary to avoid further complications, including, but not limited to, pain, infection, swelling, tooth fracture, abscess, or tooth loss.

2. No legal duty requires a clinician to continue treating former patients on recalls or subsequent emergency care once treatment is complete. Thus, completion of endodontic treatment for tooth #19 does not legally obligate the clinician to initiate endodontic therapy for tooth #3 if endodontic disease of #3 began months after the clinician completed treatment of tooth #19.
3. Any patient may be discharged from a practice for any arbitrary reason, except on the grounds of race or disability, so long as all initiated treatment is completed. Accordingly, a former patient who evokes memories of a "frictional" relationship, who is financially irresponsible, or who arrives at the office after an absence of several years with an acute

apical abscess in a site where previous care was not rendered may legally be refused treatment.
4. It is not considered abandonment if a patient is given reasonable notice to seek endodontic treatment with another clinician, and the patient is willing to seek endodontic services elsewhere.[229] Thus, if rapport with the patient dissolves, the clinician should not hesitate to suggest that the patient would be better served if any remaining endodontic treatment were performed by a different clinician.

The clinician may discontinue treatment, provided it is not done at a time when the patient's dental health will be jeopardized (e.g., in the middle of treatment). To discontinue treatment, the clinician should do the following:
1. Notify the patient of the plan to discontinue treatment after a certain date.
2. Allow enough time for the patient to obtain care with another clinician, usually 30 days.
3. Offer to make emergency service available during the interim 30 days until a new clinician is obtained.
4. Provide diagnostic-quality records, copies of radiographs, and other pertinent clinical information in transfer records to the new treating clinician.
5. Allow the patient to select a new clinician or suggest referral by the local dental society if a referral service exists.
6. Document all of this in the patient's records, including a copy of a certified letter sent to the patient notifying discontinuance of further treatment.

Expert Testimony

The standard of care a clinician must possess and exercise is particularly within the knowledge of dental experts. However, there are occasional exceptions where the conduct involved is within the common knowledge of laypersons, in which case expert testimony is not required. In determining whether expert testimony is required to establish negligence, one California court commented: "The correct rule on the necessity of expert testimony has been summarized by Bob Dylan: 'You don't need a weatherman to know which way the wind blows.'"[170]

Incorrectly operating on the contralateral side because of mismounted radiographs or marking the wrong tooth on an endodontic referral card is an example of negligent conduct within the common knowledge of laypersons for which expert testimony is not required. The tort of battery results from unconsented treatment.

Increasingly, courts act as gatekeepers regarding the admissibility of scientific expert testimony. Anecdotal comparisons appear compelling, but such evidence may be judicially excluded in the courtroom. Some experts rely solely on their skill for experience-based observation, rather than research testing of risk factors, such as occurs with epidemiologic research.

The U.S. Supreme Court (in the Daubert four-factors checklist[78]) held that in federal court an expert's testimony may be reliably admitted into evidence based on the following:
1. Whether the expert's technique or theory may be tested or refuted
2. Whether the technique or theory has been the subject of peer review or publication
3. Known or potential rate of technique error
4. Degree of acceptance of a theory or technique within the relevant scientific community

Courts determine whether testimony is based on the application of scientific principle or clinical experience. In general, courts are flexible and adaptable in determining whether a bright line separates scientific and unsupported nonscientific evidence. Often technical and specialized knowledge gained through continuing education and scientific journals, rather than an expert performing the research, is considered sufficient.

State courts are not bound to follow the Federal Rules of Evidence, but many do comply.[30] Nonetheless, U.S. Supreme Court decisions influence state courts that consider placing limitations on expert testimony. State court trial judges may either be more liberal or more restrictive in admitting expert witness testimony. If the trial court judge denies admission of scientific evidence as unreliable, untrustworthy, or irrelevant, the result may be to preclude an expert offering any opinion. On the other hand, the court may admit such evidence and instruct the jury members that they may consider the scientific basis for the expert opinions and give such evidence the evidentiary weight it deserves.

In *Weisgram v. Marley Co.*,[330] the U.S. Supreme Court held that under Federal Rule of Civil Procedure 50(a), either a district (trial) court or a court of appeals may enter judgment for defendant, notwithstanding the plaintiff's verdict, if either court determines that admitted expert testimony was unreliable and inadmissible under *Daubert v. Merrell Dow Pharmaceuticals, Inc.*[78]

National Practitioner Data Bank

Insurance carriers are obligated to report all settlements to the National Practitioner Data Bank. These data are only accessible to hospitals for staff privilege credentialing and state licensing boards but not individual patients.

Medical negligence occurring in hospitals is too often underreported.[81] The Public Citizens' Health Research Group reports that more than half of U.S. hospitals have never reported an adverse incident to the National Practitioner Data Bank, an incredulous occurrence rate.[72]

Professional Negligence Incidence

In 2011, health care spending totaled $2.7 trillion, or 17.9% of the gross domestic product. Total spending for doctors' services was $541.4 billion.[65]

Harvard School of Public Health found that 97% of claims involving medical injury are meritorious.[251] A survey of federal judges found that 85% believed frivolous lawsuits were not a problem. Juries are more likely to rule in favor of a defendant than judges.[193,320]

Tort filings in state courts, which constitute only 6% of all filings, declined by 21% over the decade 1997-2006.[231] Tort actions in U.S. district courts also dropped 21% (2003-2007).[320] Medical malpractice filings dropped 8% during this period. Contract cases, on the other hand, which are more likely to involve businesses, increased 23% over the same time period.[193,231]

A 2003 GAO report submitted to Congress found that malpractice awards are only one factor contributing to increases in premiums for medical professional liability insurance.[302] Other factors cited include falling investment returns for insurance companies and normal business cycles. The report concluded that assertions that insurance premiums are driving clinicians out of business and making health care inaccessible were "not substantiated or did not affect access to health care on a widespread basis." The GAO report also stated that it is

not possible to prepare a full analysis of the issue because comprehensive insurance data are not available.

Because malpractice premiums account for about 1% of health care spending, imposing caps in liability cases would therefore have essentially no impact on the cost of health care. Evidence that this unnecessary remedy for a perceived but absent wrong is the National Association of Insurance Commissioners' data from 1995 to 2000, which show new medical malpractice claims declined.

The existing civil liability system permits each state the autonomy to regulate the resolution of professional liability actions within its borders. This is a hallmark of our American justice system.

The popular image holds that jurors are overly sympathetic to patients with unfortunate dental outcomes, regardless of whether anyone is to blame. However, research on juror attitudes demonstrates the opposite. Thus, weak cases seldom succeed despite serious injuries.[34,302]

On any given day, even the best clinician may make a negligent mistake. About 5% of clinicians account for the majority of all malpractice claims, according to reports filed with the National Practitioner Data Bank. Among clinicians who have settled five or more malpractice claims, only 13.3% have been subject to professional discipline, according to the Public Citizens' Health Research Group's analysis of the data. About 45% of otolaryngologists admit medical errors in all phases of their practice. However, even this proportion of clinicians reporting medical errors is underestimated.[272] One study estimated the number of fatal medical errors doubled in the preceding 3 years.[81]

Managed Care and the Employment Retirement Income Security Act

Ten states passed managed care liability laws that permit patients to sue managed care organizations for damages resulting from delayed or refusing authorization for treatment the clinician recommended as medically necessary. A U.S. Supreme Court decision nullified those state laws for the 140 million Americans covered by health plans that are regulated by the Employment Retirement Income Security Act of 1974 (ERISA).[1] ERISA restrictions do not apply to government employees' dental insurance.

MALPRACTICE INCIDENTS

What to Do If You Are Sued

If served with a summons or complaint, call your carrier immediately. Your carrier will assign an attorney. Be candid with the attorney. You will be asked to provide and interpret your dental records. Assemble all available records regarding your patient's treatment and review them thoroughly with your attorney prior to your deposition. Cell and office phone records can confirm calls to the patient, pharmacy, or specialists.

Screw Posts

Screw posts represent a restorative anachronism. The risk of root fracture is too great compared with the benefit, particularly when reasonable and superior passive alternatives exist (see Chapter 22). Even if the screw post is initially placed passively, the temptation to turn the screw is too great, considering human nature. Therefore, screw posts are not a reasonable and prudent treatment choice.[68]

Paresthesia Prevention

Endodontic surgery in the vicinity of the mandibular canal or mental foramen carries with it the significant risk of irreversible injury to the inferior alveolar or mental nerve. Consequently, the clinician must advise the patient in lay terms of the risk of temporary or permanent anesthesia or paresthesia before any surgery near these structures is performed. To document that adequate informed consent was provided, the clinician should have the patient execute a written informed consent form confirming that the patient was so advised. Informed consent is no defense if the surgery was negligently chosen or performed, because informed consent only applies to non-negligent treatment risks. CBCT imaging has greatly reduced if not eliminated entirely the likelihood of IANC perforation. CBCT provides diagnostic guidance before therapy. Also, a final diagnosis with CBCT can confirm or rule out IANC penetration post treatment to facilitate prompt removal of foreign materials that have penetrated into the IANC. Also sealants can chemically leak through the paper-thin porous (5% to 10%) IANC cortical borders even if the initial overfill only reaches but does not penetrate the underlying IANC. Delayed onset of paresthesia or dysesthesia up to 4 days can occur with sealant chemicals such as eugenol or resin components.

A clinician initiating root canal therapy on a mandibular posterior tooth must have (1) an awareness of the vital neurovascular bundle containing the IAN, which commonly approximates the ends of these roots, and (2) a diagnostic-quality image that accurately demonstrates both crestal borders of the IANC. Should the radiograph only demonstrate one border of the IANC, it is highly likely the evident border is the inferior crest of the IANC and not the superior crest.[206] If endodontics is done without adequate diagnostic radiographs, the patient is placed at great risk if there is anatomic variation or vital neural bundles lie in close proximity to these roots.[110,180,206,289,336] Also be mindful that as the IAN ascends toward the mental foramen, the IAN may have an anterior loop before exiting the mental foramen.[138,175,176,277] Overfilling or overinstrumentation of the root ends can cause traumatic or chemical injury to the IAN bundle, resulting in potentially permanent altered or total loss of sensation from paresthesia (partial numbness), anesthesia (total anesthesia), or dysesthesia (burning pain).[261]

The risk of overfills containing toxic materials causing irreversible neuropathies results from (1) inadequate imaging of the proximity of both borders of the IANC (Fig. 29-28, *A* to *C*), (2) inadequate measurements during cone measurement, (3) not limiting filling materials to within the confines of the shaped root canal space, and (4) not utilizing CBCT when instrumenting or performing surgery adjacent to vital structures such as the IANC and maxillary sinus.

The eugenol component of the root canal sealer part of the overfill is known to be cytotoxic. Consequently, endodontic overfill of the root apex is capable of producing not only mechanical damage[219] but also chemical injury. Accordingly, a prompt microsurgery referral to remove the injurious materials from the IAN inside the IANC nerve space before overfill material toxins are chemically set is mandatory.[254] Better yet is to take precautions to prevent *any* significant injurious overfill.

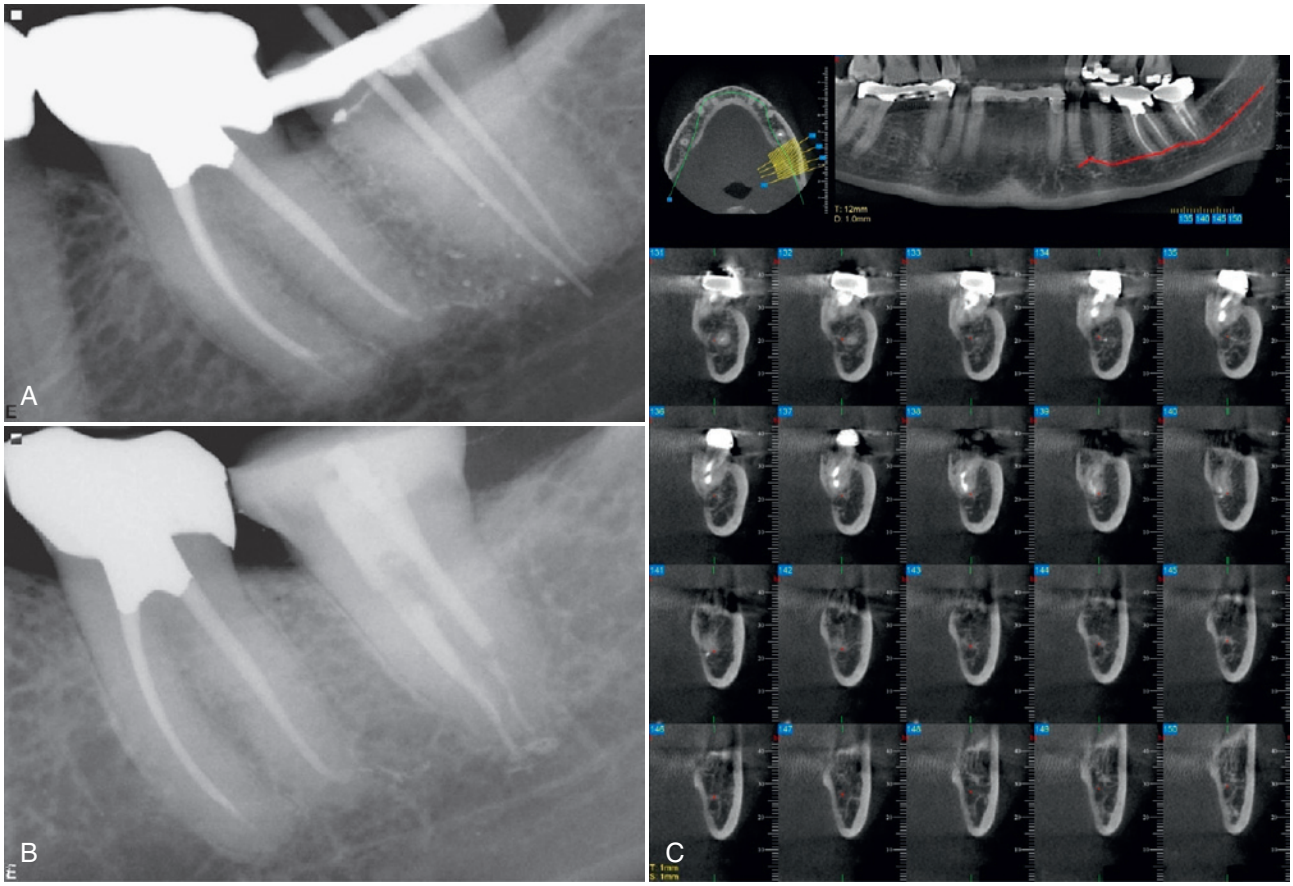


FIG. 29-28 A, Unclear periapical radiograph shows both cortical borders of inferior alveolar nerve canal (IANC). B, Cones placed into IANC, showing measurements beyond apices. C, Overfill into IANC better shown on cone-beam computed tomography (CBCT) than periapicals of A and B.

The use of electronic measuring devices such as an electronic apex locator is standard technology for accuracy when working deep within the root canal system. Combined with diagnostic quality radiographs, an apex locator helps provide accurate length readings. If it is determined that the apex is in extremely close proximity to the IANC, extra precaution must be taken to prevent overextension of instruments or materials including slightly short fills and apical packing techniques.

Precise measurements during cleaning, shaping, and obturation compacting stages of endodontics minimize significant overfill incidents. Obturating filling materials with sealants may be not only toxic to nerve cells but also create a mechanical pressure injury to the nerve cells themselves. Immediate microsurgical referral should be done on a stat basis within the first 48 hours of neuropathic symptoms for retrieval of any overfill materials that "harpoon" the IANC to protect against irreversible neuropathies (see Fig. 29-30, *C*, presented later).[254]

Serious mechanical and chemical injury to the IAN results from a lack of recognition of the overfill piercing the IANC or disregard for neuropathic symptoms the patient reports any time between 4 hours and 4 days postoperatively that are consistent with neurologic deficits such as paresthesia or burning pain dysesthesia.

Referral to an oral surgeon/microsurgeon is time dependent. Pogrel's study demonstrated complete reversal of neuropathic symptoms secondary to overfill if microsurgery was done within 48 hours.[254] Two thirds of Pogrel's patients gained significant improvement if microsurgery was done within 3 months of IAN overfill injuries. Delaying expeditious referral to a microsurgeon for removal of overfilled materials progressively worsens the toxic effects of the chemical injury over time. Delaying microsurgical referral risks permanent injury, owing to the time-dependent 48-hour window for complete reversal or within several weeks to greatly diminish the cytotoxic effects of overfill errors.

Microsurgical delay will close the window of opportunity to prevent permanent neurologic deficits of altered sensation, with resulting persistent chronic pain, numbness, and painful burning symptoms that extrusion overfills into the IANC can cause. Stat referral for microsurgical removal of paraformaldehyde-containing root canal filling materials and sealants is even more critically time dependent because of the greater toxicity and mummifying effects of these powders when mixed into pastes (e.g., N2 and RC2B). Risks include anaphylactic shock.[50,125,144,192]

For needed stat referrals, the referring clinician should phone, fax, and e-mail the microsurgeon. Patients lack the expertise to promptly obtain critically time-dependent referrals. When time is of the essence, the clinician and staff must assume direct responsibility to assure the stat referral to a microsurgeon is promptly appointed. Remind the patient that microsurgical delay is detrimental to corrective care.

Sealant Overfills

Neurotoxic and compressive effects of sealants are the most frequent causes of paresthesia after endodontic overfill.[129,185,262]

Additionally created injuries occur to the maxillary division of the trigeminal nerve or into the maxillary sinus membranes when sealants or bleach is extruded through maxillary teeth. Obturation and sealant materials should be used cautiously in all circumstances, especially in teeth in close proximity to the inferior alveolar nerve canal and maxillary sinus.

Use of syringes for applying endodontic scalers such as the Lentulo spiral require extra precaution when application is done in close proximity to neural structures because precise control is compromised.

Short fills contribute to apical infections and may cause temporary paresthesia. Paresthesia reversal can occur with retreatment gaining improved cleaning, shaping, and obturation to the anatomic apex.[3]

Standard of care requires that the clinician avoid unreasonable risks to prevent potentially serious or permanent harm to the patient. Treatment is negligent when a reasonably careful clinician should have foreseen and prevented unreasonable patient harm risks.

Treatment Failure

A clinician should not guarantee treatment success. It is foolish to assure the patient of a perfect result.[67] Endodontic failures may occasionally occur despite adequate endodontic care.[161,311] Nevertheless, negligent contributing factors to endodontic failures include perforation, missed or transported canal, uninstrumented portion of a root canal, bacterial infiltrates by way of a leaky coronal restoration contaminating the root canal filling,[66,293,298] overextension errors, and inadequate isolation of the tooth from salivary contaminants during instrumentation due to lack of a rubber dam.

Successful root canal treatment requires the prevention of microorganism toxins from the oral flora penetrating through the root canal system into the periapical tissues. Inadequate obturation of the root canal system is the most frequent cause of failure. Either coronal or apical leakage adversely affects success.[84,85,298]

Failing endodontics that necessitate endodontic disassembly should first be considered for a nonsurgical option because it is less invasive and therefore represents risk reduction. The clinician should first evaluate for coronal leakage, fractures, missed canals, silver point corrosion, and incomplete obturation. Root canal systems can then be recleaned, or correctly transported and then thoroughly disinfected, reshaped, and packed in three dimensions to avoid apical surgery by providing instead adequate coronal, lateral, and apical seal nonsurgically.

To avoid claims based on failed endodontics, the patient should be advised in advance of treatment of the inherent (but relatively small) risk of failure (i.e., about 5% to 10%). It may be adequate to advise the patient of the high statistical probability of success in endodontics if the clinical condition of the tooth and the clinician's past success rate warrant such representation.[145,291] Clinicians should avoid quoting the national success rate of endodontics when (1) the patient's tooth is of questionable endodontic and periodontal status and (2) the clinician is known to have an unusually high endodontic failure rate. A rate that varies markedly from national statistics suggests referral to an endodontist.

An endodontic treating clinician is also liable for failure to disclose evidence of other pathosis in the quadrant being treated. The patient should be advised of any periodontal disease that adversely affects the prognosis of abutment teeth for partial dentures, bridges, or an implant with a need for grafting if the endodontically treated tooth is lost. A clinician should also advise of cysts, fractures, or lesions of suspected neoplasms. In addition, the clinician must be careful not to ignore any evident pathosis that if untreated may adversely affect the dental or medical health of the patient. Again, a clinician who fails to plan treatment properly is planning for treatment failure.

The doctrine of informed consent protects both the clinician and the patient so that there will be no surprises or patient disappointment if a non-negligent adverse result occurs. Should such a failure or complication occur, the availability of a signed informed consent form can serve as a reminder to the patient that the risk of complications, including failure, was discussed in advance of treatment and that unfortunately the patient's endodontic treatment fell outside the usual 90% or greater root canal therapy success rate.

Full Disclosure

Disclosing a nonharmful mistake will not erode patient confidence. On the contrary, it bolsters trust by reassuring the patient that the dentist is honest and truthful. Increased trust through full transparency mitigates against legal action. Patients who trust their dentist are less likely to bring a malpractice suit. Truthful disclosures, sincere apologies, and appropriate acceptance of responsibility, including reimbursing corrective care, decreases litigation.

Disclosing is ethical and preserves integrity. "No harm, no foul" is a misguided approach. It is the patient's right to be informed of any intervention administered in the course of treatment that *could* have caused harm. On the other hand, major errors must be prudently disclosed to the patient for the sake of transparency, trust, integrity, and ethical responsibility.

Slips of the Drill

A slip of the drill, like a slip of the tongue, may be unintentional. Nevertheless, it can cause harm. When a cut tongue or lip occurs, it is usually the result of operator error. To paraquote Alexander Pope, "to err is human, to forgive, divine." To increase the likelihood that a patient will forego filing suit because of a cut lip or tongue, the clinician should follow these steps:

1. Inform the patient that the clinician regrets having injured the patient. This is not a legal admission of guilt in 29 states with "I'm sorry" protective statutes, but rather an admission that the clinician is a compassionate human being. In 2005, federal legislation to apply "I'm sorry" to all 50 states was proposed. The Senate bill sponsored by senators Clinton and Obama did not pass in Congress.
2. Repair the injured tissues or refer the patient to an oral or plastic surgeon, depending on the extent of the injury and whether a plastic revision is necessary if scarring is likely.
3. Advise the patient that the clinician will pay the bill for the referred treatment of the oral or plastic surgeon. Request the oral or plastic surgeon to send the bill directly to the clinician for payment. Send the oral or plastic surgeon's bill to the clinician's professional liability carrier. Some carriers will pay the claim under the medical payments provisions

of the general liability policy for an "accident" rather than as a malpractice incident compensable under the professional liability policy. Call the patient periodically to check on healing, recovery, and any follow-up plastic or other surgeries.

Fear Factor

U.S. Centers for Disease Control and Prevention's National Center for Health Statistics report that cost is the overriding factor preventing adults from seeking dental care. Fear is the second reason for delayed dental care. Medicaid recipients were at five times greater risk for worse dental health than those with private coverage. Adults on Medicaid were twice as likely to have not visited a dentist in more than 5 years.[319]

Dental fear may result in patients delaying or avoiding dental care.[199] Frequent cancellations and missed appointments are characteristically associated with fearful dental patients. Although it is ordinarily a defense of contributory negligence if patients do not follow a clinician's treatment recommendations, the patient's advocate may contend that the defendant clinician negligently failed to diagnose a fearful patient. Fearful patients tend to avoid dental treatment because they believe it may exacerbate a prior traumatic dental experience. Referral to clinicians who specialize in treating fearful patients should be considered to facilitate comprehensive dental treatment and to avoid future emergency endodontic care because of repeatedly canceled treatment visits.

Dental anxiety and finances are the two most important barriers to patients obtaining regular dental care. Fearful dental patients avoid necessary treatment, delay recalls, and are reluctant to undergo painful procedures. Therefore, it is essential that such patients be identified for proper management or referral for fear-reduction therapy. A patient who experiences intense anxiety in the dental chair, together with a history of avoiding dental care, suggests a diagnosis of dental phobia. Fearful dental patients fear loss of control during clinical treatment and require reassurance and reaffirmation that they have the power to halt the procedure by raising a hand or using another appropriate gesture. After trust is gained, additional procedures may be performed.

In addition to desensitizing techniques, the use of proven topical-anesthesia delivery systems helps to ensure a relatively painless injection of local anesthetics. Topical-anesthetic patches and oral-anesthetic rinses may prove a valuable aid for pain management of the fearful patient.

The use of psychological questionnaires, such as the Dental Anxiety Scale or the Modified Dental Anxiety Scale, may help to identify such individuals.[154] These simple questionnaires are short, quick, and easy to complete, and users are provided with cutoff scores that help the clinician identify patients who have psychological special needs. In this way, the clinician will be in a position to assist the dentally anxious or dentally phobic patient in accessing dental health care.

Leakage

Long-term seal of the root canal system is determined apically by the sealer and coronally by the final restoration.[36,134,228,293] Root canal–filled teeth should be permanently restored without undue delay to prevent leakage contamination of the previously obturated canal system, because varying canal shapes from round to oval prevent a 100% seal.[84,85,202] Bonded seals covering the canal surfaces should be used to control any leakage after compaction until a permanent restoration is cemented.[305] Besides an adequate coronal sealing, an adequate apical sealer should be well adhered to the canal walls.

The endodontic goal is to prevent bacterial contamination of the periradicular tissue by predictably providing adequately cleaned, shaped, and filled root canal systems. Any residual bacteria should be entombed in the root canal filling. A bacteria-tight apical seal should be designed to last long term with sealed portals to prevent reentry of microorganisms, which cause reentry recontamination and lead to endodontic failure.[84,298] Younger patients are more susceptible to bacterial penetration inside dentin tubules and thus recurring infections.[177]

Electrosurgery

Electrosurgery can cause problems if mishandled. Damage to the oral cavity caused by improper use of electrosurgical devices consists primarily of gingival necrosis, osteonecrosis, sloughing adjacent to the surgical field, and pulpal necrosis of affected teeth.

All equipment should be properly maintained and certified to meet the American National Standard (ADA specification no. 44 on electrical safety standards). Current equipment should be checked to see that units meet these standards and that electrical cords and other components are in good repair. Electrical receptacles should meet the requirements of the National Electrical Code for circuit grounding and ground fault protection. During use, the dispersive electrode plate should be well away from metal parts of the dental chair and the patient's clothing, because skin contact can cause burns. Use of plastic mirrors, saliva ejectors, and evacuator tips is strongly recommended.

Implant Risk Factors

Patient risk factors such as grinding teeth or diabetes increase the odds of implant failure.[166] Higher implant failure rates occur with surgeons who have less than 5 years of implant experience.[166]

Fluoride Varnish for Caries Prevention

In 2006, the ADA Council on Scientific Affairs published evidence-based research with fluoride recommendations for caries prevention. The ADA recommends that fluoride varnish treatments for at-risk patients should be done between two to four times annually, depending on risk classification.

If fluoride trays are used, trays are effective only when used for 4 minutes. One-minute foam or gel fluoride treatments are ineffective.

One of many benefits of fluoride varnish is very little systemic exposure, which does not require daily patient compliance compared to fluoride trays. Fluoride varnish containing 5% sodium fluoride with 22,600 ppm fluoride ions help occlude dentin tubules and aid remineralization.

Xerostomia and Diet

A common side effect of cancer treatment is diminished saliva production, which increases the risk of dental demineralization, caries, and candidiasis. Milk may help because it moisturizes, lubricates, and buffers acids while contributing to enamel remineralization. Demineralization aggravated by a chronic lack of saliva may result in osteoradionecrosis. Therefore, diseased teeth should be treated prior to cancer therapy.

DNA preserved in calcified bacteria on the teeth of ancient human skeletons has shed light on the health consequences of the evolving diet and behavior from the Stone Age to the present.[3] Composition of oral bacteria changed markedly with the introduction of farming and again around 150 years ago. With the introduction of processed sugar and flour in the Industrial Revolution, the diversity in oral bacteria dramatically decreased, allowing domination by caries-causing strains. As a result, the modern mouth basically exists in a permanent disease state.

Reasonable versus Unreasonable Errors in Judgment

Although a clinician is legally responsible for unreasonable errors in judgment, mistakes occasionally happen despite adherence to the standards of reasonable care. A mistake does not prove malpractice unless the mistake is caused by a malpractice error or omission.[141] For example, accessory or fourth canals on molar teeth are frequently difficult to locate and may tax the best clinicians. Failure to locate an accessory or fourth canal does not conclusively constitute an unreasonable error of judgment. Rather, this may represent a reasonable error of judgment in the performance of endodontics. Nevertheless, if the additional canal could have been diagnosed radiographically with diagnostic quality radiographs at different angles, the existence of a fourth canal should have been considered. Instrument and seal the extra canal for successful obturation.

Incorrect Tooth Treatment

A reasonable, non-negligent mistake in judgment may occur because the clinician has difficulty localizing the source of endodontic pain. Vital pulps may on occasion be sacrificed in an attempt to diagnose the pain source, but it is unreasonable and therefore inexcusable to treat the wrong tooth if it is inadequately tested with pulp tests, misidentified on the referral slip, or if radiographs are mounted or read incorrectly. Also, treating large numbers of teeth endodontically (e.g., an entire quadrant) when attempting to localize chronic pain suggests pain is probably not of pulpal origin, and other differential diagnoses should be ruled out including atypical facial pain, referred pain, or TMD pain.

If the wrong tooth is treated because of an unreasonable mistake in judgment, the clinician should be compassionate, waive payment for all endodontic treatment, and offer to pay the fee for crowning the unnecessarily treated tooth.

Post Retrieval

Ultrasonic instruments will vibrate and loosen the cement around posts.[68] The clinician can avoid overheating the post by proceeding slowly with a water coolant, periodically resting for cool down, and checking the post temperature periodically to be reasonably certain overheating is not occurring.[96,130,230] Figure 29-21 demonstrates what happened when an endodontist negligently overheated the post during attempted removal, causing tissue necrosis, bone loss, need for augmentation procedures, irreversible pulpitis in an adjacent tooth, and the loss of two teeth.[252]

Broken Files

Leaving broken files behind without referring the patient to an endodontist for attempted microscopic retrieval and not advising the patient of leakage potential may constitute fraudulent concealment (Figs. 29-29 and 29-30). Patients should be informed of such mishaps for (1) referral consultation or treatment; (2) advising the patient, who on his or her own may seek a second opinion; or (3) disclosure, so as to return if a flare-up occurs.

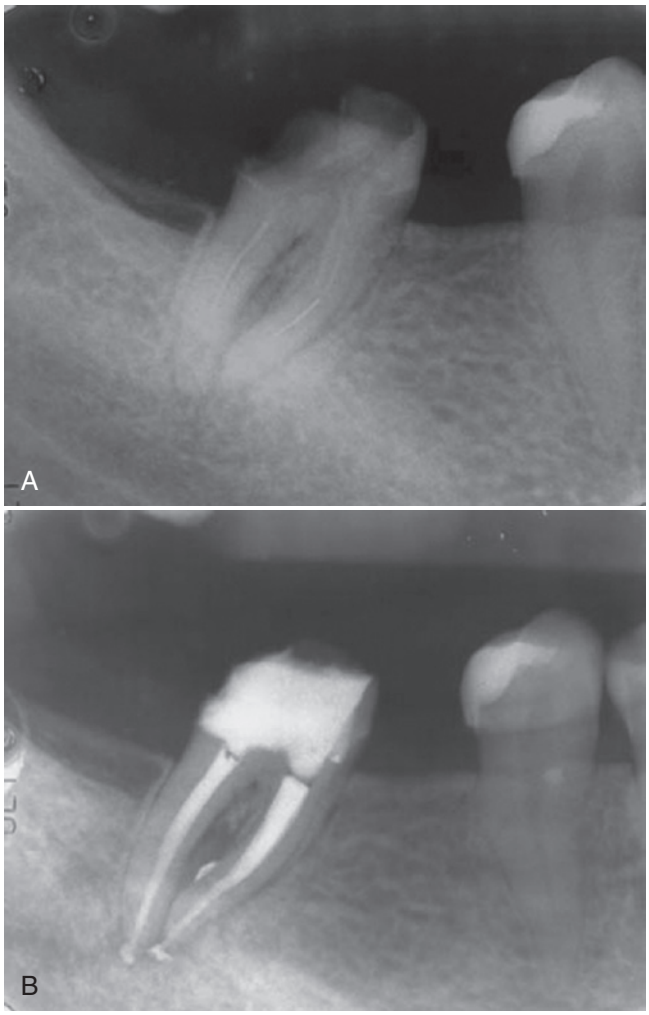


FIG. 29-29 A, Tooth #31 with three separated files, one in each canal. K-files used for cleaning canals and also obturation (1996). B, No apparent radiographic changes in 1998.

Swallowing or Aspirating an Endodontic Instrument

Use of a rubber dam in endodontics is mandatory.[14,294] Even if the endodontically treated tooth is broken down and cannot be clamped, a rubber dam, regardless of required modification, should be used in all instances (see Chapter 5). Not only is microbial contamination reduced with the use of a rubber dam, but the risk of a patient's aspirating or swallowing an endodontic instrument is eliminated (Fig. 29-31). Accordingly, if a patient swallows or aspirates a file, it is likely because of the clinician's failure to observe the standard of care. If a swallowing or inhalation incident does occur, the clinician should do the following:

1. Advise the patient that the clinician regrets and is sorry about what occurred.

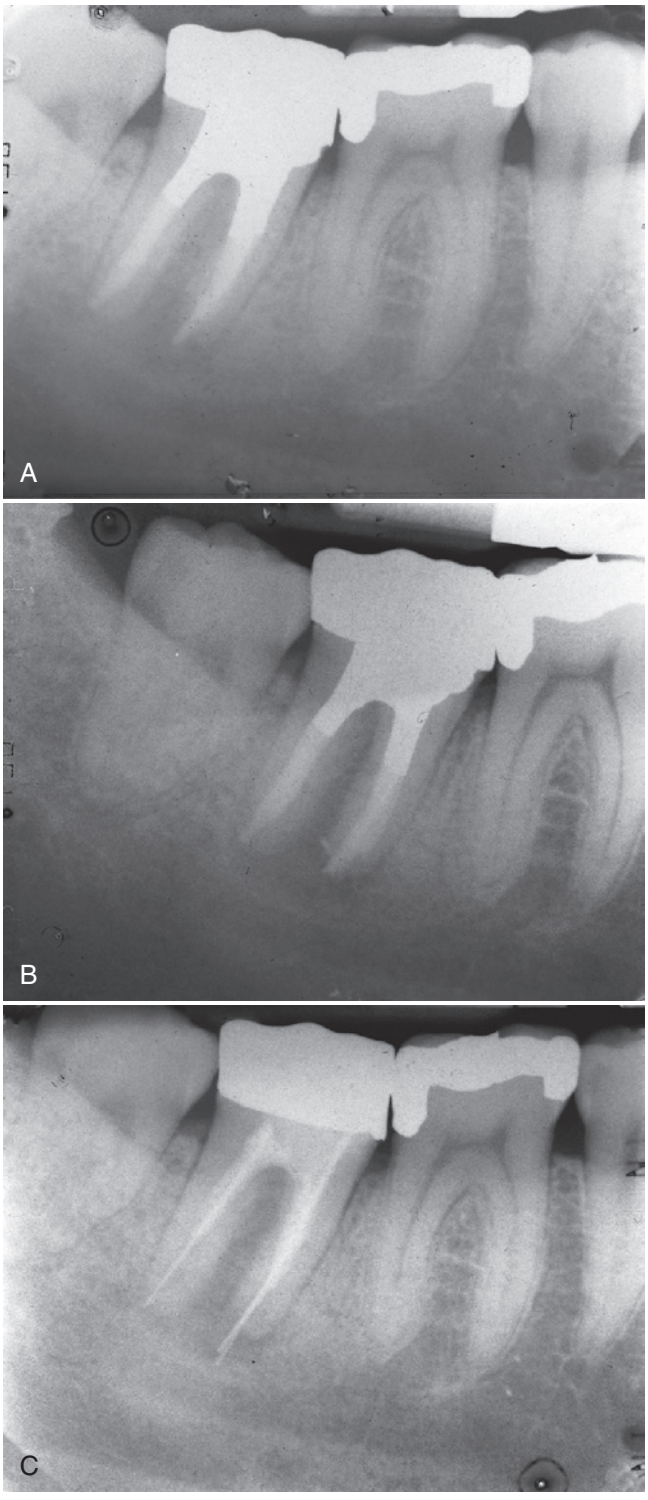


FIG. 29-30 A, Apical radiolucent changes with draining 9-mm buccal abscess (August 2003). B, Endodontist retreatment (August 2003). C, Successful retreatment with no buccal pocket (September 2003). (Courtesy James YS Ho, DMD.)

2. Refer the patient for immediate medical care, including radiographic imaging, to determine if the instrument is lodged in the bronchus, esophagus, or stomach so that appropriate medical measures are taken promptly to remove it.
3. Offer to pay for the patient's out-of-pocket medical expenses and wage loss. Some professional liability policies will cover the incident as "medical payment" for an accident and not as a malpractice claim.

FIG. 29-31 Swallowed endodontic instrument demonstrates the wisdom of using a rubber dam.

Overextensions and Overfills

A common error in utilizing periapical or Panorex imaging is presuming that when only one cortical border of the IANC is visible, the sole visible border is the superior border. Instead, virtually always when only one cortical border is clearly visible, it is the inferior cortical border.[206] (See Fig. 29-30, *B*, periapical, which may appear to show only one cortical border, and Fig. 29-30, *C*, a clearer CBCT image of the same area showing both cortical borders.)

Misassumption of the IANC borders results in mismeasurement of the IANC location. Because the IANC is approximately 3 mm in diameter, misassuming the sole observable cortical canal border to be the superior border (rather than the inferior border) can result in an imprudent treatment decision to delay microsurgery if overfill occurs and belated CBCT confirms an overfill inside the IANC. In Fig. 29-30, *B*, the defendant endodontist misdiagnosed the overfill as being superior to the IANC rather than inside the IAN canal. Resultant permanent paresthesia occurred from watching and waiting for sensory return rather than referring to a microsurgeon for a stat microsurgical removal.

A slight overextension of root canal filling with conventional obturation or sealants can occur without violating the standard of care if not in close proximity to the IANC or sinus (see Chapters 2 and 29). Gross overfill usually indicates faulty technique. Fig. 29-32, *A* and *B*, illustrates gross overfill of calcium hydroxide, causing permanent paresthesia. Nevertheless, so long as the overextension is not in contact with vital structures such as the IANC or sinuses, permanent harm is unlikely. An exception is paraformaldehyde-containing

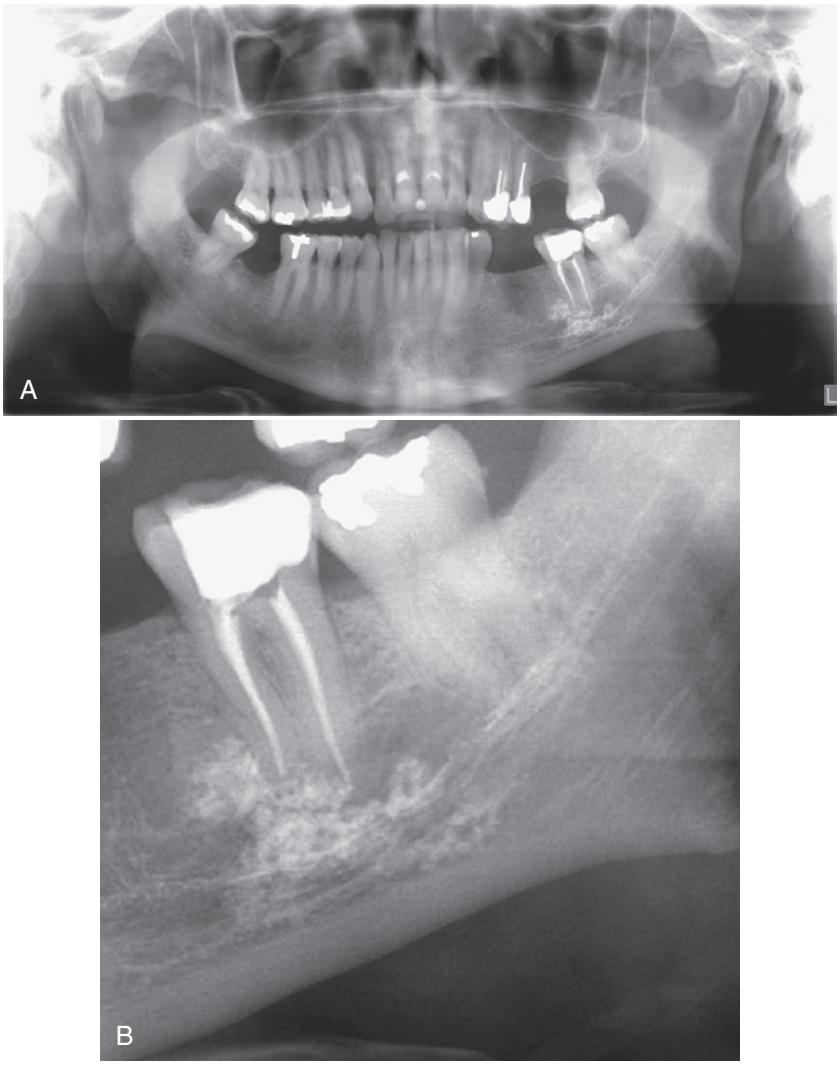


FIG. 29-32 A-B, Calcium hydroxide overfill, causing permanent paresthesia.

sealants, which cause a neurotoxic chemical burn type of injury that chemically diffuses through bone and soft tissue spaces.

If, however, postoperative pain is foreseeable as a result of overextension, the patient should be advised with postoperative written instructions of the likelihood of postoperative discomfort or paresthesia because of suspected contact of the sealant material with surrounding vital structures. A note should be made on the patient's chart of the overextension should symptoms later manifest. The clinician should observe with close follow-up visits and patient phone calls that evening, the next morning, and the following 3 days to rule out postoperative persistent numbness or dysesthesia pain resulting from any overfill. Slight overextensions with inert conventional endodontic sealers, such as gutta-percha with Grossman's sealant, can repair themselves and produce no irreversible changes if done without direct contact with the sinus or IANC or indirectly through chemical leakage and seepage beyond the initial area of operation.

Overextending the root canal filling material risks permanent consequences if the underlying IAN is initially "harpooned" with files. A portal of entry into the IANC results from overinstrumentation penetration. Flexible gutta-percha alone probably will not penetrate beyond the mature root into the IANC or other vital structures without prior excessive instrument perforation into the IANC.

Mental foramen distances can be overestimated on the panoramic radiograph. Panoramic radiography may show more deviation (+0.6 mm) from the perioperative measurement.[110,289] Paraformaldehyde-containing sealants can create cytotoxic chemical destruction of the IAN if placed in close proximity to, even though not directly contacting, the underlying IAN (see reference, Sargenti Opposition Society [SOS]).[278] On the other hand, conventional obturators and sealants usually require coming closer to contact with the IAN before resulting permanent anesthesia, paresthesia, or dysesthesia occurs (Fig. 29-33). Consequently, the incidence of permanent sequelae with conventional filling materials is lower than the greater cytotoxic potential with paraformaldehyde-containing sealants.[234] Because of the higher risks associated with paraformaldehyde-containing endodontic materials, use of N2, RC2B, or similar toxic pastes is contraindicated and violates the standard of care.[11] Permanent injury risk is substantially less likely with traditional eugenol-containing filling materials, less toxic chemicals than paraformaldehyde, which destroys and mummifies nerve tissues. When safer, less risky alternative

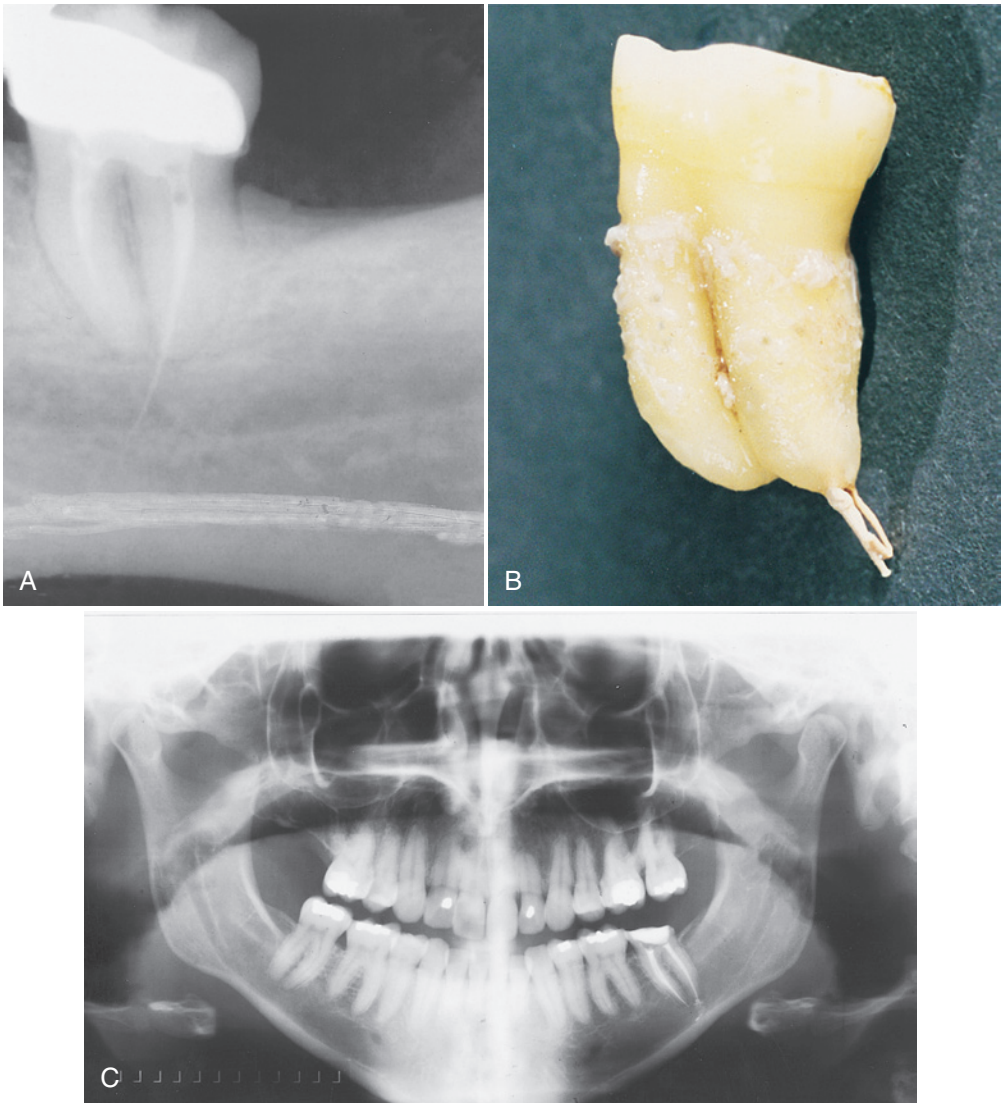


FIG. 29-33 A-C, Overextended gutta-percha and extracted tooth with gutta-percha intact.

effective therapy exists, it is unreasonable (and substandard) to elect an unsafe alternative methodology. Also, the doctrine of informed consent is highly relevant because it is contrary to public policy to request a patient to assume inherently dangerous treatment risks that are reasonably avoidable with safer and more predictable methodologies. When the risk exceeds the benefits, such risks are regarded as negligent and thus should be avoided and not undertaken.

Any significant overextension should be considered for immediate retreatment by attempted retrieval of overextended gutta-percha and sealant.[135] The clinician also has the option of immediately referring the patient to an endodontist for retrieval before the sealant sets. Conventional filling agents such as gutta-percha do not penetrate the cortical walls of the IANC unless probably preceded by prior penetration with overinstrumentation. This principle can apply to sinus perforation. If despite local anesthesia, the patient feels an electric shock during mandibular molar or premolar instrumentation, this may be a warning sign that the IAN was pierced with endodontic files. If this occurs, the root canal should not be filled; instead, periapical radiographs at different angles should be exposed with instruments in place to confirm whether any IAN penetration resulted. Removal of gutta-percha and sealants that have entered the IANC should be attempted as soon as possible (preferably no later than within the first 24 hours).[254] The eugenol component of the sealant causes an inflammatory reaction in a constricted space, which is best relieved by retrieval. If retrieval fails, a decortication microsurgical procedure with an oral surgeon is indicated at the earliest possible time (preferably within the first 24 hours) owing not only to eugenol chemical toxicity but also eugenol reaction with gutta-percha, which causes expansion and thus a mechanical compression ischemic injury.[219,254]

Compartment Syndrome

Inflammatory edema that compresses and compromises blood supply to soft tissues and nerves in limited spaces with resulting ischemia is termed *compartment syndrome*.[285,339] Compartment syndromes are a group of conditions that result from increased pressure within a limited anatomic space, acutely compromising the circulation and ultimately threatening the function of the tissue within that space. Compartment syndrome occurs from an elevation of the interstitial pressure in a closed osseofascial compartment that results in microvascular compromise.

The pathophysiology of compartment syndrome is an insult to normal local tissue homeostasis that results in increased tissue pressure, decreased capillary blood flow, and local tissue necrosis caused by oxygen deprivation. Compartment syndrome is caused by localized hemorrhage or postischemic swelling. The pathophysiology of compartment syndrome is a consequence of closure of small vessels. Increased compartment pressure increases the pressure on the walls of arterioles within the compartment. Increased local pressure also occludes small veins, resulting in venous hypertension within the compartment. The arteriovenous gradient in the region of the pressurized tissue becomes insufficient for tissue perfusion.

The clinician should have a high index of suspicion whenever a nerve is located inside a closed bony compartment where nerve injury within the surrounding neurovascular bundle has the potential for bleeding or swelling. Compartment syndromes are characterized by pain beyond what should be experienced from the initial injury. Also, diminished sensation may be noted in the distribution of the nerve within a compartment that is being compressed, such as the IAN, which is enclosed by bone on all sides.

Root canal followed by root resection without an adequate retrograde restoration will likely fail. Open drainage is no longer recommended. Dissect out subapical granulation tissue cleanly and without damage to vital structures. Caution should be taken when granulation tissues are attached to vital anatomic structures such as neurovascular bundles or sinus linings. A potential for recontamination exists unless the root apex is removed. Apical resection of 3 mm will remove potential problems. This resection will additionally permit inspection. A straight fissure bur design with appropriate air venting is recommended rather than a conventional air rotor to prevent air emphysema.

Currently preferred methodology utilizes specifically designed ultrasonic tips inserted into a piezoelectric ultrasonic handpiece. Amalgam is no longer recommended as a material for root-end filling. MTA and newer silicate trioxide materials are recommended.[243]

Periradicular Surgery (PRS)

Accessing mandibular posterior teeth can generate increased heat during removal of significant quantities of bone to gain apical access. Thus, copious coolants must be applied to avoid risking injury to adjacent structures. Molar teeth surgical access should avoid the facial artery as it crosses the border of the mandible adjacent to the first molar.

Avoiding the Inferior Alveolar Nerve Canal

When placing implants or performing apicoectomies in close proximity to the mental foramen in the bicuspid region, the clinician should consider the anterior loop of the IAN.[277] The IAN ascends as it approaches the mental foramen, compared with the IAN's more inferior location in the molar region. The IANC varies from buccal to lingual as it courses the length of the mandible. Thus, CBCT imaging aids in assessing the IANC location for planning implant placement or apical surgery[75,97] (Fig.29-34, *A* to *C*[138]; this Los Angeles case settled for $915,000, with permanent dysesthesia).

Figure 29-35 illustrates avoidance of the IANC to demonstrate that IANC penetration with the implant or drill is a preventable adverse event.

Mental Nerve Injuries

The mental nerve, as the terminal branch of the IAN, is at risk during elevation of the mandibular mucoperiosteum. Careful flap design and elevation are important to avoid mental nerve injury while working on the buccal surface of the mandible in the region of the mental foramen.[207] In performing incision and drainage in the region of the mental foramen, a diagnostically accurate radiograph is necessary to avoid incising through the course of the mental nerve.[84,180]

When carrying out periradicular surgery (PRS) in the mandibule, consideration must be given to the path of the inferior dental nerve bundle in the mandibular canal, which lies in close proximity to the root apices.[277] Be aware of the position of the mental nerve exiting through the mental foramen. A shallow sulcus, coupled with a prominent mandibular protuberance, together with the lingual inclination of the roots may make access to the root apices difficult. Approximately 40% of lower incisors have two root canals, which may preclude adequate resection and retrograde filling of the root apex, which may ultimately lead to treatment failure.

Local Anesthesia: Septocaine

Septocaine and Articaine are not suitable for an inferior dental nerve block due to the potential increased risk of paresthesia and nerve damage.[91]

Implant versus Endodontics

A natural tooth with its periodontal support, function, and proprioception is superior to an implant-retained restoration. Whenever reasonably feasible, teeth should be retained.

Reasonable clinicians may differ in recommending extraction versus periradicular surgery depending on location near vital structures and clinical conditions. Extraction of the diseased tooth and placement of a dental implant has a better long-term prognosis and thus should be the treatment of choice is one author's conclusion.[275] Conversely, every reasonable effort should be made to retain the natural tooth with its periodontal support, function, and proprioception. Thus, an implant-retained restoration should be considered as the last possible treatment option as concluded by other clinicians.[24,73,310]

Infraocclusion of the implant restoration in teenage patients with residual craniofacial growth occurs as the adjacent teeth erupt. Therefore, delay implant placement until dental and skeletal maturation occurs.[309]

Aesthetics is an important outcome for patients receiving implant-supported restorations. To achieve this outcome, patients require good bone volume and quality, as well as soft-tissue height. Following extraction, the alveolar bone resorbs vertically, as well as horizontally (buccolingually). Socket preservation intervention grafting may aid in reducing the bony dimensional changes after tooth extraction but ridge resorption is not completely prevented. Intervention techniques including autografts, allografts, xenografts, guided bone regeneration (GBR) and growth factors accomplish varying degrees of success in maintaining the anatomic dimensions of the alveolus before implantation.[54]

Current Use of Silver Points

Based on what has been known for more than 3 decades, use of silver points in lieu of gutta-percha or other conventional endodontic filling materials represents a departure from the

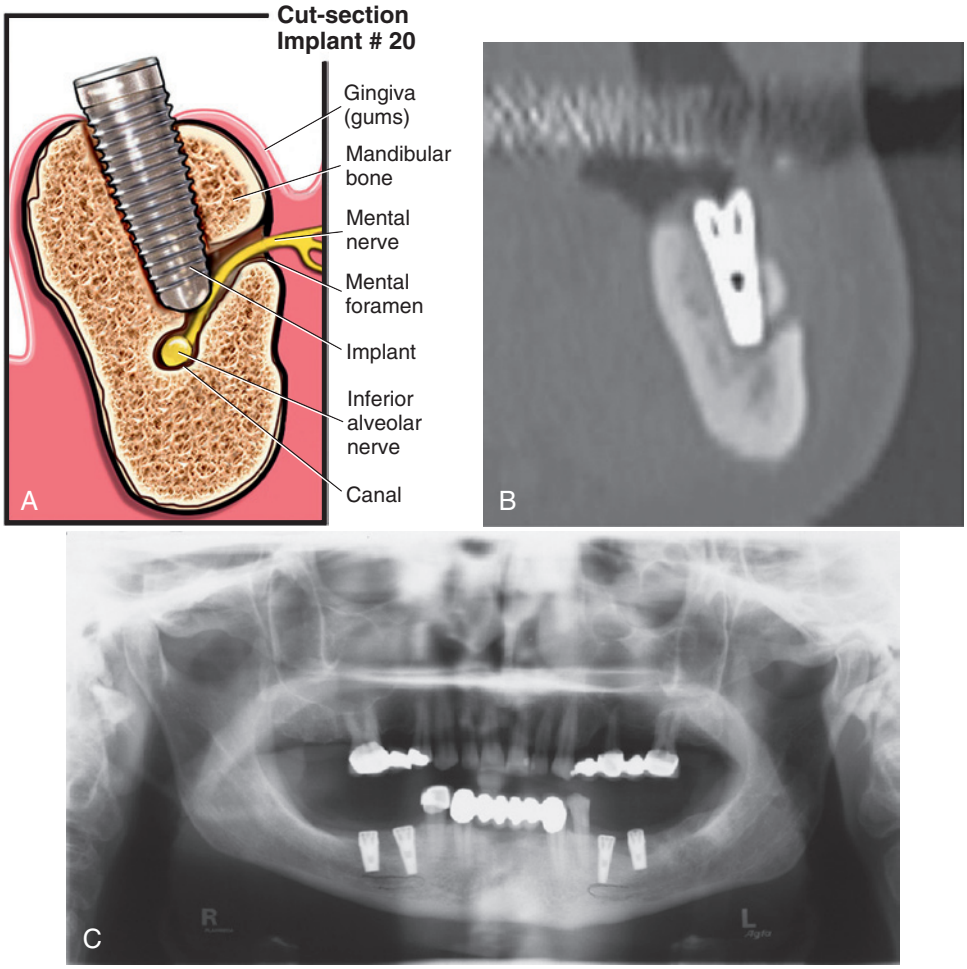


FIG. 29-34 A, Illustration showing implant placed into ascending inferior alveolar nerve canal (IANC). B, Cone-beam computed tomography (CBCT) image of implant into ascending IANC. Settlement of $915,000. C, Implant into IANC; immediate postoperative Panorex image. Same patient but with CBCT image.

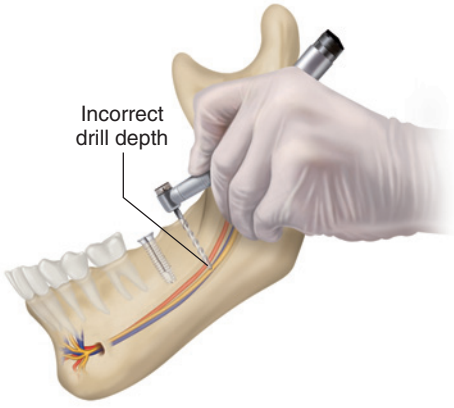
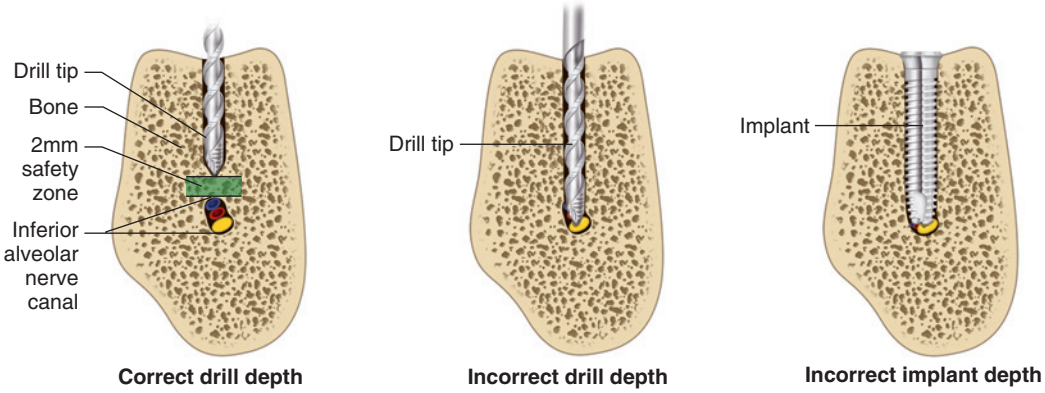


FIG. 29-35 Medicolegal implant image.



current standard of care.[13] This is because silver points corrode in time, and a tight 3D apical seal is lost. Figure 29-36, *A* to *C*, represents gross overextension with a silver point that ultimately caused the loss of tooth #14 as a result of endodontic failure.

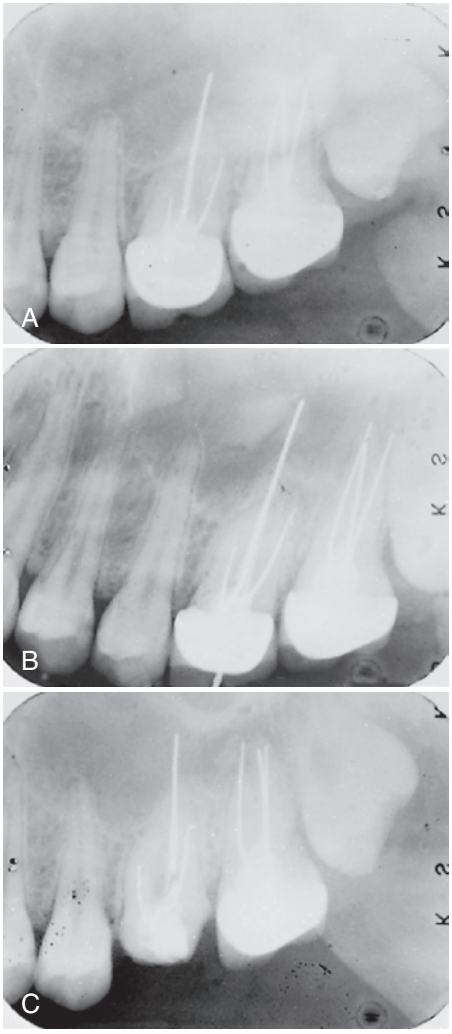


FIG. 29-36 Gross overfill into sinus with a silver point, which ultimately caused sinusitis and loss of tooth #14 as a result of endodontic failure.

The AAE Position Statement does not recommend the prophylactic revision of silver point obturation, unless there is clear evidence of endodontic pathosis or if the silver points complicate proper restoration of the tooth.[13]

N2 (Sargenti Paste)

Dental literature reports that permanent paresthesias are associated with gross overfilling with paraformaldehyde sealant (N2) (Fig. 29-37).[200,234] Current use of paraformaldehyde-containing endodontic sealants is not merely the result of a philosophic difference between two respectable schools of thought. Rather, the distinction is between the reasonable and prudent school of thought that advocates conservative conventional endodontics and the imprudent and radical school of paraformaldehyde providers who unreasonably risk permanent, deleterious injury with N2 overextensions. Regardless of the small number of clinicians professing to use N2, it is cytotoxically unsafe and should be avoided. A customary negligent practice by some clinicians is no defense to safe and prudent practice, which the standard of care requires. No matter how few or how many do it wrong, this never makes it right.

The AAE recommends against the use of paraformaldehyde-containing materials as they have proved to be both unsafe and ineffective. Accordingly, the use of such materials is below the standard of care for endodontic treatment.[11]

Four years after her settlement, a Sargenti paste overfill patient wrote about her persistent dysesthesia as follows:

> I have been in a pain cycle that is almost unbearable. I fully understand why the closest God given disease to what I suffer, trigeminal neuralgia, is nicknamed the suicide disease. There are days where that option enters my mind but I believe that suicide is a sin and will land me in an eternal life worse than what I was trying to escape.

Clinicians may be liable for fraudulent concealment, intentional misrepresentation, or co-conspiracy if they discovered that a previous clinician's negligence is the cause of dental disease and both the prior clinician and subsequent treater concealed the prior clinician's negligence. For instance, if a gross overextension of a paraformaldehyde packing or sealant is evident radiographically and the patient reports that another

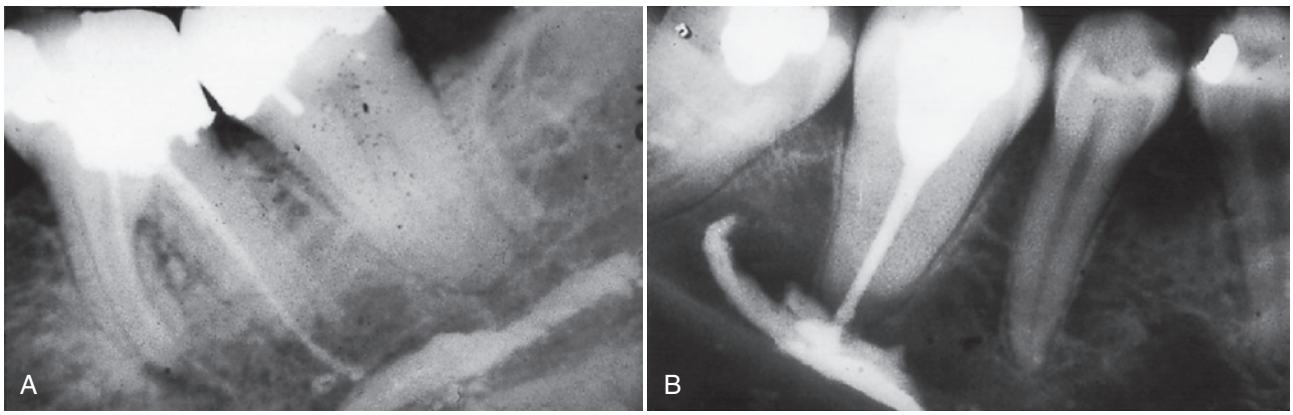


FIG. 29-37 **A and B,** Overextensions of Sargenti paste filling the inferior alveolar nerve canal. Both cases could have been avoided if the practitioners had selected a conventional sealing material and used a technique that emphasizes length control.

clinician caused the overfill (that resulted in permanent lip and chin anesthesia), subsequent treating clinicians may be liable for fraudulent concealment if they misinform the patient that the anesthesia will probably disappear shortly and that using N2 merely reflects a philosophic difference rather than substandard practice. Some clinicians who differ on therapies regard such differences as controversial. The difference between standard of care and substandard practice is not controversial but rather an indisputable difference between right and wrong. For instance, quackery based on pseudoscience is not controversial. Instead it is fraudulent practice.[37,224] Likewise, if the radiographs indicate sealant is inside the IANC and the patient complains of persistent anesthesia, the patient should not be told to wait for return of sensation. The clinician should refer the patient immediately for microsurgical consultation for decortication and decompression surgery.[263]

The Federal Food, Drug, and Cosmetic Act prohibits interstate shipment of an unapproved drug or individual components used to compound the drug.[100] On February 12, 1993, the FDA dental advisory panel confirmed that N2's safety and effectiveness remain unproven. N2 may not be shipped interstate or distributed intrastate if any of the N2 ingredients were acquired interstate. Mail-order shipments of N2 from out-of-state pharmacies in quantities greater than for single-patient use are considered a bulk sales order rather than a prescription, thus violating FDA regulations.[63] A San Francisco jury awarded punitive damages against an N2-distributing New York pharmacy for knowingly shipping N2 in violation of FDA regulations, done with deliberate disregard for patient safety.[162]

Defective Restorations

Marginal gaps greater than 50 µm lead to cement dissolution and cause 10% of crown failures within 7 years after cementation. Dull or worn explorers substantially increase the likelihood of nondetection of open margins. A sharp explorer can detect margin defects as small as a 35-µm opening.[36] Accordingly, a sharp clinician should utilize a sharp explorer to detect open margins. Open crown margins contribute to endodontic failure and should be avoided (see Chapter 22).

Restoration Marginal Integrity

A pathologic apical lesion will resolve when the origin of infection is eradicated, the root canal system effectively sealed, and a satisfactory coronal restoration placed. Many clinical studies report a high success rate for nonsurgical root canal treatment (NSRCT) of greater than 90%.

PROPHYLACTIC ENDODONTIC PRACTICE

Malpractice Prophylaxis

Most negligently injured patients do not sue.[51] Nonetheless, litigation serves a prophylactic purpose because litigation fears make some clinicians more careful and help promote professional guidelines. For example, after the American Society of Anesthesiologists adopted practice guidelines to reduce patient harm, deaths and professional liability premiums decreased dramatically.[252,283]

One-Visit versus Two-Visit Endodontics

One visit endodontics, if done well, will allow disinfection of the canals and obturation without risk of recontamination through the coronal seal between visits. Treatment of apical periodontitis with signs or symptoms of a beginning lesion is determined by the sound judgment choice of the clinician. Either one or two visits will comply with the endodontic standard of care.[242]

Prevention of Orthopedic Joint Infections

Available evidence is insufficient to recommend prophylactic antibiotics for dental procedures in persons with joint replacement, according to the American Academy of Orthopedic Surgeons (AAOS) and the American Dental Association (ADA) clinical practice guidelines.

This new guideline, titled "Prevention of Orthopedic Implant Infection in Patients Undergoing Dental Procedures," replaces the previous AAOS Information Statement, "Antibiotic Prophylaxis for Bacteremia in Patients with Joint Replacement."[8,21]

A collaborative systematic review of the correlation between dental procedures and prosthetic joint infection (PJI) found no direct evidence of a causal link. The new clinical practice guidelines were developed using the published AAOS development process, which meets or exceeds recommended Institute of Medicine standards for the development of systematic reviews and clinical practice guidelines.

Research showed that invasive dental procedures, with or without antibiotics, did not increase the odds of developing a prosthetic joint infection.

Clinical practice guidelines include three specific recommendations:

1. Practitioners should consider changing their customary practice of prescribing prophylactic antibiotics to patients with prosthetic joints undergoing dental procedures. This recommendation is based on limited evidence that dental procedures are unrelated to PJI.
2. There is no direct evidence that the use of oral topical antimicrobials before dental procedures will prevent PJI. This is an inconclusive recommendation. The guidelines' authors were unable to recommend for or against the use of topical oral antimicrobials in patients with prosthetic joint implants or other orthopedic implants
3. Good oral hygiene should be maintained. This is the only consensus recommendation in the new guideline.

Potential adverse effect of routine prophylactic antibiotic use may include increased bacterial resistance, allergic reactions, diarrhea, and death.

Immunocompromised patients may be at greater risk for implant infections. Antibiotic use might be considered in this group.

Immunocompromised patients include patients with diabetes mellitus type 1 and type 2, autoimmune diseases, post–organ transplants, chemotherapy, bone marrow transplants, HIV positive status, chronic steroid use, obesity, hemophilia, malnutrition, tobacco or alcohol use, leukemia or other cancers, radiation therapy history, immunomodulated therapy, or the elderly.

Disclosure Errors

Should dental negligence harm a patient, then full disclosure, including accepting the clinician's responsibility along with providing an apology and explanation, results in the best

outcome for the clinician and patient. Also, assurance of efforts to prevent a recurrence results in greater patient satisfaction and retains trust with the disclosing clinician to sustain a continuing clinician-patient relationship. Patients want to be told of treatment errors, regardless of whether the error can be corrected. One study in which there was full disclosure of negligently caused therapeutic errors resulted in only one patient thereafter seeking legal advice.[210] In the same study, patients paradoxically acknowledged, "It is realistic to expect that doctors will make errors. … Patients have a right to expect that their doctors will not make errors." Nonetheless, virtually all patients concluded, "Patients should be able to trust their doctors to give them the right care." As noted earlier, apologetic expressions of sorrow or empathy for a patient's injuries are barred in evidence from being construed as admissions of fault in 29 states.[182] Protecting the patient's best interest includes telling the patient when negligent errors occur.

Future of Antibiotics and Resistance

Clinical signs, symptoms, and outcomes of infection result as much from the host response to the microbe as from the direct effects of the microbe itself. The speed with which antibiotic resistance spreads is driven by microbial exposure to all antibiotics, whether appropriately prescribed or not. Even if all appropriate antibiotic use were eliminated, antibiotic-resistant infections would still occur (albeit at lower frequency). Widespread antibiotic resistance was recently discovered among bacteria found in underground caves that had been geologically isolated from the surface of the planet for 4 million years.[45] Resistance was found even to synthetic antibiotics that did not exist on earth until the 20th century. To slow the spread of resistance in order to prolong the useful lives of currently available antibiotics, careful clinicians should prevent infections from occurring in the first place.

In 2009, more than 3 million kilograms of antibiotics were administered to human patients in the United States alone. In 2010, a staggering 13 million kilograms were administered to animals, the majority to promote the growth of livestock.[297]

Temporomandibular Disorders

Occlusal prematurities can trigger temporomandibular disorders (TMDs) in patients with a prior TMD history, so during or following endodontic therapy for such a patient, it is essential that temporary and final restorations not open the bite or significantly alter existing occlusion.[194] Hyperocclusion is a potential TMD trigger that can result from open bite or on a contralateral side, when a terminal abutment tooth is taken out of occlusion.

Patient Rapport

Good patient relations are 15% dependent on the clinician's competency to cure and 85% dependent on the clinician's ability to assure the patient that the treatment being given will be done with the clinician's best judgment and care.

Rapport between clinician and patient reduces the likelihood the patient will sue, despite an adverse result. The clinician can develop rapport by demonstrating genuine interest in the patient and making the patient feel valued. Patients feel important if they are seated in the operatory within a reasonable time after arriving. The longer a patient is kept waiting, the more frustration and animosity build. If the patient cannot be seen within a reasonable time, a staff member should communicate the reason and, if appropriate, offer to reschedule the appointment. Staff or the clinician should telephone the patient at the end of the day after any difficult procedure or surgery to check on the patient's status and remind the patient to follow postoperative instructions. The clinician should record any patient complaints, symptoms, and noncompliance with instructions. The latter can be used as evidence of patient contributory negligence if litigation occurs.

The clinician should remember that the patient lacks the information and expertise to evaluate quality and performance. Patients have their own experiences and perceptions and often rely on lay information when gathering facts about dental procedures and treatment options. However, clinicians should expect some patients (especially those with access to the Internet) to ask technical questions and expect sophisticated responses.[199,246]

Rapport Building Blocks

Sir William Osler advised, "Listen to the patient. He is trying to tell you what's wrong with him." The best communicators listen more than they speak. When they do speak, it is mostly to clarify what the patient has said. Difficult clinician-patient relationships create poor communications. Improved understanding of the patient's complaints fosters better rapport, aids treatment, and reduces the likelihood of litigation.

In discussing the patient's complaints, the clinician should ask, "What do you think is causing the problem?" Otherwise, the clinician may solve the patient's dental problem while failing to solve the patient's perceived problem. Failing to clarify the patient's expectations about diagnoses and recommended treatment leaves the patient with unresolved worries and concerns. For instance, a patient may fear that a retained endodontic file is carcinogenic unless this fear is allayed with a careful explanation that it is not.

Unhurried Visits

Paying full attention to the patient's complaints, making good eye contact, and respectful addressing of the patient will gain rapport, improve communication, and prevent lawsuits. The clinician should avoid questions that require a yes-or-no answer and instead ask what the patient perceives as the problem, rephrase the patient's complaints to prevent miscommunication, and ask if the patient's complaints have been summarized accurately. Summarizing clarifies understanding by repeating important points. The clinician should also ask whether there are any remaining questions. Nonverbal communication is a powerful tool. The clinician should shake hands initially and comfort with an outstretched hand if pain is provoked.

Emotions are the dominant force behind most malpractice claims. Patients who feel misled, betrayed, or abandoned become angry and may seek vindication instead of simply seeking financial compensation. Thus, the clinician should maintain a tactful and courteous approach and be attentive to the patient's needs and complaints. In addition, the clinician should always make sure that communications with the patient are clear, even to the point of being repetitious, by asking if the patient has any questions. The patient should never be abandoned in the middle of a course of treatment. Also, the clinician should always be available to provide

follow-up care. Clinicians should avoid making telephone diagnoses. Instead, recommend an office exam to assess treatment needs.

Good telephone communication is a matter of asking the right questions, such as asking a patient complaining of postoperative swelling if there is difficulty breathing or swallowing, as well as the degree and location of swelling. In cases of suspected infection, clinicians should ask the patient or family member to call back with a temperature reading to verify the patient is afebrile.

Off-the-Cuff Diagnoses

One clinician misdiagnosed a patient's party guest's endodontic problem as "sensitivity caused by gum recession" and recommended a desensitizing toothpaste. Although the conversation took place in a social setting, a lawsuit can result based on an inadequate diagnosis. Suggest instead a thorough office exam with diagnostic-quality radiographs or provide a referral.

Keeping Conversations Professional

Making light of a minor occurrence, such as the dropping of an instrument, with a quip about the clinician's "one drink too many" at lunch may seem funny at the time. However, it may not sound so funny if the patient soberly reiterates the quip to a jury.

The clinician must not let a patient's flattery of the clinician's abilities undermine one's best professional judgment. Heroic measures usually result in treatment failures, dissatisfied patients, and, ultimately, lawsuits for uninformed consent.

A patient dissatisfied with prior treatment that appears adequately performed should prompt the clinician to stop treatment. Young clinicians are more apt to walk into traps involving a patient's request for unreasonable treatment. A compassionate clinician who is able to communicate conscientious concern avoids many malpractice actions. Thus, when an iatrogenic mishap occurs, it behooves the clinician to be frank and forthright with the patient. Moreover, negligence concealment may extend the statute of limitations. Most states with discovery statutes construe *discovery* as the date on which the patient discovered or reasonably suspected the negligent cause of the injury, not the date of the injury itself.[89,117,267,331] Further, belated discovery of injury from another clinician evokes a feeling of betrayal in the patient and destroys rapport that would otherwise dissuade the patient from instituting litigation. Beginning in July 2001, the Joint Commission on Accreditation of Health Care Organizations required hospitals to provide an honest explanation to patients regarding medical mishaps. These standards are designed to prevent errors and reduce medical negligence claim payouts. In 1999, the *Annals of Internal Medicine* concluded that "extreme honesty may be the best policy." A full-disclosure policy in a study involving a hospital in Lexington, Kentucky, showed that this hospital was in the top 25% of claim incidence, but it was also in the bottom 25% of claim payouts ($1.3 million over 7 years).

Fees

Clinicians should clarify fees and payment procedures before initiating treatment. If the dental treatment becomes more extensive than originally planned, the clinician should discuss any increased charges and reasons for those charges with the patient before continuing treatment. Charging for untoward complications, such as extended postoperative visits or retrieving broken instruments, should be resisted.

An overzealous receptionist who places payment pressure on a dissatisfied patient, or a clinician who sues to collect a fee from an already displeased patient, may invite a countersuit for malpractice. Refunding fees or paying for the treatment fee of the subsequent treating clinician is usually much less expensive than 1 week in court and a jury award for a patient's pain and suffering. If clinicians must sue for a fee, they should do so only if treatment is beyond reproach and records substantiate proper diagnosis, treatment, and informed consent options.

Suing for unpaid fees continues to be a proven method for getting countersued for dental malpractice. The client who has paid fees in the past but who stops at some point is either unhappy with the dental service received or short of funds. Dealing with a patient's countersuit takes time, and collection of unpaid fees may prove difficult. The patient being sued may seek an attorney to scrutinize the clinician's records for a potential cross-complaint suit against the clinician who initiated the lawsuit for fee collection.

Some cross-complaints for dental malpractice lack merit, whereas others have genuine merit. Pain, suffering, and lost wages may provide a larger potential patient recovery amount than the clinician's fees in dispute. Whenever possible, clinicians should avoid suing patients for unpaid fees. Before considering suing, the clinician should discuss the fee situation with the patient and consider a payment schedule, fee reduction, or fee waiver.

The amount of money being awarded by juries is increasing. In 1999, a New York City jury awarded $3.5 million against a clinician who had replaced three amalgams with composites. To relieve postoperative sensitivity, endodontics was performed and subsequently failed. Chronic TMD followed the extraction, from which the patient was likely to suffer lifetime pain. In Michigan, two endodontists were disciplined in 2008 for sedation overdose after settling for $3.5 million.[278] In 2008, a Spokane, Washington, jury awarded $14.2 million for unnecessary temporomandibular joint (TMJ) surgery. In Figure 29-38, a Los Angeles jury awarded $2.7 million against a periodontist who "harpooned" the IANC with his implant.

Post Perforation

Post selection is important for minimizing the risk of perforations.[68] Generally, posts should not exceed one third of the mesiodistal width of a tooth, should follow the canal anatomy, and should leave 4 to 5 mm for sealant in the opened post space; this is described more thoroughly in Chapter 22. When a tooth is already crowned, access to the root canal entrance

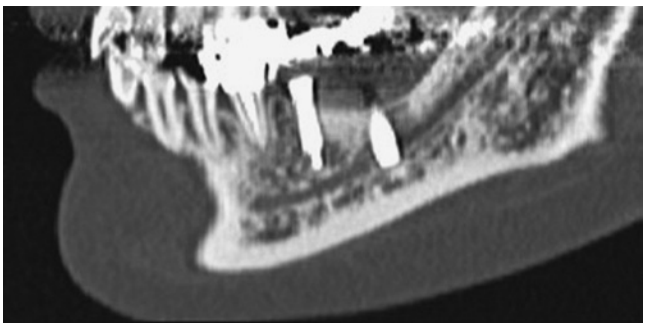


FIG. 29-38 Implant "harpooned" the inferior alveolar nerve canal.

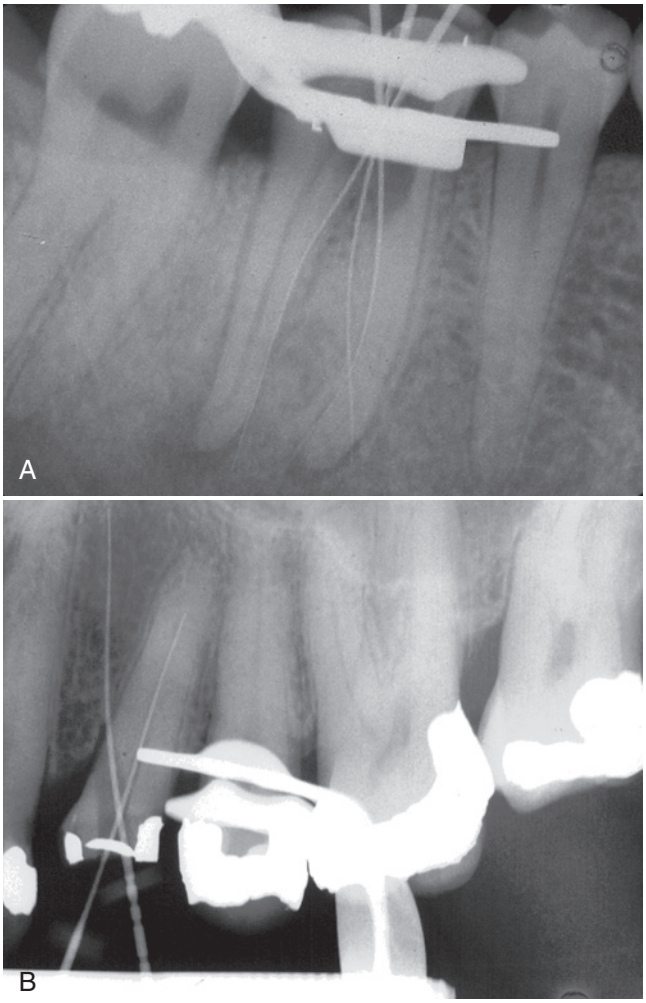


FIG. 29-39 Avoidable perforation. (Courtesy Stephen Cohen, MA, DDS.)

surface should be located before rubber dam placement. This will aid in orienting the root's long axis, which may vary from the prosthetic crown's long axis. Figure 29-39, *A* and *B*, represents an endodontist's perforation of tooth #7 because of difficulty locating the entrance into the root canal apical to the pulp chamber.[107] Figure 29-40 shows a general clinician's misaligned post and perforation of tooth #5.

Ordinarily, a careful clinician performing endodontic therapy should be able to avoid post perforations. If post perforation occurs, early diagnosis and treatment are important. Belated diagnosis and treatment substantially increase the risk of endodontic failure. If the perforation is relatively small (i.e., 1 mm or less) and promptly diagnosed at the time of the post perforation, immediate treatment with intracanal sealants (MTA) or tricalcium silicate in the area of the perforation will probably succeed. However, delayed diagnosis and treatment (beyond 24 to 72 hours) result in bacterial contamination in the area surrounding the perforation. Delayed perforation repair therapy can cause periodontal or endodontic lesions and lateral periodontal abscesses occurring secondary to delayed diagnosis, which usually prognosticate a high failure risk.

Perforation Prevention

Irreversible endodontic complication such as furcation perforation during pulpal chamber access is usually preventable with due care.[228] Furcation perforations are avoided by adhering to basic principles of pulpal chamber removal such as cleaning and shaping in a coronal rather than an apical direction. Innovative bur designs can access the pulp chamber without risking furcation perforation by preventing cutting or ditching of the pulp chamber by use of a non–end-cutting bur.[87]

FIG. 29-40 Post perforation of tooth #5.

Bleach Injury

Because of the varying shapes of the root canal system in the apical third after cleaning, shaping, and obturation, some residual bacteria may remain, which are entombed. Sodium hypochlorite is the usual disinfectant irrigant used to minimize these remaining bacteria.[268] Apical or lateral extrusion occurs when bleach is extruded past the protective apical constriction or through a root perforation. The common denominators of bleach injuries are sudden severe pain and facial swelling.* Because of the clinician's concern for emergency treatment and the patient's pain, further endodontic therapy is deferred and usually not completed at the same visit. However, two cases report that compaction was completed during the same visit, despite a bleach injury.[215,276] One article reports that subsequent flare-up may be related to the original bleach injury.[52]

Prevention of bleach extrusion is done with bleach syringes with safe, side-venting needles constantly moving to prevent hydrostatic pressure buildup from bleach fluid. Sodium hypochlorite is extruded into the periapical tissues during root canal therapy if excessive pressure is placed on the irrigating syringe, resulting in an expression of sodium hypochlorite beyond the apex. Subsequent pain is caused by the solution diffusing into the surrounding bone and soft tissue, causing swelling, tissue necrosis, and paresthesia. Surgery is usually contraindicated, and palliative treatment such as cold compresses accompanied by reassurance that most symptoms will dissipate is usually adequate.[186] However, if the bleach diffuses into vital nerve tissue of the infraorbital or inferior alveolar nerves, permanent paresthesia and occasional dysesthesia may result.[101,214,249,336] Figures 29-41 and 29-42, *A* to *C*, represent bleach injuries secondary to a root canal perforation. Paresthesia persists. Note the periapical film does not show the perforation, but the Accuitomo CBCT does.

*References 2, 101, 186, 214, 249, 260, 268, 336.

FIG. 29-41 Bleach injury.

Bleach Usage

Use of 35% H_2O_2 as a bleaching agent can be clinically adverse in the long term or after recurring bleaching treatments.

Precautions include the following:

- Reduce hydrogen peroxide concentration
- Reduce the time of each application, and increase the time between applications
- Avoid reaction catalysts, such as lamps or lasers

Post-bleaching sensitivity is mostly due to reversible pulpitis that may progress in some cases.

Cores

Incorrect choice of cores can contribute to failure, including fractures. Some manufacturers (e.g., 3M ESPE, St. Paul, MN, for Ketac silver) recommend against use of their core material unless at least two thirds of the tooth remains before buildup. Failure to follow the manufacturer's directions can be considered when an expert determines whether the standard of care was met.

Resin-reinforced post-and-core systems show promise for structurally weakened incisors, but long-term longevity has not been reported. A ferrule or other counter-rotational core design is an important consideration for fracture resistance and retention,[155] although it has not been proved as statistically significant for the resin-reinforced core systems.[279]

Absorbable Hemostatic Agents

Absorbable collagen hemostatic agents should not be placed on or adjacent to peripheral nerves because of the potential for neural injuries, particularly in bony nerve canals.[105,200] Also, as this material is absorbed, its chemotactic properties promote collagen formation and scarring. Compression injuries of peripheral nerves in bony canals can result from expanding scar tissue. FDA adverse incident reports list 11 patients with severe neural defects, including paraplegia secondary to absorbable collagen products placed in the spinal canal for hemorrhage control. Thus, the FDA warned of paralysis from absorbable hemostatic agents.[105,200] Accordingly, after hemorrhage is controlled with hemostatic agents, absorbable collagen agents should not be left in situ or near bony neural spaces. This will avoid having remaining hemostatic material cause swelling, pressure, or migration to adjacent neural tissues. The minimum amount of collagen agents necessary to achieve hemostasis should be used.

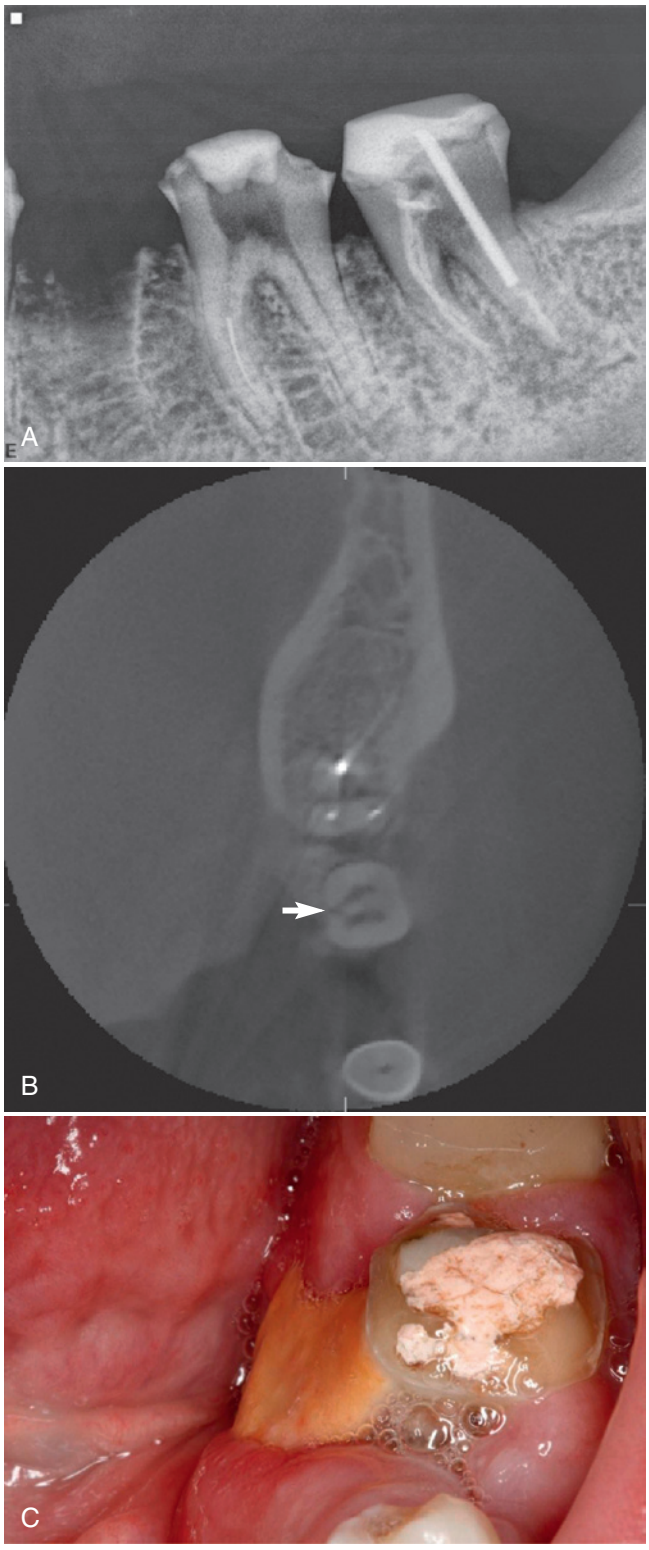


FIG. 29-42 **A**, Periapical tooth #19 showing fractured file but no perforation. **B**, Accuitomo cone-beam computed tomography (CBCT) showing perforation on the lingual side above the tooth #19 furcation. Perforation not evident in periapical view. Image demonstrates Accuitomo accuracy. **C**, Bleach extrusion through tooth #19 perforation, with resultant slough of lingual tissue.

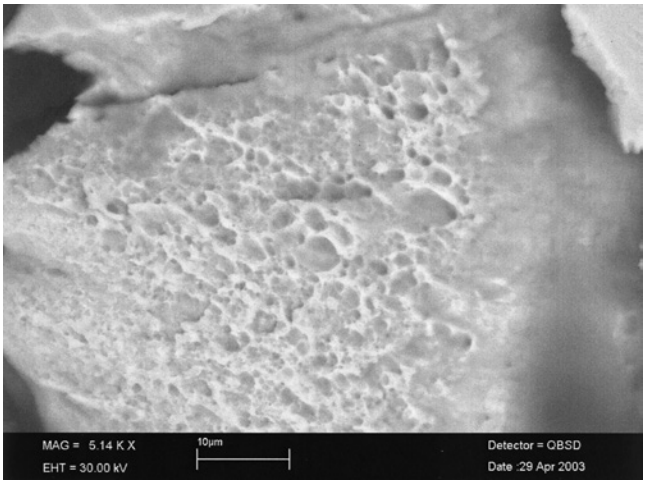


FIG. 29-43 Tensile overload shown in elongated ductile dimple voids.

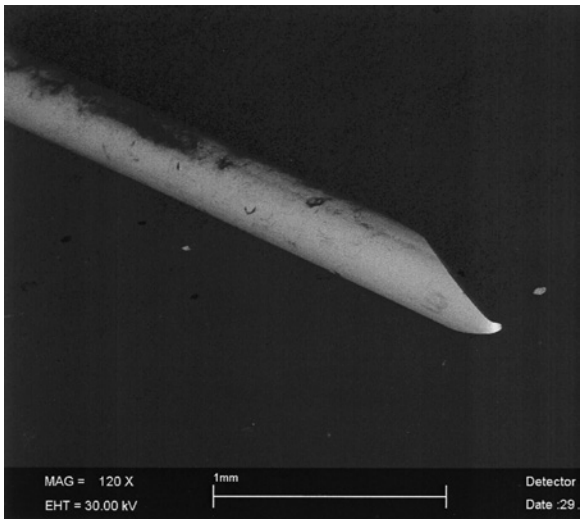


FIG. 29-44 Needle blunting of broken needle tip demonstrating striking hard object (ramus).

Pneumomediastinum (Air Embolus)

When performing endodontic surgery, a surgical handpiece should be used that ventilates air through the back of the handpiece rather than an air turbine that directs air into open tissue spaces. Pneumomediastinum, also known as *mediastinal emphysema,* may result from air embolisms dissecting down the neck facial planes from air forced into the submandibular or sublingual spaces contiguous with neck spaces.[40,301]

Broken Needle

Scanning electron microscopy (SEM) with an energy-dispersive x-ray spectrometer attachment can analyze whether a broken instrument is due to excessive operator trauma or a manufacturing defect. Figure 29-43 represents a combination of low-cycle bending fatigue and tensile overload. Accordingly, the local anesthetic needle likely fractured because of bending deformation as it struck the ramus.

Ductile rupture in metals results from shearing along planes that are oriented at 45 degrees to the tensile stress. Tensile "necking" results from multiple slipping deformations in all the 45-degree planes to the needle axis because of significant overload. Final rupture results from a combination of bending and tension. Figure 29-44 shows the needle tip bent and blunted as it hit a hard object. No manufacturing defects were found. Figure 29-45, *A* and *B*, shows SEM views of the fracture. Figure 29-45, *C*, is a SEM view of the needle fracture taken at the hub side of the fracture. These illustrations demonstrate why longer 25-gauge needles should be used for mandibular blocks rather than shorter 30-gauge needles.[207]

Preventable causes of a broken local anesthesia needle are as follows[207]:

- Use of a 30-gauge needle for mandibular block
- Bending needle
- Insertion of needle to the hub
- Multiple use of the same needle

Sterilization

The nonsurgical use of the dental unit water supply should comply with the Environmental Protection Agency regulatory standard of less than 500 colony-forming units (CFU)/mL.

CDC guidelines require sterile water or sterile solutions for all surgical procedures.[188] Disinfected water from handpieces or ultrasonic devices is no longer acceptable as a water coolant during surgery. Instead, an ultrasonic device that can deliver a sterile irrigant during surgery should be used. Dental unit water lines are available with tubing that can be disposed or autoclaved for surgery. Some ultrasonic devices are also available with disposable tubing to maintain a sterile water coolant delivery system.

Rotary NiTi files likely contain bioburden material that after sterilization may act as a foreign body. Efficiency degradation after sterilization should suggest to the prudent clinician a single-use philosophy.[31]

Digital radiographic devices that contact the oral mucosa region require a combination of barrier protective sheath and chemical disinfection in accordance with prudent manufacturers' directions.[151]

Medication Errors

The FDA has received more than 95,000 reports of medication errors since the year 2000. Medication errors involve the wrong drug, an extra or wrong dose, omission of a drug, or administering a drug by the wrong route or at an incorrect time.[190]

Medication errors are often preventable.[306] Two major sources of errors in prescribing are poor penmanship and the use of error-prone abbreviations. Writing "1.0 mg" can be read as "10 mg" if the decimal point is not clearly visible. The FDA recommends that no trailing zeros be used when denoting doses expressed as whole numbers and that preceding zeros be used whenever a decimal point is needed for a dose that must be administered as a fraction of a whole number (e.g., 1 mg, 0.5 mg). Recent drug label changes can be readily accessed on the National Library of Medicine's DailyMed website.

Posttrauma Therapy

The reader is referred to Chapter 20 for a full discussion of how to treat and manage patients who have sustained a traumatic injury.

Millennium Management of Endodontic Advances

Technologic advances are touted as ideal endodontics. However, the standard of care is a minimal standard of

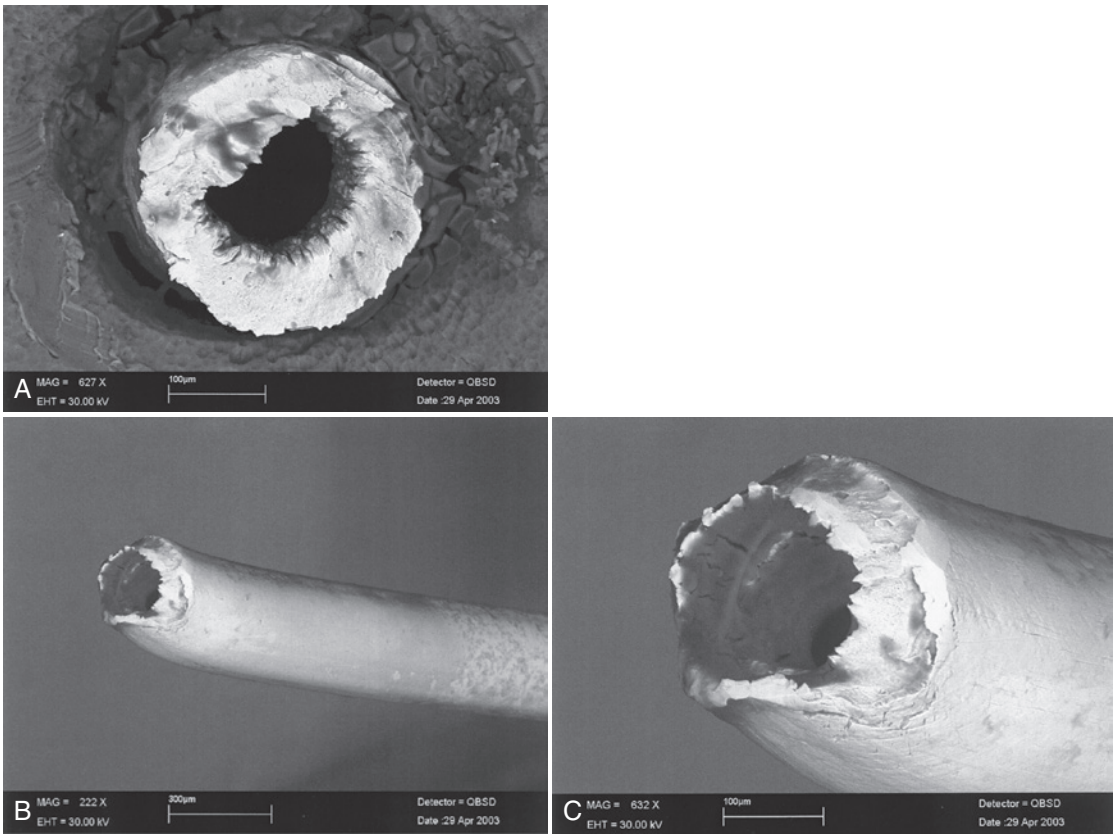


FIG. 29-45 A, Needle fracture with scanning electron microscopy (SEM). B, Needle fracture, side view (SEM). C, Needle fracture at high SEM magnification.

reasonably acceptable practice rather than the perfect ideal. The reasonable and prudent clinician is not required to know and use all the latest technologic advances in endodontics. On the other hand, the reasonable and prudent clinician must keep current with available advances that are generally accepted and proved by research. Microsurgical endodontics is an example of improved endodontic technology; use of magnifying loupes or similar devices may prove inadequate for apical surgery or fractured instrument retrieval compared with microscopes. Therefore, the clinician should adopt proven improvements in the endodontic field. Three-dimensional reconstruction of mandibles can be accomplished with cone-beam volumetric tomography to accurately locate the course of the IANC or mental foramen. Figure 29-46 shows an example of volumetric tomography usage for implant placement or apical surgery.

If studies demonstrate significantly superior results for some alternative to surgical endodontics, the informed consent standard of care may require that the patient be advised of the alternative technique, even if it is more expensive. There may be more than one path to success. So long as the clinician uses reasonably acceptable techniques and informs the patient of reasonable alternatives, the standard of care is met, but clinicians should remember that today's surgical advance may be tomorrow's retreat. For example, temporomandibular joint (TMJ) implants were inadequately tested technology on loading joints and proved virtually 100% disastrous.[102,258] Microscopic apical surgery has gained general acceptance, is performed by the majority of endodontists, and represents the current standard of care.[15,76,166,183]

FIG. 29-46 Cone-beam computed tomography usage for implant placement or apical surgery.

Clinicians should evaluate the quality of peer-reviewed research articles for new products rather than accepting them at face value. A case report amounts to no more than the author's personal experience with one patient. Some authors report a "series" of patients if there are only two patients. A brash conclusion is to state something has occurred time and time again if similar findings were observed with only two or three patients. Valid scientific principles mandate that other competent scientists duplicating a particular research protocol can replicate a test result. If research cannot be duplicated,

sweeping conclusions should not be made. Comparative effectiveness studies should demonstrate not only better results than a placebo but also relatively better results compared with similar marketed products tested under similar clinical conditions.

Statistical Research

Statistical research conclusions may vary, because research protocols may not similarly control variables of population groups studied.[137,253] Discrepancies occur even within trials or among large trials. Meta-analyses of multicenter randomized controlled trials aim to reduce bias by estimating the effect size for outcomes and adverse effects after pooling all qualified research studies. This approach will not assist if the supporting randomized controlled trials are derived from confounded data from heterogeneous trials. Such research results represent an estimate of the average difference in the responses of the tested treatment groups. These differences in results of different centers from various test groups may reflect important distinctions in the clinical conditions of enrolled research subjects. Such distinctions may help identify subgroups of patients harmed by the researched product, although on average, treatment has an overall benefit.[153]

Because most research is currently controlled and sponsored by companies marketing a product, only favorable studies are usually submitted for publication. Literature reviews are prone to publication bias because journal editors are more likely to publish favorable than unfavorable conclusions. The most important use of literature review articles is to develop research questions that ultimately must be tested using a randomized controlled trial. Studies other than randomized controlled trials were the basis for prescribing hormone replacement therapy to millions of women to prevent cardiovascular disease. A randomized controlled trial of this therapy was terminated early because increases in cardiovascular disease and breast cancer were discovered.

Before the 1980s, pharmaceutical industry grants to academic institutions to fund studies by faculty members gave investigators total responsibility to design the study, analyze and interpret the data, write the papers, and decide where and how to report the results. Neither the investigators nor their institutions had other financial connections to sponsoring companies. Drug companies now finance most clinical research on prescription drugs. There is mounting evidence that research is skewed to make drugs look better and safer than research findings warrant.*

Negative results are often not published. For example, a study of 74 clinical trials of antidepressants found that 37 of 38 positive studies were published. Conversely, of 36 negative studies, 33 were either not published or published in a form that conveyed a positive outcome.[316]

Published clinical research is too often biased, usually by designing the studies in ways that will almost inevitably yield favorable results for the sponsor.[216,337] For example, comparative drugs may be administered at a too-low dose so that the sponsor's drug looks more effective, or at a too-high dose so that the sponsor's drug has relatively fewer adverse effects. Bias also takes the form of comparing a new drug with a placebo when the relevant research question is how it compares with an existing drug. A fair comparison would contrast the new drug with an older FDA-approved drug at equivalent doses. As the former editor for two decades of the *New England Journal of Medicine* concluded in a 2008 *JAMA* article, "It is naive to assume bias is only a matter of a few isolated instances. It permeates the entire system. Physicians can no longer rely on the medical literature for valid and reliable information."[27]

Prudent clinicians do not adopt every new technology. Before adoption, such technology must have demonstrated benefits with acceptable levels of risk. It must also be adequately tested, with sufficient numbers of test subjects, over a significant length of time. Because manufacturers too often rush their products to market, newer products may not meet this criterion. Therefore, except for breakthrough technologic changes, a clinician will not likely be judged negligent for failure to adopt each latest device or technique. However, clinicians must keep in mind that in the information technology industry, 1 year is considered several generations, if not an eternity.[44,123] In general, do not be the first or the last to adopt improved technology.

The standard of care usually does not mandate incorporation of every new technology. However, in those states with informed consent laws that are based on what a prudent patient would want to know rather than what prudent clinicians should do, the patient may argue that an alternative technology or technique used by a different clinician would have been chosen had the clinician provided the patient with such information. Microscopic endodontics for broken file retrieval and CBCT to aid apical surgeries and implant placement are pertinent examples of what a reasonable patient would wish to know.[75,157]

Other Clinicians' Substandard Treatment

Clinicians should not be overly protective of blatant examples of another clinician's substandard dental treatment. On discovery of apparent negligent treatment by a previous clinician, the clinician should consider investigation. Begin by obtaining the patient's written authorization for transfer of a copy of the previous clinician's records, including radiographs. If negligence is still suspected after reviewing the records, the clinician should consider talking with the previous clinician to learn the circumstances of what occurred during the patient's past treatment (after obtaining the patient's written consent pursuant to HIPAA).

On discovery of a gross violation of the standard of care, a clinician has an ethical responsibility to report the matter to the local dental society, peer review, dental licensing board, or agency.[18,122] If the patient was misinformed and later discovers patently obvious negligent treatment, the subsequent clinician could arguably be sued as a co-conspirator to fraudulent concealment of the prior clinician's neglect.

Peer Review

If despite good rapport, candid disclosure, and an offer to pay corrective medical or surgical bills the patient is still unsatisfied, the clinician should consider referring the patient to peer review. Peer-review committees award damages for out-of-pocket losses but not for pain and suffering or lost wages. Consequently, even if the committee's decision is adverse to the clinician, the damage award will probably be less than a jury's verdict. If peer review finds for the clinician, the patient may be discouraged from proceeding further with litigation.

*References 27, 113, 190, 196, 269, 274, 284, 295, 316, 337.

DENTAL AUXILIARY CONFIDENTIALITY AGREEMENT

I, _____, have been informed by
(Dental Assistant)
_____, DDS, that all dental, medical, and
(Dentist Name)
financial information concerning patients is confidential.

As a condition of employment, I agree to maintain the confidentiality of all oral and written information, including treatment charts, and to not disclose such information to any unauthorized outside persons, including any family members, except upon request and authorization by the patient, the patient's agent, or supervising dentist.

I understand and agree that breach of this employment agreement shall constitute good cause for my discharge from employment. I acknowledge that I may be personally liable for any violation of a patient's privacy or civil rights including HIPAA committed by me without consent of the patient, or the patient's agent, or approval by above-named dentist.

Date: _____ _____
Signature of Dental Assistant

Witness: _____

FIG. 29-47 Dental auxiliary confidentiality agreement.

Peer review proceedings, including the committee's decision, are not admissible in court.[61] Insurance carriers usually honor and pay a peer-review committee award, because a fair adjudication of the merits has been determined; the award is usually less than a jury would award. Also defense costs, including attorney's fees, are saved with peer review.

Human Immunodeficiency Virus and Endodontics

A clinician may not ethically refuse to treat an HIV-seropositive patient solely because of such diagnosis.[82] Although in the 1980s, no federal law had clearly extended the protection of the handicapped laws to patients with AIDS, federal congressional action in 1990 extended this protection to the dental office setting with the passage of the Americans with Disabilities Act.[23] Many states already offer additional protection under state law.[59,83]

Confidentiality for patients disclosing their HIV status is important. An inadvertent disclosure to an insurance carrier or to other third parties without any need to know may result in cancellation of the patient's health, disability, or life insurance. This cancellation could result in a claim against the clinician whose office disclosed the information without authorization. Therefore, employees should sign the confidentiality agreement shown in Figure 29-47. In signing this agreement, the staff may be alerted to the seriousness and importance of maintaining the confidentiality of patient health histories, because these histories may document AIDS, venereal disease, or other socially stigmatizing diseases.

If a patient requests that the clinician not inform the staff of his or her HIV status, the clinician should refuse to treat that patient. This information is essential to staff members who may come in contact with the infection.[48] An accidental needle stick with HIV-infected blood, which carries a risk of approximately 1 in 250 chances of seroconversion, may occur. Current medical protocol includes prophylactic administration of zidovudine (AZT), either to prevent or to slow the manifestation of AIDS from a deep, penetrating, accidental needle-stick exposure.

Although a treating clinician risks devastating a dental practice by informing patients that the clinician has contracted AIDS, the legal risk of not informing patients is much greater. The health care provider may be required to advise patients of positive HIV test results under the doctrine of informed consent (i.e., advising of a known risk of harm from accidental exposure).[88] Even if an uninformed patient never contracts AIDS, in those states that use a reasonable patient standard for disclosure of material risks of treatment (which could include accidental direct contact such as an unintended cut or needle stick), the patient may decide to bring an action for intentional concealment as a variant of informed consent and seek to recover emotional distress and punitive damages. Conversely, patients may be legally liable for intentionally misrepresenting their health history regarding their HIV status.[51]

SUMMARY

If the clinician performs endodontics within the standard of care as described in this chapter, there should be little concern that a lawsuit for professional negligence will be successful. Prophylactic measures suggested in this chapter should lessen the likelihood of litigation by reducing, if not eliminating, avoidable risks associated with endodontic care.

Both the patient and the clinician benefit from risk reduction.[119] To do it wrong does not take long, but it is far better for the clinician to take the extra precautionary time to do it right. We are in a profession that deserves the public's trust, but only if that trust is earned. Clinicians deservedly earn public trust by providing safe and excellent quality patient care. The best prophylaxis against being sued is to make protecting the patient's best interest our paramount goal. No matter how many others do it wrong, this never makes it right. Our ethical and legal obligation adheres to the identical bedrock principle, which is service to our patients first and foremost.

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CHAPTER 30

Key Principles of Endodontic Practice Management

ROGER P. LEVIN

CHAPTER OUTLINE

Endodontic Management Systems
Systems Design
Management
Vision
Mission
Goal Setting
Target Setting
Key Production Indicators
Staff Training and Development
Scheduling
Customer Service
Marketing
Endodontic Referral Marketing
Leadership
The Endodontic Leader
Summary

Operating a successful endodontic practice requires both excellent clinical *and* management skills (Fig. 30-1). Endodontists are extremely well trained in their clinical specialty, but most do not receive extensive business training prior to becoming a practice owner. This scenario is beginning to change as more in the dental profession recognize the value of practice management skills and the role they play in an endodontist's ability to have a successful and rewarding career.

Endodontists who possess management skills are better positioned to handle the demands of operating an endodontic practice—a business that can generate significant revenue. Historically, there was a time when endodontists could enter practice without strong business skills and enjoy a long, prosperous career, but the business side of endodontics has become more complicated due to the difficult economy.

Young endodontists are leaving dental school and residency with far more debt than even a decade ago. The cost of purchasing or opening an endodontic practice continues to rise. In addition, endodontists face increased competition from other doctors who perform root canal treatments. The success of endodontic practices can also be affected by the development of new clinical techniques or services. For example, some doctors believe that dental implants represent a better treatment option than root canals, depending on the difficulty of saving the infected tooth. As dental implant procedures become easier and more common, this trend may continue to grow.

To meet these and other challenges, endodontists need to supplement their clinical expertise with excellent practice management skills. *Practice management* involves the use of documented systems to achieve superior business results. Effective practice management allows endodontists to provide optimal patient care while creating a highly productive, low-stress office environment. The design and implementation of effective practice management and marketing systems enables endodontists to operate their practices as successful businesses with steady growth and maximum efficiency. Endodontists who can combine excellent clinical skills with strong practice management skills will experience greater practice success (see Fig. 30-1).

ENDODONTIC MANAGEMENT SYSTEMS

Written, documented systems are the basic foundation for all successful operations within an endodontic practice. Step-by-step, reproducible systems enable a practice to consistently operate and be successful as both a provider of patient care and a business enterprise. Documented systems reduce unnecessary stress and allow endodontists to remain chair-side so they can focus on providing exceptional quality of care. As each system is established, every step in that system must be clearly documented and then implemented. Many endodontists fail to establish efficient systems in the early years of a new practice, and inefficient systems can hinder the practice's growth, increasing stress for the doctor and team.

Endodontists should begin their careers by developing and implementing effective practice systems. This way, when an endodontic practice begins to grow, the office has a solid management foundation in place to handle the growth with low stress as well as to reach more challenging goals. If the practice waits until it becomes more established, there is less time to design effective systems, and the endodontist often becomes

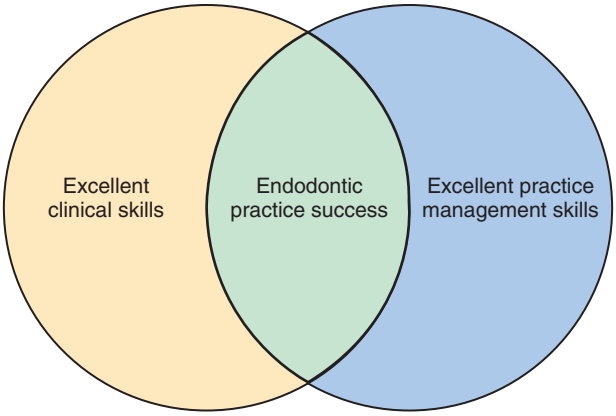

FIG. 30-1 The keys to achieving practice management success.

less motivated to do so owing to an increasingly busy office and other responsibilities in life.

Systems Design

New endodontic practice owners have an excellent opportunity to design systems because the office is nowhere near its capacity. This type of foresight sets the stage for practice growth in the years to come. However, systems should be evaluated and updated throughout an endodontist's career. It will be necessary to replace most major endodontic practice systems on a 5-year cycle to maintain growth and profitability. The reason is that practices regularly outgrow their systems as a result of changes in staff, equipment, technologies, referral relationships, and the economy.

Immersed in the day-to-day pressures of running the practice, evaluating systems can be difficult. Therefore, endodontic practices are well advised to seek outside experts to help them determine the "state of the practice." An in-office practice analysis should include the following:

- First-hand observations of the team and practice operations
- Interviews with the doctor and key team members
- In-depth assessment of all key production indicators
- Thorough evaluation of all practice systems, physical layout, and patient flow

Through such careful analysis, it is possible to accurately determine the effectiveness of practice systems that are in place.

Systems should be designed based on the specific characteristics of each practice. Areas to be considered include the following:

- Days of operation
- Hours of operation
- Number of staff
- Number of patients
- Number of true emergencies
- Average daily patient flow
- Types of procedures offered
- Number of chairs

Effective management systems can make the difference between a successful endo practice and a *highly* successful endodontic practice. Many endodontists cannot fathom how they could see any more patients, produce any more revenue, or work any harder. Yet, there exist similar practices that have much higher production in the same number of days per week with the same number of staff. It is always interesting to reflect on why one endodontic practice can be so much more financially successful than another practice in similar circumstances.

Assuming a sufficient patient flow, the answer always comes down to management systems. It is not about working harder or faster, but it is about creating systems that allow the practice to operate at a specific production and profitability level in a low-stress environment. Key systems and principles for effective endodontic management and marketing include these:

1. Vision
2. Mission
3. Goal setting
4. Target setting
5. Key production indicators
6. Staff training and development
7. Scheduling
8. Customer service

MANAGEMENT

Vision

The first question endodontists should answer before opening or purchasing an endodontic practice is "What kind of endodontic practice do I want to have?" This question requires the endodontist to take some time and identify a vision, which is a description of where the doctor wants the practice to be in 3 years. Whereas some vision statements may reflect a longer period, this shorter time frame works well for most endodontists because it is difficult to predict changes in dentistry, the economy, and other events more than 3 years into the future. In addition, after 3 years, it is time for the doctor to review what has been accomplished and possibly establish a new vision for the practice.

To create a vision statement, the following questions should be answered:

- What location is most suitable for the practice?
- What size should the practice be in 3 years?
- Does the doctor want to be in solo practice or multidoctor practice?
- What working days and hours will be satisfactory for the practice?
- What services will the practice provide?
- Is the intent to have only one practice or to have more than one location?

These are just examples of questions that can stimulate the design of a vision statement.

A vision statement clearly articulates where the practice is going. The length of a vision statement can vary between a short, single paragraph and several paragraphs. It is probably counterproductive to have a multipage vision statement; it will be difficult for both the endodontist and the staff to fully grasp such a lengthy and complex vision.

The vision guides the long-term development and growth of the endo practice. Once the vision statement has been created, it should be shared with the staff on a regular basis. Good leaders always communicate their vision to their staff. When team members understand where the practice is headed, they are empowered to help the endodontist achieve his or her vision. Sharing the vision inspires commitment and participation in achieving the vision. The following are two examples of vision statements.

Short Vision Statement

In 3 years, we will be the leading endodontic practice in the area. We will be known for exceptional quality of care and outstanding customer service. We will grow by 15% per year. Our staff is motivated and committed to continuously improving our management systems and to a lifelong learning process. We strive to meet or exceed our goals in a low-stress work environment.

Long Vision Statement

In 3 years, we will be known in the community for being the most comprehensive and skilled endodontic practice in the area, offering our patients an atmosphere of compassion and comfort.

Our excellent management skills create an environment that is low-stress and productive. We consistently meet our production goals without creating a rushed atmosphere, and our days are fulfilling.

We provide exceptional customer service that consistently generates spontaneous, positive comments. Team members have a clear understanding of their roles and responsibilities. We have developed practice systems that foster a sense of fulfillment and accomplishment for all. In year 3, we will engage an associate doctor.

Mission

Once the vision statement has been created, the next step is the development of a mission statement.

Whereas the vision statement is about where the practice will be in the future, the mission statement is focused on where the practice is today.

Once again, a strong leader has a clear practice mission and shares it with the team. Because of the similar services provided by endodontic practices, many mission statements resemble one another—they talk about quality, patient care, practice services, and customer satisfaction. It is not important to have a mission that differentiates one practice from another, but it *is* essential to have a mission that reflects the doctor's true vision. Here is an example of a mission statement:

> Our endodontic practice is dedicated to delivering quality care to meet the endodontic needs of our patients and referring doctors. We provide exceptional clinical care and customer service in a comfortable, relaxed environment.

One way to keep the mission statement on everyone's mind is to display it prominently in several places throughout the office, as well as on the practice's website. The most powerful reason for displaying the mission statement is to reinforce to the team that the mission is a living document that guides the practice. An added benefit of displaying the mission statement is the opportunity to let patients know that they are important to the practice.

The endodontist should refer to the mission statement on a regular basis. Reinforcing the mission during meetings helps staff members focus on why the practice exists and the difference the team is trying to make in the lives of patients.

Goal Setting

Goal setting represents one of the most important business activities for endodontists. It is critical to achieving success in a doctor's professional and personal life. Once a vision and mission are established, annual goals are the method to make the vision and mission realities. Goal setting is a step-by-step methodology that leads to the achievement of the overall vision. To be effective, a goal must be

1. Written
2. Deadline driven
3. Measurable

Goals that lack these three characteristics cannot be considered goals and are much less likely to be achieved. Be sure that all of the goals are aligned with the practice's vision and mission to achieve the desired results.

The pace of today's endodontic practice forces many doctors to focus on day-to-day operations. In doing so, they cannot see the big picture until many years have gone by. Goal setting and regular goal reviews can help endodontists manage their practice—instead of being managed by it.

Far more than just a motivational concept, goal setting acts as a timeline, benchmark, and directional process to achieve the vision and mission. A vision or mission without specific goals is merely an idea. Endodontists who set goals are much more likely to succeed.

Target Setting

Targets measure whether you are accomplishing your short-term and long-term goals. Each year, endodontists need to set 12 to 15 specific targets to be achieved for the next year. This helps motivate the team to reach goals and drive practice growth. Targets should be reviewed on a weekly basis. For example, if the practice is only collecting 85% of its fees, then the office needs to implement more effective financial procedures to reach the 99% target (if that is the target that has been set).

Sample endodontic targets to achieve include the following:

- Increase practice production by 15% in the next 12 months
- Ensure that 98% of all nonemergency patients are scheduled at all times
- Collect 99% of all money owed to the practice
- Maintain a less than 1% no-show/last-minute cancellation rate

Key Production Indicators

Without data, an endo practice cannot be well managed. To obtain a clear picture of current and potential practice performance, practice goals must be monitored and measured. Key production indicators (KPIs) provide an accurate assessment of the practice's most critical functions, including the following:

- Production
- Collections
- Profit
- Overhead
- New patients
- Accounts receivable
- Number of re-treats versus initial root canal treatments
- Insurance percentages
- Production per hour

These KPIs offer endodontists a starting point for analyzing their practices. Once the information has been collected, weekly measurement and analysis are necessary. Although this may sound arduous, a weekly review should take only a few minutes. Such frequent analyses can bring substantial benefits to the practice.

Evaluating KPIs every week allows endodontists to rapidly identify trends or performance deviations that may be difficult to discern otherwise. With accurate practice data, endodontists are then able to take appropriate actions.

Staff Training and Development

Establishing practice systems will only be effective if team members understand their roles in making those systems work. Training the endodontic team begins with the development of job descriptions that list the skills, responsibilities, and accountabilities for each staff position.

Bear in mind that the majority of staff will be hired without previous endodontic or dental experience and must be trained on the job. Training is specific to each position so that all team members can perform their duties as independently and effectively as possible. During training, it is critical that practices document training policies and procedures so they can be clearly communicated to new staff and regularly reinforced with established team members.

One frequent problem is that many staff members fail to fully understand their job responsibilities and are not held accountable for their performance. As a result, the endodontist will continue to handle numerous responsibilities that should be performed by other team members.

Although this type of behavior may be common in the early stages of an endo practice, endodontists who become effective leaders learn how to delegate appropriate tasks to the team. The goal for all endodontists should be to spend 98% of their workday involved in direct patient care or communication with referring doctors. To reach this goal, it is recommended that endodontists make a list of all activities they perform and which of those responsibilities can be assigned to the staff. This exercise will enable the endodontist to clearly identify which activities should be handled by the doctor only and which should be delegated to the team.

Delegation empowers the team to take ownership of their roles in contributing to practice success. The concept involves giving responsibilities to staff members so that endodontists can concentrate on what they do best. Without delegation, endodontists often carry out numerous responsibilities that can easily be performed by others. To spend more time in direct patient care, endodontists must delegate appropriate responsibilities to specific staff members. In addition, the team should be cross-trained on systems to cover for any illnesses, vacations, or turnover.

Once all team members have achieved a high level of expertise and independence, the endodontic practice can operate at peak performance. Efficiency, professional satisfaction, and quality of care steadily climb to maximum levels. At this stage, endodontists trust their teams to effectively perform their duties with minimal oversight. Best of all, doctors can focus on providing exceptional patient care without distractions and unnecessary stress.

In highly successful endodontic practices, the doctor focuses 98% of working hours strictly on patient care, interdisciplinary treatment, and referring-doctor communication. These practices will typically reach 90% to 100% of their production potential.

Scheduling

Making changes in the area of practice management should begin with a thorough evaluation of the schedule because of its impact on all other major systems. If the schedule is integrated properly with all other endodontic management systems, the practice has the best opportunity to achieve the endodontist's targets.

Schedules are created to achieve specific practice production in a low-stress environment. This means that an endodontic practice must establish a target for annual production and design the schedule accordingly. Each year, endodontic practices should calculate the desired level of growth and create a schedule that allows for that growth. The greatest challenge to increasing production is scheduling inefficiencies that make it all but impossible for the practice to grow.

The following principles are essential when designing the ideal schedule for any endo office.

Schedule for Production

Design the schedule to achieve a specific production target. Endodontic practices are typically capable of growing between 5% and 15% annually. Less than 4% growth represents moving onto a plateau. There are endodontic practices that can grow by as much as 20% to 25% in a single year as optimal systems are implemented.

Scheduling is a quantitative and mathematical activity because time is quantitative and therefore measurable. The schedule should answer questions like these: *How many root canals will we need to perform this year? Will those numbers allow for the production goal to be achieved?* Benchmark these production goals against the hours and days per week the practice is available for patient care, and adjust the schedule accordingly.

Set the Daily Production Target

Setting the daily goal is a reflection of the annual production target. To determine the daily goal, simply take the annual production goal and divide it by the number of days the practice will operate that year. Then build the schedule around this key number. Front desk personnel should become aware that the top practice management priority is to achieve the daily goal. By implementing an excellent scheduling system, they will have a far greater chance of successfully accomplishing this task.

Perform Procedural Time Studies

Many endodontic practices are unrealistic about how much time they actually need for procedures. One common example is for root canals to simply be scheduled for one set length of time, regardless of the level of complexity involved. Performing "procedural time studies" is recommended to determine the exact amount of time needed for each appointment.

During procedural time studies, each type of treatment performed by the office should be measured approximately 10 times to develop an average procedure time. Once these studies are performed and the schedule is updated, most endodontists will usually be able to increase their endodontic workload by using time more efficiently.

Young endodontists should perform procedural time studies once a year for the first 5 years they are in practice. After that, studies can be conducted at less frequent intervals (every 3 to 5 years) to evaluate whether the endodontist has become faster or more efficient in terms of experience, technique, or the use of new technologies in the practice. Procedural time studies play a critical role in building an effective and accurate

schedule, which directly affects the success of the endodontic practice.

Create the Ideal Day

The schedule should be designed to perform larger and more complex appointments in the morning whenever possible. This can be extremely challenging owing to the number of emergencies endodontists see during a normal day. By scheduling larger procedures in the morning, practices can take advantage of the high energy level for both the endodontist and the team earlier in the day. Of course, emergencies will continue to occur, but effectively managing other appointments will better position the practice to handle end-of-day emergencies. Other recommendations for creating the ideal day include the following:

- Handle less complex cases in the early afternoon.
- Perform consults and diagnoses in the midafternoon.
- Schedule all minor procedures, such as posttreatment evaluations, for the end of the day.

Basically, this means that the more difficult or larger cases will occur in the morning when the doctor and team are fresh. As the day goes forward, the doctor and staff have the opportunity to work on less complex cases, such as second visits and patient consultations, with all minor procedures taking place at the end of the day. A highly organized schedule will allow for increased productivity and higher profitability while decreasing stress for the endodontist and the team throughout the day.

Manage Emergencies

No discussion of endodontic scheduling would be complete without focusing on emergency management. Obviously, emergency procedures are a frequent occurrence in endodontic practices. Managing emergencies well requires a combination of referring-doctor management, setting patient expectations, and effective scheduling. The result will be a highly productive, satisfying professional environment for any endodontic practice.

The first step in managing emergencies is to develop a schedule that allows some flexibility. It does not benefit the practice to create a schedule that is 100% maximized without accounting for emergencies. Instead, analyze how many genuine emergencies occur on an average day, and then leave time for that number of emergencies in the schedule. Many high-level endodontic practices will schedule only 80% of their day, leaving time slots for emergencies. The key is to not ignore the time slots and invite every emergency patient to visit the office immediately, but to manage each situation appropriately.

For example, a patient who is in mild discomfort calls at 10 a.m. The practice does not have an opening until noon. If the patient is allowed to come in immediately, the office feels a need to work the patient into the schedule, not only causing stress for the doctor and team but possibly forcing already-scheduled patients to wait longer than expected. If the practice instead schedules the patient in an emergency time slot, there is a much greater opportunity to maintain excellent flow for the day and create a positive customer service experience for every patient. The main point is to provide optimal care and exceptional customer service without disrupting the daily schedule.

The keys to effective emergency management are as follows:

- Evaluate each call from a patient or referring office to determine the level of emergency. Many calls are not true emergencies, and these patients do not need to be seen immediately. Scripting and training can help staff members elicit specific information from patients about the level of discomfort (mild tenderness versus extreme pain) while providing excellent customer service.
- If it is a true emergency, bring the patient in immediately, but still evaluate the schedule to facilitate the overall process. Highly systemized endodontic practices use trained staff to evaluate emergency situations as much as possible, including preliminary assessment and x-rays.
- If the emergency call does not constitute a true emergency, it is important to use effective scripting to move the patient into an emergency time slot that will prevent the appointment from disrupting the entire schedule. This approach can be used with either a referring office or a patient.
- If the practice notices ongoing disruptions from emergencies, it should reevaluate the emergency policy and scripting to create a more effective methodology. Many endodontic practices tell every emergency patient to come in immediately. The problem arises from mistaking high-level customer service with providing immediate treatment. It is much more effective to manage the emergency patient into a time slot reserved for emergencies, unless the patient is experiencing an endodontic problem that necessitates immediate treatment. Staff training is critical for effectively managing emergencies while providing quality customer service.

Scheduling is the single most important system, because it integrates with all other systems. When an effective scheduling system has been established and properly implemented, the practice will immediately see an increase in productivity and a decrease in stress. Most stress in endodontic practices is based on poor scheduling methods that overwhelm the doctor and team and lower customer service. Even though the endodontist and team are actually working harder, profitability will be lower. An efficient schedule enables the endodontic practice to provide a high level of care and customer service to all patients.

Customer Service

Customer service today means much more than just "being nice" to patients. It is a system focused on managing the patient experience from the initial referral to treatment to postoperative care. Outstanding customer service is critical to exceeding patient expectations and building strong relationships with referring doctors.

The majority of endodontic patients are referred to the practice by general dentists. The growth of the endodontic practice therefore depends on the satisfaction and confidence of referring doctors. General dentists want to know that their patients' expectations, as well as their own, are being met. When patients receive excellent clinical treatment and superior customer service, they are likely to provide positive feedback when returning to their general dentist. Positive feedback from patients reinforces the dentist's confidence in the endodontist and leads to continuing referrals.

Developing a customer service system is more complex than simply telling the staff to be nice to patients and referral sources. A customer service system requires that patients consistently receive the same level of responsiveness, caring, and concern from every member of the endodontic practice.

Consistency and quality are at the core of a practice's customer service system.

Although the patient's time in the endodontic practice may be limited, the endodontist must realize that the patient will be returning to the referral source and sharing his or her experience with the referring doctor or the clinician's staff. The patient will also share information about the experience with family, friends, and others. Negative word-of-mouth descriptions about the office will obviously hurt the endodontic practice's reputation within the community.

Patients want to believe they are the practice's *only* priority when they come in for their appointments. Endodontic practices with excellent customer service strive to meet this expectation. If a practice fails to return phone calls quickly, is running late when patients arrive, or does not seem prepared for appointments, patient confidence and trust disappears, as does the patient's perception of quality care.

Endodontic practices should manage expectations by making everything about the patient's treatment plan value based. Do patients fully understand the value of their treatment plans? Do they know exactly what the treatment entails, what the fees are, and why the treatment must be done? Does the patient know the projected outcome? All these questions must be satisfactorily answered before treatment starts.

Endodontic practices have an opportunity to exceed expectations when the following occurs:

- There is complete understanding about why, how, and when endodontic treatment will take place
- Patients have realistic expectations of outcomes
- Patients and referring doctors receive accurate and timely information about the treatment process
- The practice has timely follow-up posttreatment communication with patients

MARKETING

Endodontic Referral Marketing

The business model of an endodontic practice is based on receiving referrals from general dentists and other doctors. There will certainly be the occasional patient who selects an endodontic practice through telephone directories, Internet, social media, or recommendation from a friend, but the majority of endodontic patients will be referred by other doctors. For endodontic practices to reach their true production and profitability potential, an effective referral marketing program should be in place. There are major challenges with implementing a referral marketing program.

Expertise

Referral marketing is a specialized form of marketing. Endodontists are trained doctors with little to no experience or expertise in this type of marketing; most endodontists typically learn about referral marketing through trial and error. This process can be time consuming and costly in terms of missed opportunities.

Time

It is not unusual for endodontists to work 8 hours a day highly focused on providing patient care and managing the practice, with little time to think about referral marketing. This is problematic because effective referral marketing involves not a few sporadic activities but a complete program that must be carried out in a systemized manner to achieve desired results.

Consistency

Inconsistent referral marketing leads to inconsistent referrals. The typical pattern is that if the practice has slowed down, the endodontist will attempt to increase referral marketing until positive results are generated. Once referrals increase, then referral marketing slows down or ceases altogether. This stop-and-start approach to marketing leaves practices more susceptible to the effects of difficult economic conditions, increased competition, and a host of other factors. A consistent referral marketing program is the solution to numerous challenges that face endodontic practices.

Referral marketing should be viewed as a mandatory part of the practice's systems. Referral marketing has two major components:

1. Establishing strong relationships with referring doctors (Fig. 30-2)
2. Designing a consistent marketing program that reaches the doctor's targeted demographic

Establishing Strong Relationships with Referring Doctors

Referral marketing differs from traditional marketing because it is relationship driven. A successful referral marketing program requires positive and consistent contact with referring doctors and offices. It is an investment in time and energy, but when done correctly it can generate a steady influx of referred patients from dentists and other doctors. Communication is critical to establishing and maintaining strong relationships with referring dentists and their teams. Sharing information about mutual patients throughout the treatment process creates a sense of partnership and teamwork that is essential to relationship building. To strengthen relationships with their referral base, endodontists should meet regularly (e.g., quarterly) with their referring doctors. These meetings can be formal or informal occasions, but they provide an opportunity to enhance both the treatment process and the referral relationship.

Designing a Consistent Marketing Program

Building a strong relationship with referring doctors requires more than one or two conversations or meetings a year. By implementing a series of consistent, ongoing marketing strategies that reach referring doctors, endodontists create greater awareness for their practices and services while building and maintaining referral relationships. Referral marketing strategies are selected from areas such as professional relationship, clinical education, social activities, and patient satisfaction. For example, hosting a practice management seminar is a "professional relationship" strategy, and attending a sporting event with a referring doctor is considered a "social activity" strategy. An effective referral marketing program encompasses multiple strategies, resulting in stronger relationships with referring doctors and their teams. The more referrals an endodontist receives, the greater potential for long-term growth and practice success.

LEADERSHIP

Leaders possess many skills, qualities, and attributes that are the result of learning, experience, and self-motivation. Many

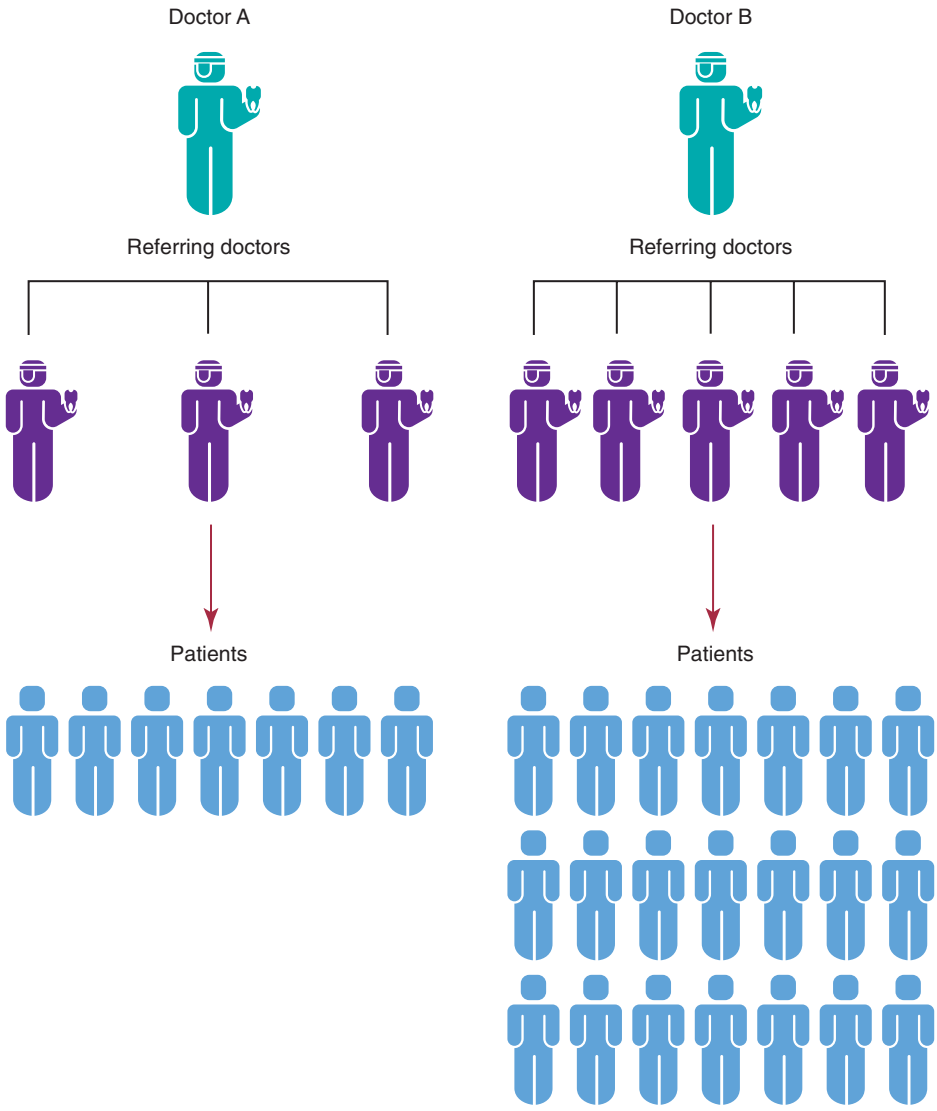

FIG. 30-2 The impact of referral marketing. *Doctor A* does not have a formal referral marketing program in place. *Doctor B* does have a formal referral marketing program in place. The results are dramatic. *Doctor B* has many more referring doctors, who refer more patients for treatment.

people have great ideas or vision, but true leaders are able to implement those ideas or carry out the steps necessary to achieve predetermined goals. They have the ability to inspire others to do their best. The most successful endodontic practices are guided by strong leaders.

Becoming an excellent leader starts with the desire to have an outstanding practice. Most endodontists are highly motivated to be excellent clinicians. Endodontic continuing education is focused mainly on clinical topics, which is appropriate, given that doctors have an obligation to provide the best possible care to patients. This makes it even more important for endodontists to seek out opportunities to develop leadership, business management, and marketing skills.

The Endodontic Leader

As practice owners, endodontists are automatically viewed as leaders by team members. The more leadership skills endodontists master, the better leaders they become. Though not an exhaustive list, the following leadership skills are critical for endodontists.

Interpersonal Communication

Interpersonal skills are the foundation of leadership. Endodontists work with a small group of people in a fairly limited space. Good interpersonal skills are essential for a smoothly functioning office environment. In business, there are many successful "tough" bosses. Even though they may be disliked, they are respected. However, being tough or inflexible is not always the best strategy, especially in dentistry. Few endodontists can be tough leaders in a very small office environment and still expect to have happy team members and a successful practice.

The best practice leaders can motivate their staff, accomplish goals, and earn the respect of team members. Regular morning meetings and monthly staff meetings provide excellent opportunities to communicate with the team, increase awareness of practice challenges and goals, and build stronger interpersonal relationships with team members.

Decision Making

As the CEOs of their practices, endodontists must make business decisions. There are many people who do not progress in

business simply because they vacillate between two choices and fail to make a decision in a timely manner. No matter what the issue, excellent leaders make decisions and trust their instincts, based on analysis, education, and experience. Why? Strong leaders believe in themselves and their knowledge. They are confident that they have the ability to make decisions that are in their best interest and that of their teams.

Motivation

The ability to motivate is one of the most important leadership skills. By earning the trust and respect of the team, the endodontic leader is able to bring together the individuals to achieve shared goals and move toward the practice vision. Many people are not self-motivated. Instead, some external force motivates them. In this case, the external force is the practice leader.

Pursuit of Knowledge

Practice leaders are usually in constant pursuit of knowledge. They want to excel in the management of the endodontic practice and will read, listen, and learn as much as possible about the subject of practice management. Leaders are typically lifelong learners who do not seek knowledge sporadically but take advantage of opportunities to learn new ideas and strategies on an ongoing basis.

Teamwork

An endodontic leader never works on his or her own. These leaders rely on their team to help achieve practice goals. However, many people mistake the concept of being a leader with being a "ruler." The "ruler" approach is not an effective one when working closely with a team whose performance is tied to the ultimate success of the practice. Therefore, endodontic leaders must remember that they are part of a group dynamic. In fact, the practice leader is like the coach of a basketball or football team. He or she creates a game plan, guides the team, and facilitates teamwork.

Time Management

Time is one of the leader's most important assets. Endodontic leaders must not only manage their own schedule but their team's as well. However, good leaders do not micromanage their team. They educate staff members, provide guidance, set expectations, and hold the team accountable for the results.

Successful leaders effectively and efficiently use their time, creating positive outcomes for themselves, their teams, and their practices. Good leaders also eliminate extraneous items from their lives that take time away from achieving their goals.

Leading by Example

The best way to earn the team's respect is to "talk the talk and walk the walk." The way the endodontist behaves will have a significant effect on how the team acts. The professionalism exhibited toward patients, the doctor's attitude and interactions with individual staff members—all of these behaviors send messages to the team. The team is looking to the endodontist for guidance, and the best strategy is to serve as an effective role model. If the team is expected to show up on time, the endodontist cannot stroll in 15 minutes late every day without causing resentment. Good leaders adhere to the rules others are expected to follow.

Leadership is a way of thinking and acting based on desire, skills, training, and specific behaviors. Endodontists who are willing to develop leadership skills will inevitably build better teams with lower turnover and higher efficiency.

FIG. 30-3 Key principles of endodontic practice management.

SUMMARY

Effective practice management involves management and referral marketing systems that enable an endodontic practice to provide outstanding patient care while operating as a successful business enterprise (Fig. 30-3). The number one source of practice stress is the lack of efficient systems. When an endo practice operates year after year without strong systems, the endodontist experiences increased stress and frustration. On the other hand, endodontists who implement documented, high-performance systems enjoy greater satisfaction (and profitability) throughout their careers.

Endodontics is a challenging and rewarding profession. With the right systems, team training, and leadership skills, endodontists can achieve their vision and create a highly successful practice.

Index

Page numbers followed by "*f*" indicate figures, "*t*" indicate tables, and "*b*" indicate boxes.

A

A fibers, 543-544, 543*f*, 543*t*, 548-549, 551
AAE. *See* American Association of Endodontists
AAOS. *See* American Academy of Orthopaedic Surgeons
A-beta fibers, 685, *e*1
Abscess
  acute apical, 30
    description of, 30, 614, 617*f*
    pulpal necrosis with, 708-711
  in apical periodontitis, 641-642, 642*f*
  definition of, 641
  management of, 715-716
  periapical, 642*f*
  swelling with, 710-711, 711*f*
Abutment teeth
  endodontic disease of, 332, 332*f*
  root-treated teeth used as, 506
  survival rates for, 515
Access cavity preparation
  access cavity
    incisal wall of, 152
    visual inspection of, 152-153
  angulations that affect, 164
  anterior teeth, 151-153, 236*f*
    coronal flaring of orifice, 152, 152*f*
    external outline form, 151, 151*f*
    inadequate, 153*f*
    lingual shoulder removal, 152, 152*f*
    pulp chamber roof penetration and removal, 151-152, 151*f*
    restorative margin refinement and smoothing, 153
    straight-line access determination, 152, 153*f*
    visual inspection of access cavity in, 152-153
  in calcified canal teeth, 162-163, 163*f*-164*f*
  cervical dentin bulge removal, 156-157, 158*f*
  challenging cases for, 157-165
  coronal, 335-336, 335*f*, 337*f*-338*f*
  coronal seal for, space adequacy evaluation for, 147
  in crowded teeth, 163-165
  errors in, 160*f*, 165, 166*f*-167*f*, 733*f*
  in heavily restored teeth, 159-162, 161*f*-162*f*
  illumination for, 147
  inadequate
    in anterior teeth, 153*f*
    complications of, 362-363
  instrumentation for
    access cavity wall preparations for, 147
    burs, 148-150, 148*f*-149*f*, 235*f*
    description of, 240
    endodontic explorer, 150, 150*f*, 164*f*
    endodontic spoon, 150, 150*f*
    handpieces, 147-148
    illumination, 147
    magnification, 147
Access cavity preparation *(Continued)*
    Micro-Openers, 146, 146*f*
    ultrasonic unit and tips, 150, 150*f*
  lingual surface for, 146, 146*f*
  magnification for, 147
  mandibular incisors
    central, 183-185, 188*f*
    lateral, 183-185, 188*f*
  mandibular molars, 236*f*
    first, 193-199, 197*f*
    second, 199, 200*f*
  mandibular premolars
    first, 155*f*, 185-192, 191*f*
    second, 192-193, 194*f*
  maxillary canines, 169, 172*f*-173*f*
  maxillary incisors
    central, 165, 167*f*-168*f*
    lateral, 165-166, 170*f*
  maxillary molars
    first, 171*f*-172*f*, 175-177, 179*f*
    second, 177-183, 182*f*-183*f*
    third, 183, 183*f*, 185*f*
  maxillary premolars
    first, 169-173, 175*f*
    second, 173-175, 175*f*-176*f*, 179*f*
  in metalloceramic crown, 159, 162, 162*f*
  with minimal or no clinical crown, 157-159
  "mouse hole" effect, 156, 157*f*
  objectives of, 145
  occlusal surface for, 146, 146*f*
  posterior teeth, 237*f*
    cervical dentin bulge removal, 156-157, 158*f*
    coronal flaring of orifice, 156-157
    external outline form, 154, 154*f*-155*f*
    mandibular first premolars, 155*f*
    pulp chamber roof penetration and removal, 154, 156, 156*f*
    restorative margin refinement and smoothing, 157
    straight-line access determination, 157, 158*f*
    visual inspection of pulp chamber floor, 157, 159*f*
  pulp chamber roof penetration and removal
    in anterior teeth, 151-152, 151*f*
    in posterior teeth, 154, 156, 156*f*
  pulpal irritation caused by, 585
  restorative margin refinement and smoothing
    in anterior teeth, 153
    in posterior teeth, 157
  in rotated teeth, 163-165
  steps involved in, 145-147
    access cavity wall preparation, 147
    cementoenamel junction evaluation, 145-146, 145*f*
    lingual surface, 146, 146*f*
    occlusal tooth evaluation, 145-146
Access cavity preparation *(Continued)*
    pulp chamber wall and flow inspection, 147
    removal of defective restorations and caries before pulp chamber entry, 146-147, 147*f*
    removal of unsupported tooth structure, 147
    space adequacy evaluation for coronal seal, 147
    tapering of cavity walls, 147
    visualization of anatomy, 145
  straight-line
    in anterior teeth, 152, 153*f*
    description of, 145, 145*f*
    in posterior teeth, 157, 158*f*
Accessory canals
  apical, 414*f*
  description of, 132-133, 286
  illustration of, 414*f*
  in mandibular first molars, 133, 133*f*
Acellular afibrillar cementum, 630
Acetaminophen
  analgesic uses of, 117
  nonsteroidal anti-inflammatory drugs and, 121
  in pregnancy, 74
Acetylcholine, 557-558
Actinomyces, in periapical granuloma, 483*f*
Activ GP, 292, 292*f*, 299
Acute apical abscess, 30
  description of, 30, 614, 617*f*
  pulpal necrosis with, 708-711
Acute apical periodontitis, 638, *e*5
  apical bone destruction in, 641
  clinical features of, 641
  description of, 638
  macrophages in, 639
  natural killer cells in, 639
  outcomes of, 641-642
  polymorphonuclear neutrophilic leukocytes in, 638
Acute endodontic pathosis, 83*f*
Acute inflammation, 475
Adaptive immune response, 634-635, 636*f*, 642-643
Adaptive immunity, 633*t*, 634
Adaptive prior image constrained compressed sensing algorithms, 52
A-delta fibers, 685-686, *e*1
Adhesive systems, 844, *e*2
Afferent neurons, 542
Aging, pulp changes caused by, 566
Air emphysema, 749-750, 749*f*-750*f*
Allergic reactions
  to latex, 93-94
  to local anesthetics, 93-94
  sulfite-induced, 94
Allodynia, 550-551, 550*t*, 554, 693-694

Allograft, 435*t*
Alloys
  nickel titanium, 219-222, 221*f*-222*f*
  of root canal instruments, 219-222, 222*f*
Alprazolam, for oral conscious sedation, 113
Alternating wall contact points, 231
Alveolar bone, 631
Alveolar nerve blocks
  anterior middle superior, 103-104, 104*f*
  inferior. *See* Inferior alveolar nerve block
  palatal–anterior superior, 103, 104*f*
Alveolar process, 631
Amalgam core, 826, 833*t*, 840*f*, 844
Amalgam restorations, *e*10
American Academy of Oral Medicine, 71, 78
American Academy of Orthopaedic Surgeons, 78
American Academy of Pediatric Dentistry, *e*1
American Association of Endodontists
  Case Difficulty Assessment Form and Guidelines, 707*f*, 708
  description of, 73, 637
  vertical root fracture criteria, 802
American Board of Endodontists, 637
American Society of Anesthesiologists
  Physical Status Classification system of, 71, 72*b*
  surgical risk system, 395-396
AMPA/kainate antagonists, 554
Anachoresis, 601
Analgesics
  flexible strategy for, 121*f*
  non-narcotic
    acetaminophen, 117
    alternatives to, 117
    contraindications for, 117
    drug interactions with, 117, 117*t*
    efficacy of, 116*t*
    limitations of, 117
    nonsteroidal anti-inflammatory drugs, 114-115
    overview of, 114-117
    types of, 115*t*
  opioid
    description of, 117-118, 118*t*
    nonsteroidal anti-inflammatory drugs and, 121
  pain management using, 719
Anastomoses, root canal, 140
Anesthesia
  for endodontics
    infiltrations, 109
    intraligamentary, 110-111
    intrapulpal injection, 110-111
    local, 109-111
  future directions for, 114
  for incision and drainage procedures, 113-114
  intraligamentary
    amount of solution delivered, 108
    anesthetic solutions for, 107
    avulsion after, 109
    back-pressure for, 107
    computer-controlled local anesthetic delivery system for, 108, 108*f*, 110
    description of, 101
    discomfort during, 108
    duration of anesthesia with, 108
    endodontic uses of, 110-111
Anesthesia *(Continued)*
    indications for, 107
    mechanism of action, 107
    onset of anesthesia, 108
    periodontal safety with, 108
    postoperative discomfort caused by, 108
    primary teeth safety with, 109
    pulpal safety with, 109
    success of, 107
    systemic effects of, 108
  intraosseous
    alveolar mucosa injection of, 106
    attached gingiva injection of, 106
    bupivacaine for, 106
    definition of, 104
    description of, 101
    discomfort associated with, 106
    duration of anesthesia with, 104, 105*f*, 106
    endodontic uses of, 109-110
    failure of, 105
    heart rate increase after, 106-107
    injection pain with, 110
    injection site locations, 106
    lidocaine levels in plasma after, 107
    for mandibular posterior teeth, 111
    medical considerations for, 107
    onset of anesthesia, 106
    perforator breakage with, 105-106
    postoperative discomfort from, 107
    repeating of, 106
    Stabident system for, 104, 105*f*
    success with, 105
    X-Tip delivery system for, 104-106, 105*f*, 110
  intrapulpal, 581
  local. *See* Local anesthesia/anesthetics
  for mandibular anterior teeth, 112
  for mandibular first molars, 99*f*
  for mandibular posterior teeth, 111, 113
  maxillary, 101-103, 102*f*
  for maxillary anterior teeth, 112
  for maxillary molars, 102*f*
  for maxillary posterior teeth, 112
  noncontinuous, 96
  pain satisfaction with, 110-111
  for periapical surgery, 114
  pulpal
    articaine for, 99
    confirming of, 95, 95*f*
    inferior alveolar nerve block for, 96-97, 97*f*
    maxillary, 102-104
  for pulpal necrosis teeth, 112
  selective, 20
  summary of, 114
Angiogenesis, in wound healing, 392-393
Anterior middle superior alveolar nerve block, 103-104, 104*f*
Anterior teeth. *See also specific anterior teeth*
  access cavity preparation in, 151-153, 236*f*
    coronal flaring of orifice, 152, 152*f*
    external outline form, 151, 151*f*
    inadequate, 153*f*
    lingual shoulder removal, 152, 152*f*
    pulp chamber roof penetration and removal, 151-152, 151*f*
Anterior teeth *(Continued)*
    restorative margin refinement and smoothing, 153
    straight-line access determination, 152, 153*f*
    visual inspection of access cavity in, 152-153
  structurally compromised, 834
  structurally sound, 830
Antibiotics
  bacterial resistance to, 718
  definition of, 718
  description of, 119-120
  endodontic infections treated with, 717-719
  overprescribing of, 119
  prophylactic administration of
    before periradicular surgery, 396, 402-404
    description of, 119
  selection of, 718-719
  systemic, 787
Anticoagulants
  guidelines for, 73
  heparin, 397
  novel oral, 397
  periradicular surgery in patients receiving, 396-397
Antigen-presenting cells, 538-539, 634
Antimicrobial nanoparticles, 265
Antiresorptive agent-induced osteonecrosis of the jaw, 394-395
Anxiety, 87
Apex locators, 237-238, 238*f*
Apexification
  apical periodontitis treated with, 467
  bioceramic barrier, 770-773, 771*f*
  calcium hydroxide for, 458, 770, 770*f*, 772*f*
  dentinal wall reinforcement, 772
  disadvantages of, 458
  hard tissue apical barrier, 770-773, 770*f*-772*f*
  of immature teeth, 769-773, 770*f*-772*f*
  indications for, 458, 769
  mineral trioxide aggregate, 462-464, 468, 771*f*
Apical abscess
  acute, 30
    description of, 30, 614, 617*f*
    pulpal necrosis with, 708-711
  chronic, 30, 646, 800-801
Apical barrier
  calcium hydroxide for, 314
  creation of, 770-773, 770*f*-772*f*
  mineral trioxide aggregate as, 314, 314*f*, 771*f*
Apical constriction
  description of, 142, 142*f*, 285, 285*f*
  localization of, 144
  root apex and, 143*t*
  root canal procedure termination at, 144, 238, 285, 286*f*
Apical cysts, 647-649, 649*f*, 654
Apical diameter, 238
Apical foramen
  cementum extension into, 142
  description of, 142
  diameter of, 142
  localization of, 144

Apical foramen *(Continued)*
in maxillary anterior teeth, 142-143
odontoblasts, 533
size of, 143*t*
Apical granuloma, 642-646
Apical infection, 632
Apical inflammation, 632
Apical lesion, 327*f*
Apical papilla
anatomy of, 449-450, 450*f*
stem cells of, 449-451, 450*f*, 460
Apical pathosis, 211*f*
Apical periodontitis
abscess in, 641-642, 642*f*
acute
apical bone destruction in, 641
clinical features of, 641
description of, 638
macrophages in, 639
natural killer cells in, 639
outcomes of, 641-642
polymorphonuclear neutrophilic leukocytes in, 638
apexification for, 467
asymptomatic
bacteria in, 645-646, 647*f*
bone resorption in, 645
cells involved in, 643-645, 647
characteristics of, 642
chronic focal sclerosing osteomyelitis associated with, 650
clinical features of, 646
condensing osteitis associated with, 650
cyst formation with, 646-649, 654
description of, 30, 331*f*, 642-646
epithelial cell rests of Malassez in, 644, 645*f*
external root resorption in, 645
fibroblasts in, 644-645
histopathology of, 645-646
inflammatory mediators in, 645, 647
lymphocytes in, 643
macrophages in, 643
osteoclasts in, 643-644
outcomes of, 646, 649
periapical bone destruction in, 643
radicular cysts with, 646-649, 649*f*, 654
reactive bone formation with, 650
bacteria in, 600, 602-603, 622-623
as biofilm-related disease, 609-610
caries as cause of, 631-632
cells involved in
dendritic, 643
endothelial cells, 638
epithelial cell rests of Malassez, 644, 645*f*
fibroblasts, 644-645
lymphocytes, 643, 645
macrophages, 639, 643, 645
mast cells, 638
natural killer cells, 639
osteoclasts, 643-644
platelets, 639
polymorphonuclear neutrophilic leukocytes, 638-639
cemental changes in, 645
cholesterol crystals in, 645

Apical periodontitis *(Continued)*
chronic
asymptomatic, 632-633
bacteria in, 624
cysts associated with, 647-649, 648*f*
inflammatory mediators in, 647
nerve fibers in, 646*f*
clinical features of, 641
clinical findings, 635
cone beam computed tomography diagnosis of, 328-329
definition of, 599
development of, 637-638
diabetes mellitus and, 650-652
diagnosis of, 635-636
endogenous factors, 631
etiology of, 631-632
exogenous factors, 631
genetic risk factors for, 651-652
healing of, 85, 283
histologic findings, 635-636
histopathology of, 636-637, 641, 645-646
illustration of, 603*f*
as infectious disease, 599-600
inflammatory mediators in, 639-641, 640*t*, 645
intraradicular infection as cause of, 614*f*, 622-623
lesion associated with, 614
microbial causation of, 614-615
microbiologic issues for, 82
microorganisms that cause, 600
multifactorial nature of, 604
nonsurgical therapy for, 389
outcomes of, 641-642, 649
pathogenesis of, 633-635
adaptive/specific immune response, 634-635, 636*f*, 642-643
innate immune response in, 633-634, 633*t*
neurogenic inflammation, 635, 636*f*
periradicular surgery for, 389, 394-395
periradicular tissue responses, 631
persistent
genetic risk factors for, 651-652
systemic disease risk factors for, 652
pocket cyst in, 646-649, 648*f*
posttreatment, 329, 380-381, 620*f*
prevalence of, 631
prevention of, 477
primary intraradicular infection as cause of, 614*f*
pulpal infection as cause of, 631-632
after pulpal necrosis, 637-638
radiographs of, 361*f*, 636
risk factors for, 651-652
symptomatic
cells involved in, 638-639
description of, 19, 614-615, 637-642
herpesvirus in, 619
histopathology of, 641
inflammatory mediators in, 633*t*, 639-641
trephination for, 708-709
systemic diseases associated with, 650-652
wound healing of
cytokines in, 653, 653*t*
description of, 652-653
diabetes mellitus effects on, 654-655

Apical periodontitis *(Continued)*
factors that affect, 654-655
growth factors in, 653, 653*t*
periapical, 652-654
Apical pressure, 156
Apical pulpitis, 637-638
Apical root resorption, 237
Apical surgery
nonsurgical retreatment versus, 334
periapical healing following, 482*t*, 519*t*-520*t*, 521*f*
posttreatment disease after, 334*f*
in silver point cases, 361, 363*f*
Apical tissue, 29
Apicomarginal defect, 426-428
Apoptosis, 634
Arachidonic acid metabolism, 640
Archaea, 619
Argyrophilic collagen fibers, 537
Articadent. *See* Articaine, with 1:100,000 epinephrine
Articaine
with 1:100,000 epinephrine, for inferior alveolar nerve block, 97-98
for endodontics, 109
insurance carrier warning about, 98
supplemental infiltrations of, 101, 104
As low as reasonably achievable principle, 52, 62, 329
Asians
mandibular second molar root configurations in, 137, 140*f*
maxillary first premolars in, 169
Aspergillosis, maxillary sinus, 741*f*
Aspirin
analgesic use of, 719
description of, 73
discontinuation of, before periradicular surgery, 397
opioid analgesics and, 121-122
Asymptomatic apical periodontitis
bacteria in, 645-646, 647*f*
bone resorption in, 645
cells involved in, 643-645, 647
characteristics of, 642
chronic focal sclerosing osteomyelitis associated with, 650
clinical features of, 646
condensing osteitis associated with, 650
cyst formation with, 646-649, 654
description of, 30, 331*f*, 642-646
epithelial cell rests of Malassez in, 644, 645*f*
external root resorption in, 645
fibroblasts in, 644-645
histopathology of, 645-646
inflammatory mediators in, 645, 647
lymphocytes in, 643
macrophages in, 643
osteoclasts in, 643-644
outcomes of, 646, 649
periapical bone destruction in, 643
radicular cysts with, 646-649, 649*f*, 654
reactive bone formation with, 650
Asymptomatic irreversible pulpitis, 28-29
symptomatic irreversible pulpitis versus, 109
Asymptomatic vital teeth, pulp anesthesia determinations in, 95, 95*f*
ATD Automatic Crown & Bridge Remover, 336

Atypical facial pain, 694-695
Atypical odontalgia, 44, 690, 694-695
Austenite, 219-221, 222*f*
Autogenous bone graft, 435*t*
Autonomic nervous system, 687
Avulsed teeth
  consequences of, 784-785
  description of, 759
  emergency management of, 785
  pulpal necrosis caused by, 785
  replantation of, 785
  root preparation in, 786-787
  socket preparation in, 787
  treatment of, 785

B

B cells, in adaptive immunity, 635
Bacteremia, periradicular surgery and, 396
Bacteria. *See also* Microorganisms
  antibiotic-resistant, 718
  in apical periodontitis, 600, 602-603, 622-623
    asymptomatic, 645-646, 647*f*
    chronic, 624
  biofilms, 602, 603*f*, 604, 606*f*
  in caries, 601
  in chronic apical periodontitis, 624
  in dentinal tubules, 591-592, 600-601
  host defense mechanisms against, 603-604
  intraradicular infection caused by, 614, 615*f*-617*f*
  multidrug-resistant, 718
  nutrient sources for, 617-618
  during obturation, 620
  phagocytosis of, 647*f*
  in radicular cysts, 649*f*
  root canal infection caused by, 616-617
  structure of, 603-604, 604*f*
Bacterial endocarditis
  description of, 73
  periradicular surgery in patients with, 396
Balanced force technique, for canal enlargement/preparation, 243, 243*f*, 737
Barbed broaches, 224, 224*f*
Batt tips, 214, 218*f*
B-cell antigen receptors, 634
Behavioral disorders, 78
Bioactive glass, 262
Bioactive protein graft, 435*t*
BioAggregate, *e*3
Biocalex 6.9, 356-358, 358*f*
Bioceramics. *See also* Mineral trioxide aggregate
  pulp capping uses of, 766
  as root-end filling materials, 422
Biodentine, *e*3
Biofilms
  antimicrobial resistance of, 608-609
  apical periodontitis associated with, 609-610
  bacterial interactions with, 607-608, 607*f*
  community lifestyle of, 608
  definition of, 607
  description of, 602, 604
  extracellular polymeric substances in, 607-608
  extraradicular, 609, 624
  illustration of, 603*f*, 606*f*
  infections and, 609
  intraradicular, 609
Biofilms *(Continued)*
  microcolonies in, 608
  in root canal system, 631
Biologic debridement, 654
Biopsy, in periradicular surgery, 411-412
BioPure MTAD, 258-259, 258*f*
BioRaCe instrument, 231
Bisphosphonates
  indications for, 394
  medication-related osteonecrosis of the jaw caused by, 76, 655
  osteonecrosis of the jaw caused by, 394
  wound healing affected by, 394-395
Bite test, 19-20, 20*f*
Biting force, 829
Bleeding, after periradicular surgery, 437
Blood clots
  formation of, 392
  Nygaard-Ostby's work with, 448
  prevention of, 767. *See also* Anticoagulants
Blood pressure
  elevated, 73
  measurement of, 5
Bone
  burs for removal of, 411
  glucocorticoids effect on, 395
  heating effects on, 411
  homeostasis of, 395
  loss of, vertical root fracture-associated, 800*f*-801*f*
  regeneration of, 653-654
  resorption of
    in asymptomatic apical periodontitis, 645
    inflammation-induced, 633
    nonsteroidal anti-inflammatory drugs effect on, 395
    osteoclasts as cause of, 643, 644*f*
Bone formation
  lamellar, 393-394
  phases of, 393
  woven, 393
Bone grafts, 435*t*
Bone marrow stem cells, 449
Bone morphogenetic proteins, *e*2
Bone morphogenic proteins, 434
Bone sialoprotein, 452-453
Bone wax, for hemostasis, 413
Bradykinin, 551, 640
Brasseler Endo Extractor Kit, 362*f*
Brasseler extractors, 369
Breast cancer, 73
Breastfeeding
  concerns for, 75
  drugs used during, 74*b*
Buccal space, 712*f*-714*f*, 713
  infection of, 9-10, 9*f*-10*f*
  swelling of, 9*f*-10*f*
Buccal vestibular space, 712*f*-714*f*, 713
Buccinator muscle, 713
Bupivacaine
  intraosseous anesthesia using, 106
  local anesthesia using, 98, 98*f*
Burs
  access cavity preparation using, 148-150, 148*f*-149*f*
  bone removal using, 411
  diamond, 148, 148*f*-149*f*, 235
  Extendo, 149, 149*f*
Burs *(Continued)*
  fissure carbide, 148, 148*f*
  Gates-Glidden, 152, 156-157, 225*f*, 235, 236*f*
  Mueller, 149, 149*f*
  root canal system cleaning and shaping using, 224
  root-end surface preparation in periradicular surgery using, 416
  round carbide, 148, 148*f*
  round diamond, 148, 149*f*
  transmetal, 148-149, 149*f*
  types of, 148-150, 148*f*-149*f*

C

C fibers, 30, 543*f*, 546, 548-549, 551, 686-689
C+ files, for gutta-percha removal, 348-349, 349*f*
Calamus flow obturation delivery system, 309*f*, 310
Calcific metamorphosis, 566, 566*f*
Calcifications
  pulpal, 372*f*, 564-566, 564*f*-566*f*
  root canal
    access cavity preparation in, 162-163, 163*f*-164*f*
    cone-beam computed tomography of, 49-50
    removal of calcified material, 163
Calcifying odontogenic cysts, 647-649
Calciobiotic root canal sealer, 292
Calcitonin gene-related peptide, 544*f*-545*f*, 547, 552*f*, 577, 632, 653*t*
Calcium hydroxide
  antibacterial use of, 788
  apexification using, 458, 770, 770*f*, 772*f*
  apical barrier use of, 314
  application of, 261*f*
  benefits of, 260
  characteristics of, 260, *e*2
  chlorhexidine with, 256
  clinical protocol for, 260
  direct pulp capping using, *e*2
  disadvantages of, 766
  Enterococcus faecalis resistance to, 622
  illustration of, 770*f*
  inflammatory response caused by, 587*f*
  as intracanal medicant, 708
  intracanal use of, 459-460
  limitations of, 260-261
  obturation uses of, 292, 314
  partial pulpotomy, 766*f*
  pH of, 765-766, 781*f*
  pulpal irritation caused by, 586
  in pulpal necrosis, 285, 765-766
  reparative bridge formation using, *e*4*f*
  sodium hypochlorite and, 679
  studies of, 459-460
  uses of, 260
  vital pulp therapy use of, 765-766, 765*f*, *e*5
Calcium silicate cements, *e*1, *e*6-*e*10
Calcium silicate sealers, for obturation, 294-295, 295*t*-296*t*
Calcium sodium phosphosilicate bioactive glass, 579
Calcium sulfate, 770-771
  for hemostasis, 413

Camphorated paramonochlorophenol, 255
Canal transportation, 267-268
Cancellier extractors, 369, 369*f*
Candida albicans, 622
Canine space, 715
  infection of, 9, 9*f*
  swelling of, 9*f*
$Ca(OH)_2$. *See* Calcium hydroxide
Capillary plexuses, 392
Capsaicin, 547
Carbamide peroxide, for vital bleaching, 590-591
Carbocaine, 97
  with Neo-Cobefrin, 97
Carbon dioxide, for cold testing of pulp, 16, 16*f*
Carbonated anesthetic solutions, 100
Cardiac pain, 697
Cardiovascular disease, 72-73
Cardiovascular system, 93
Caries
  apical periodontitis caused by, 631-632
  bacteria in, 601, *e*1
  deep
    in mandibular first molars, *e*8*f*
    in mandibular second molars, *e*13*f*
    occlusal, *e*8*f*
    radiographs of, 730*f*, *e*5*f*, *e*16*f*
  dendritic cell accumulation caused by, 575-576, 576*f*
  dentin layers of, *e*6
  dyes for detecting, *e*6
  humoral immune response to, 576
  inflammatory response to, 575, 575*f*
  lasers for prevention, diagnosis, and treatment of, 588-590, 589*f*
  proliferative response to, 577*f*
  pulpal exposure caused by
    description of, 578
    direct pulp capping for, 587-588
    disinfectants effect on, 588
    hemostatic agents effect on, 588
    illustration of, 578*f*, 603*f*
  pulpal reactions to, 573-576, *e*2-*e*6
  radiation therapy and, 652
  remaining dentin thickness after progression of, 584
  removal of, *e*9
  removal of, before access cavity preparation, 146-147, 147*f*
  Sjögren syndrome and, 652
Carrier-based gutta-percha, 311-313
Case Difficulty Assessment Form and Guidelines, 707*f*, 708
Case selection
  description of, 71
  factors that affect, 86-88
Cast core, 826-827
Cast gold post and core, 844-846
Cast posts, 337, 826-827, 833*t*
Caufield silver point retrievers, 359-360, 360*f*
Cautery, in periradicular surgery, 414
Cavernous sinus thrombosis, 715
Cavit, 315
CBCT. *See* Cone beam computed tomography
CD4+ T cells, 77, 634
CD8+ T cells, 634-635
CD14, 450-451
CDJ. *See* Cementodentinal junction
CEJ. *See* Cementoenamel junction
Cell homing, 455-456, 456*f*
Cellulitis
  description of, 710
  management of, 715-716
Cement(s)
  calcium silicate, *e*1, *e*6-*e*10
  glass ionomer
    luting, 827
    pulpal responses to, 586-587
    as root-end filling material, 420
  luting, 827-828
  polycarboxylate, 827
  resin-based, 827-828
  resin-modified glass ionomer, 826, *e*5-*e*7
  self-adhesive, 828
  tricalcium silicate, 294
  zinc phosphate, 827
Cementation, of post, 337-339
Cementoblasts, in hard-tissue healing, 394
Cementodentinal junction
  description of, 142
  evaluation of, 145-146, 145*f*
Cementoenamel junction
  anatomy of, 667
  location of, 145
Cementogenesis, 394
Cementoma, 22*f*
Cementum
  acellular afibrillar, 630
  apical foramen extension of, 142
  definition of, 630
  formation of, 478*f*
  growth factors in, 630, 653
  resorption of, 643-644
  in root resorption, 653
Central nervous system, 686-687
Central neuropathy, 696
Central sensitization, 553-555, 687, 688*b*, 695
Centrality, 145
Ceramic graft, 435*t*
Ceramic onlays and overlays, 822, 830*t*
Cervical dentin
  bulges of, 156-157, 158*f*
  root canal cleaning and shaping objectives for, 211
Cervical fasciitis, 716
Cervical lymph nodes
  palpation of, 9
  tuberculosis involvement of, 5-6
Cervical pulpotomy, 768
Cervical spaces, 715
Cervicofacial subcutaneous emphysema, 748-750, 749*f*-750*f*
C-factor, 821-822, 830-833
c-fos, 553
Charged coupled device
  description of, 33-34, 61
  external inflammatory resorption diagnosed using, 666
Chemokines
  cells that produce, 575
  description of, 638, 641
Chemomechanical debridement, 666, 679
Chemotherapy, wound healing affected by, 75-76
Chief complaint, 699
  diagnostic use of, 2-5
  investigating of, 2-5
Children
  crown fractures in, *e*14*f*
  pulpal exposure in, *e*16*f*
Chlorhexidine
  allergic reactions to, 256
  antibacterial efficacy of, 255
  before periradicular surgery, 402-404
  calcium hydroxide with, 256
  cytotoxicity of, 254-255
  as decalcifying agent, 256-257
  dentin bonding and, 256
  as endodontic irrigant, 255
  history of, 254
  indications for, 404
  as intracanal medicine, 255
  mode of action, 254
  molecular structure of, 254, 254*f*
  sodium hypochlorite and, 256*f*
  substantivity of, 254
Chloroform, for gutta-percha removal, 347-348, 348*f*, 742-743
Chondroitin sulfate, 541
Chronic apical abscess, 30, 646
Chronic apical periodontitis
  asymptomatic, 632-633
  bacteria in, 624
  cysts associated with, 647-649, 648*f*
  inflammatory mediators in, 647
  nerve fibers in, 646*f*
Chronic continuous dentoalveolar pain disorder, 44
Chronic focal sclerosing osteomyelitis, 650
Chronic inflammation, 475
Chronic pain, 700-702
Chronic renal failure, 77
Citanest Forte, 97
Citanest Plain, 97
Citric acid
  root-end surface modification using, 416
  smear layer removal using, 288-289, 416
Clinical decision making
  for periradicular surgery, 389-390
  shared approach to, 389-390
Clot formation, 392
Cluster headaches, 692
CO_2 lasers, 588
Coaggregation, 619
Codeine, 118, 122
Cold testing, of pulp, 16, 16*f*-17*f*, 547
Collagen
  in dentin, 452-453
  odontoblast synthesis of, 535
  procollagen, 541-542
  in pulpal interstitium, 540
  as scaffold, 455
  synthesis of, 541-542
  in wound healing, 392-393
Collagen fibers, in pulp, 541-542, 542*f*
Collagen fibrils, 537
Collagen-based hemostasis products, 412, 413*f*
Colloid osmotic pressure, 558
Colony-stimulating factor, 653*t*
Communities, of microorganisms, 606-607
Complement cascade, 640-641

Complementary metal oxide semiconductor, 33-34, 34*f*
Complicated crown fractures, 763-765, 763*f*-764*f*
Composite resin core, 826, 833*t*, 842*f*-843*f*
Composite resins
  polymerization of, shrinkage during, 584
  as root-end filling material, 420-421
Computed radiography, 34-35
Computed tomography
  cone beam volumetric, 36-37
  cone-beam computed tomography versus, 400-402
  history of, 36-37
  micro-, 37
  multidetector, 37
Computer-controlled local anesthetics delivery system
  description of, 100
  for intraligamentary anesthesia, 108, 108*f*, 110
Concentricity, 145
Concussion, 777
Condensing osteitis, 650
Cone beam computed tomography
  American Association of Endodontists/American Academy of Oral and Maxillofacial Radiology position statement on, 63-66
  anatomic landmarks on, 55*f*-58*f*
  apical periodontitis diagnosis using, 328-329
  classification of, 400-401
  computed tomography versus, 400-402
  dental anomalies diagnosed using, 45-46
  description of, 33, 37
  diagnostic uses of, 23-24, 24*f*, 37, 38*f*, 328-329, 401*f*
  differential diagnosis uses of, 43-44, 44*f*
  endodontic treatment applications of
    complications
      calcified canals, 49-50
      dentoalveolar trauma, 51-52, 52*f*-53*f*
      external root resorption, 52-55, 54*f*
      fractured instruments, 48-49, 51*f*
      internal root resorption, 52-55, 54*f*
      materials extending beyond root canal, 46-48, 50*f*
      perforations, 50-51
    failure of treatment, 44-45, 45*f*
    outcome assessments, 402
  external cervical resorption evaluations, 670*f*-671*f*, 672
  external inflammatory resorption evaluations, 666
  field of view, 39-42, 41*f*
  full, 400-401
  future of, 60
  hardware for, 23*f*
  imaging software, 39
  implant site assessments using, 60
  inferior alveolar nerve injury, 748
  internal root resorption evaluations, 677
  Intersocietal Accreditation Commission for, 62
  limited, 400-401
  mechanism of action, 37, 400-401
  outcomes assessment using, 56-60
  patient characteristics, 40-42
Cone beam computed tomography *(Continued)*
  periradicular surgery applications of
    computed tomography versus, 400-402
    description of, 400-402, 479
    mandibular canal identification, 398
    maxillary sinus identification, 399
    mechanism of action, 400-401
    scan times, 401-402
  posttreatment disease applications of, 328-329, 402
  presurgical visualization using, 55-60, 55*f*-59*f*
  principles of, 23, 23*f*-24*f*, 37-42
  radiation source for, 24, 24*f*
  risks associated with, 402
  root canal imaging using, 46, 49*f*, 130-131, 283
  root fracture diagnosis using, 51-52, 329, 389
  root perforations detected using, 50-51, 681
  spatial resolution requirements, 42, 43*f*
  traumatic dental injury evaluations, 760-761, 763*f*
  treatment planning uses of, 681
  two-dimensional radiographs and, 24, 25*f*
  vertical root fracture diagnosis using, 45, 46*f*, 389, 808-809
  voxel sizes, 38-39, 42
  voxels, 38-39, 38*f*-40*f*
  wound healing assessments, 58-60
Cone beam volumetric computed tomography, 36-37
Connective tissue, 540
Conscious sedation
  nitrous oxide for, 113
  oral, with triazolam and alprazolam, 113
  for periradicular surgery, 402-404
CONSORT, 709
Consultation letter, 71, 72*f*
Contrast resolution, 35
Core
  amalgam, 826, 833*t*, 840*f*, 844
  cast, 826-827
  cast gold, 844-846, 845*f*
  characteristics of, 825-826
  clinical procedures for, 838-844, 840*f*-843*f*
  composite resin, 826, 833*t*, 842*f*-843*f*
  glass ionomer, 826
  materials used in, 825-826
Coronal access cavity preparation, 335-336, 335*f*, 337*f*-338*f*
Coronal binding, 240
Coronal leakage
  coronal orifice seal for, 315-316
  description of, 159
  treatment failure caused by, 281
Coronal orifice seal, 315-316
Coronal preflaring, 235-236, 236*f*-237*f*
Coronal pulp
  calcification of, 564, 564*f*
  inflammation of, *e*7
  odontoblast layer of, 533
Coronal restorations, 505
Coronal root fractures, 774, 776*f*
Coronally located sinus tracts, 801*f*, 803-805
Corticosteroids
  description of, 118-119
  endodontic pain managed with, 119
  intracranial administration of, 118
  systemic administration of, 119
Cost-effectiveness of treatment procedures, 474
COX-1, 114, 395, 551
COX-2, 114, 395, 551
COX-2 inhibitors
  description of, 115
  prothrombotic adverse effects of, 115-117
  wound healing affected by, 395
COX-inhibiting nitric oxide donators, 122
Crack(s)
  craze lines, 25
  cusps, 794-795
  definition of, 793-794
  diagnostic challenges for, 793
  emergency management of, 719-720
  optical coherence tomography of, 25
  propagation of, 796-797
  split tooth/roots, 26
  staining and transillumination to detect, 20, 21*f*, 719-720, 794, 796*f*
  types of, 25-26
  unobserved trauma as cause of, 793
Cracked tooth
  clinical manifestations of, 795-798
  definition of, 795
  diagnosis of, 795-798
  etiology of, 798
  masticatory forces as cause of, 798
  treatment planning for, 798
Cracked tooth syndrome, 26, 719-720
Craze lines, 25
Crowded teeth, access cavity preparation in, 163-165
Crown
  design of, 812-815
  electric pulp testing in patients with, 18
  fatigue failure of, 823-824
  full, 822-827
  length of, 238
  minimal or no clinical crown, access cavity preparation in patients with, 157-159
  preparation for
    dentinal tubule exposure during, 584-585, 585*f*
    description of, 846
  provisional, 846
  recurrent caries under, 161*f*
  removal of, instruments for, 336, 337*f*
Crown fractures
  in children, *e*14*f*
  description of, 759
  pulpal necrosis secondary to, 764, 764*f*
  vital pulp therapy for
    pulp capping, 766-767
    requirements for, 765-766, 765*f*
Crown infraction, 763
Crown-down technique, for canal enlargement/preparation, 241-243, 267
Crown-root fractures, 759, 773-774
Crown-to-root angulation, 159
CS 9300 3D Extraoral Imaging System, 39-40, 42*f*
C-terminal cross-linking telopeptide of type 1 collagen, 395
C-terminal telopeptide of type 1 collagen chain test, 77
Curettage, in periradicular surgery, 411-412
Cuspal flexure, 593

Cusps
cracked, 794-795
fractured, 794-795, 797*f*
Cvek pulpotomy, 767, 768*f*, *e*1-*e*2
CXCL12, 575
CXCL14, 575
Cyclic fatigue, 726
Cyclooxygenase, 395
CYP2D6, 122
Cysts
formation of, 326
periapical
incidence of, 326
pocket, 326
true, 326
types of, 326
posttreatment disease caused by, 326, 327*f*
types of, 326
Cytokines
biomineralization induced by, *e*2
description of, 551, *e*1
in osteoclast progenitor cell differentiation, 643
in pulpal inflammation, 560
in wound healing, 392, 653, 653*t*
Cytolysin, 634-635
Cytolytic T lymphocytes, 634-635
Cytotoxic T cells, 634-635

D

Danger zones, 241, 268
Deafferentation, 553
Debridement, 654, 666
Deep caries
in mandibular first molars, *e*8*f*
in mandibular second molars, *e*13*f*
occlusal, *e*8*f*
radiographs of, 730*f*, *e*5*f*, *e*16*f*
Deep pain, 699
Denaturing gradient gel electrophoresis, 611
Dendritic cells
in asymptomatic apical periodontitis, 643
in dental pulp, 662
description of, 538-539, 538*f*-539*f*, 575-576, 634
mononuclear, 662
Dens invaginatus, 45-46, 48*f*, 166
Dental anxiety, 87
Dental artery, 556
Dental follicle stem cells, 449, 450*f*
Dental history
definition of, 6
form for, 3*f*, 7*f*
history of present dental problem, 6
interview portion of, 6-9
questioning areas of, 6-9
SOAP format for, 6
software for, 6, 8*f*
Dental implants
osseointegrated, 593
periradicular lesions effect on success of, 594
prosthetic, treatment planning affected by, 77-78
pulpal reactions to placement and function of, 593-594
single-tooth, 85-86
Dental implants *(Continued)*
site assessments for, using cone beam computed tomography, 60
survival rates for, 85-86
after tooth extraction, 332
treatment planning and, 85-86
Dental injection needle, for root canal obstruction removal, 365-366, 368*f*
Dental injury, traumatic. *See* Traumatic dental injury
Dental operating microscope, 131, 132*f*
Dental pain, 5-6
Dental pulp stem cells, 449, 450*f*, 451, 457
Dental resorption, 660
Dental trauma. *See* Traumatic dental injury
Dentin
age-related changes of, 566
carious, *e*6
cervical
bulges of, 156-157, 158*f*
root canal cleaning and shaping objectives for, 211
collagen fibers in, 452-453
composition of, 452-453
cracks in, 593
in endodontically treated teeth, 819
fibrodentin, 574-575, 575*f*
hypersensitivity of
dentinal fluid blockage as cause of, 593
description of, 549-550, 579, 580*f*
lasers for, 590
periodontal scaling as cause of, 592
root planing as cause of, 592
intertubular, 819
lasers as protective measure for, 590
nonvital, 819
in nonvital teeth, 819
peritubular, 585, 585*f*, 819
permeability of
demineralization effects on, 289
dentin sclerosis, 574
description of, 600-601
illustration of, 601*f*
pulpal irritation affected by, 585
variability in, 586
primary, 560-561
probing of, 549
radicular, 811, 811*f*
reactionary, 561, 574-575, 574*f*
remaining thickness of, 584, 763, 812
reparative, 132, 133*f*, 549-550, 560-561, 561*f*-563*f*, 563, 575*f*, *e*2-*e*3
resin-based luting cement adhesion to, 828
resorption of, 643-644
secondary, 560-563, 566
sensitivity of, 547-550
tertiary
description of, 560-562
formation of, 574-575
transparent, 819
ultimate tensile strength of, 819
undermineralized, 585
vital, 819
Dentin bonding, 256
Dentin chips, 314
Dentin matrix, growth factors in, 453*t*
Dentin phosphoprotein, 452-453
Dentin sclerosis
definition of, 574
description of, 566, 593, 600-601, 819
illustration of, 574*f*
Dentin sialoprotein, 451-453
Dentinal bridge, 587*f*
Dentinal fluid, 593
Dentinal pain, 549
Dentinal tubules
A-delta fibers in, 685-686
anatomy of, 548, 600-601
bacterial infiltration through, 591-592, 600-601
cocci bacteria in, 607*f*
Enterococcus faecalis in, 622, 622*f*
exposure of
in crown fracture, 763
during crown preparation, 584-585, 585*f*
fluid movement through, 28, 28*f*, 548*f*
hypersensitivity and, 579, 580*f*
infections of, 604, 607*f*
irrigant penetration in, 257*f*
odontoblast displacement into, 549, 549*f*
Dentinogenesis
odontoblasts in, 535
reparative, 574
tertiary, 573-574
Dentin-pulp complex. *See* Pulp-dentin complex
Dentoalveolar ankylosis, 778
Dentoenamel junction, 536
DENTSPLY, 734
Dermatan sulfate, 541
Desiccation, 583, 586
Detergents, irrigants and, 258
Dexamethasone, for endodontic pain, 119
DGGE. *See* Denaturing gradient gel electrophoresis
Diabetes mellitus
apical periodontitis and, 650-652
hyperglycemia associated with, 652
oral infection risks, 74
pathophysiology of, 74
prevalence of, 73-74
tooth survival after root canal treatment affected by, 509-512
treatment planning affected by, 73-74
wound healing affected by, 654-655
DIAGNOdent, 588-589
Diagnosis
chief complaint, 2-5
data gathering for, 2
definition of, 2
examination for. *See* Examination
imaging for. *See* Imaging
referred pain effects on, 30
stages of, 2
Diaket, 420
Dialysis
drugs affected by, 77
treatment planning affected by, 77
Diamond burs, 148, 148*f*-149*f*, 235, 235*f*
DICOM. *See* Digital Imaging and Communications in Medicine
Differential diagnosis, 2
Difluorodichloromethane, 759, 759*f*, 788
Digital Imaging and Communications in Medicine, 36, 60-61

Digital radiography
advantages of, 60-61
description of, 22-23, 23*f*
diagnostic uses of, 22-23, 23*f*, 36-37
direct, 35-36
file formats for, 36
healing evaluations using, 36
image perception, 60
viewing environment for, 60
working length determination using, 36, 37*f*
Digital subtraction radiography, 43-44
Dihydroxyphenylalanine, 558
1,25-Dihydroxyvitamin D3, 454
Diphenhydramine, 100
Direct composite restorations, 821-822, 835*f*-836*f*
Direct digital radiography, 35-36
Direct pulp capping
bonding agents for, *e*2
calcium hydroxide for, *e*2
cariogenic bacteria survival after, *e*7
definition of, *e*1
hemostatic agents for, 588
of mandibular molars, *e*6*f*
mineral trioxide aggregate for, 587-588, *e*7, *e*8*f*, *e*13*f*
outcomes of, 483, 488*t*-490*t*
partial pulpotomy versus, *e*2
Discoloration, tooth, 821, 821*f*
Disinfectants, 588
Disinfection, in root canal system cleaning and shaping
antimicrobial nanoparticles for, 265
description of, 373-374
of immature tooth, 770
intracanal medications for
bioactive glass, 262
calcium hydroxide. *See* Calcium hydroxide
chlorhexidine. *See* Chlorhexidine
formaldehyde, 261
halogens, 261
lubricants, 262
phenolic preparations, 261
steroids, 261-262
triple-antibiotic paste, 262
irrigants. *See* Irrigants
irrigation. *See* Irrigation
manually activated techniques for, 263
photoactivation, 265
superoxidized water for, 265
syringe delivery for, 262
Displacement trauma, *e*5
Distobuccal angle, 175
Doping, 34-35
Double-flare technique, for canal enlargement/preparation, 243
Drugs
in pregnancy, 74, 74*b*
vasoconstrictor interactions, 94*t*
Dyract, 421
Dystrophic calcification, 565

E

Easy Pneumatic Crown and Bridge Remover, 336
Ecosystem, 606-607, 616-619
ECR. *See* External cervical resorption
EDTA. *See* Ethylenediamine tetraacetic acid
Effective dose, 401-402
Efferent neurons, 542
Eggler Post Remover, 341, 344*f*
EGPR-L/R/U/D hand instruments, 743
EIR. *See* External inflammatory resorption
EI DownPak device, 308, 308*f*
Elastic fibers, 541
Elastin fibers, 541
Electric motors, for rotary instruments, 233
Electric pulp testing
description of, 547
pulp anesthesia determinations using, 95, 95*f*
pulp vitality tests using, 16-19, 18*b*, 18*f*, 92, 578
responses to, 468
Electronic speckle-pattern interferometry, 823
Electronic standards, 36
Electropolishing, 221-222
Electrosurgery, 414
Elements obturation unit, 311
Embryonic stem cells, 448-449
Emergencies
abscess, 715-716
analgesics for, 719
cellulitis, 715-716
classification of, 706
cracked teeth, 719-720
fascial space infections as, 711-715, 712*f*-714*f*
flare-ups, 719
incision for drainage of, 716
irreversible pulpitis, 707-708
leaving teeth open, 716-717, 717*f*-718*f*
reversible pulpitis, 707
symptomatic teeth with previous endodontic treatment, 716
types of, 706
in vital pulp, 707-711
Enamel
cracks in, 602-603
penetration of, 154
Endo Bender pliers, 372-373, 374*f*
Endo Extractor kit, 366-367, 368*f*
EndoActivator, 263, 263*f*
Endocal 10, 358*f*
Endocarditis
bacterial
description of, 73
periradicular surgery in patients with, 396
infective, 73
Endocrown, 822, 830*t*, 839*f*
Endodontic explorer, 150, 150*f*, 164*f*
Endodontic infections
acute apical abscesses as, 614, 617*f*
antibiotics for, 717-719
apical periodontitis. *See* Apical periodontitis
culture analyses of, 610
description of, 599
extraradicular, 650
intraradicular, 612
microorganisms that cause
Archaea, 619
ecology of, 616-619
fungi, 619, 622
geographic influence on, 615-616
viruses, 619
molecular biology methods for studying of, 610, 611*f*
Endodontic infections *(Continued)*
persistent/secondary
bacteria at root obturation stage, 620
definition of, 619
treatment failure and, 619-620
primary, 618-619
pulp vitality in, 82-83
pulpal exposure as cause of, 601
pulpal necrosis caused by, 602
types of, 612
Endodontic lesions
cone beam computed tomography of, 43-44
differential diagnosis of, 43-44
Endodontic pain
dexamethasone for, 119
management of, 120
Endodontic spoon, 150, 150*f*
Endodontic surgery
goal of, 467
guided tissue regeneration and, 426-435
healing after, 84-85
indications for, 83, 84*f*
outcomes of, 85
referral for, 87
traditional root-end surgery versus, 85
Endodontic treatment
extraoral time less than 60 minutes, 787-788
failure of, 44-45, 45*f*
goal of, 599
traumatic dental injury, 787-788
Endodontically treated teeth
aesthetic changes in, 821
Candida albicans in, 622
dentin structure in, 819
discoloration of, 821, 821*f*
Enterococcus faecalis in, 620-622, 621*f*-622*f*
features of, 818-821
fracture resistance of, 819-821
microbiota in, 620-622, 621*f*-623*f*
periodontal evaluation of, 829
provisional crowns for, 846
restorations for
adhesive systems, 844
aesthetic evaluation and requirements, 829-830
biomechanical evaluation, 829
clinical procedures, 834-846, 835*f*-843*f*, 845*f*
direct composite, 821-822, 835*f*-836*f*
endocrown, 822, 830*t*, 839*f*
ferrule, 822-823
foundation, 822-823
full crowns, 822-827
indirect, 822
luting cements used with, 827-828
onlays and overlays, 822, 830*t*, 838*f*
partial, 844
periodontal evaluation, 829
post and core, 824-827, 838-844, 840*f*-843*f*
pretreatment evaluation, 828-830, 829*f*, 830*t*
provisional crowns, 846
resin composite, 821-822
in structurally compromised anterior teeth, 834
in structurally compromised posterior teeth, 834

Endodontically treated teeth *(Continued)*
in structurally sound anterior teeth, 830
tooth preparation for, 838
veneer, 830*t*, 837*f*
tooth stiffness of, 819-821
vertical root fractures in, 800, 809-810
Endodontics
anesthesia for
infiltrations, 109
intraligamentary, 110-111
intrapulpal injection, 110-111
local, 109-111
anxiety effects on, 87
case selection for, 86-88
description of, 30
factors that affect, 86-88
goal of, 467
scheduling considerations, 88
Endo-Eze file system, 232
Endogenous opioid peptides, 554
EndoHandles, 146, 146*f*
EndoSequence Root Repair Manual, 422
EndoSequence rotary instrument, 231
Endosolv-E, 351*f*, 356
Endosolv-R, 356
Endotec II device, 308, 308*f*
Endothelial adhesion molecules, 640*t*
Endothelial cells
in apical periodontitis, 638
in wound healing, 392-393
EndoTwinn instrument, 308
End-stage renal disease, 77
Engine-driven instruments, for gutta-percha removal, 350
Enterococcus faecalis
calcium hydroxide resistance of, 622
in dentinal tubules, 622, 622*f*
description of, 388, 499-504, 739, *e*3
in endodontically treated teeth, 620-622, 621*f*-622*f*
in root canals, 632
sodium hypochlorite for, 253
Epidermal growth factor, 653*t*
Epinephrine
in cardiovascular disease patients, 72-73
cardiovascular effects of, 93
hemostatic uses of, 413-414
Epinephrine pellets, 413-414
Epiphany Obturation System, 351*f*
Epithelial cell rests of Malassez
in asymptomatic apical periodontitis, 644, 645*f*, 647
description of, 326, 631
Epithelial cells, 392
Epithelial stem cells, 449
Epoxy resin sealers, 292, 293*f*
Epstein-Barr virus, 619
Er:YAG laser, 588-589, 590*f*
Ethylenediamine tetraacetic acid
biocompatibility of, 289
description of, 234, 257
endodontic applications of, 257-258
history of, 257
mode of action, 257
root-end surface modification using, 416
smear layer removal using, 289, 416
sodium hypochlorite and, 258
Eucalyptol, for gutta-percha removal, 347-348, 348*f*
Eugenol, 827
Examination
bite test, 19-20
extraoral, 9-11, 9*f*-12*f*
intraoral. *See* Intraoral examination
periodontal, 15
pulp testing. *See* Pulp testing
Explorer
endodontic, 150, 150*f*, 164*f*
operative, 150*f*
Extendo bur, 149, 149*f*
External cervical resorption
clinical features of, 667-668, 668*f*-669*f*
cone beam computed tomography of, 670*f*-671*f*, 672
definition of, 667
etiology of, 667
histologic appearance of, 667, 668*f*
internal root resorption versus, 677, 678*f*-679*f*
management of, 672-675
nonsurgical treatment of, 675
pathogenesis of, 667
predisposing factors for, 667
radiographic features of, 668-672, 668*f*-673*f*
traumatic dental injury as cause of, 667
treatment of, 672-675
External inflammatory resorption
apical, 663*f*-664*f*
clinical features of, 665
cone beam computed tomography of, 666
diagnosis of, 662, 784
etiology of, 665
follow-up of, 666
histologic appearance of, 665
illustration of, 663*f*-664*f*
imaging of, 665-666
luxation as cause of, 662, 664-665
management of, 666
pathogenesis of, 665
pink spots associated with, 784, 784*f*
prevalence of, 662
prognosis for, 666
progression of, 665
radiographic features of, 662, 665-666, 783, 784*f*
subepithelial, 784
surface resorption versus, 665
treatment of, 662, 666
External root resorption
apical neurovascular supply damage as cause of, 779-781, 779*f*-782*f*
in asymptomatic apical periodontitis, 645
cervical. *See* External cervical resorption
cone beam computed tomography of, 52-55, 54*f*
description of, 777-778
diagnosis of, 783-784, 783*f*-784*f*
external root surface injury as cause of, 778-779
general histologic features of, 660-662
inflammatory type of. *See* External inflammatory resorption
luxation injury as cause of, 778-781
pulpal space infection as cause of, 779-781, 780*f*
External root resorption *(Continued)*
radiographic features of, 783, 783*f*-784*f*
treatment of, 779
Extracellular matrix
connective tissue in, 540
glycoproteins of, 540
of periodontal ligament, 630-631
Extracellular polymeric substance, 607
Extraction-replantation, 381, 382*f*
Extractor System, 360, 362*f*
Extraoral examination, 9-11, 9*f*-12*f*
Extraradicular biofilms, 609
Extraradicular endodontic infection, 650
Extraradicular infections
description of, 612, 622-624
development of, 623-624, 624*f*
intraradicular infection and, 624
posttreatment disease caused by, 326

F

Facial pain, 684
Facial root fractures, 775*f*
Facial swelling
buccal space, 9-10, 9*f*-10*f*
canine space, 9, 9*f*
examination of, 9-11, 9*f*-12*f*
odontogenic causes of, 9
submental space, 10, 10*f*
Factitious pain, 696-697
Famotidine, 117
Fascial spaces
anatomy of, 712, 712*f*-714*f*
infections of, 711-715, 712*f*-714*f*
Fatigue failure, 823-824
Fatigue root fracture, 798
Ferric sulfate, for hemostasis, 413
Ferrule, 822-823
Fetal hypoxia, 74-75
Fiber posts, 825, 834
Fiber-reinforced composite post, 341-343
Fibroblast(s)
in asymptomatic apical periodontitis, 644-645
in cell-rich zone, 537
description of, 537-538
in wound healing, 392
Fibroblast growth factor, 653*t*
Fibrodentin, 563, 574-575, 575*f*
Fibrogenesis, 393
Fibronectin, 540
Field of view, of cone beam computed tomography, 39-42, 41*f*
File(s)
Endo-Eze, 232
flute of, 216, 218*f*
GT, 229
GTX, 229
history of, 223
ISO-normed, 218
K-, 164*f*, 213-214, 223
LightSpeed, 227, 244-245, 246*f*
nickel-titanium, 240
nickel-titanium rotary, 214-216, 218*f*-219*f*
patency, 236-237
pitch angle of, 216-217
Profile Vortex, 231
RaCe, 231
retrieval kit for, 729-731, 732*f*

File(s) *(Continued)*
self-adjusting, 226, 228*f*, 232, 245
separated, 365, 367*f*-368*f*, 726-727, 728*f*-729*f*
stainless steel, 223, 224*f*, 237*f*, 246-247
ultrasonically activated, 356, 357*f*
Finger spreaders, 302*f*-303*f*, 303-304
Fissure carbide burs, 148, 148*f*
Fistula, 12
Fixed partial dentures
fabrication of, abutment teeth affected by, 332*f*
after tooth extraction, 332
Flare-ups, 719
Flexible probes, 802-803, 806*f*
FlexMaster, 230-231
Flexural fracture, of instruments, 266
Flute of file, 216, 218*f*
Forceps, for crown removal, 336, 337*f*
Foreign bodies
in open teeth, 717, 718*f*
periapical infection caused by, 632
Foreign body reaction
definition of, 643
posttreatment disease caused by, 326
Formaldehyde, 261
Formocresol
dentin tensile strength affected by, 819
description of, 261
Formocresol pulpotomy, *e2*
Foundation restorations
cast post and core as, 826-827, 830
direct, 824-826
general considerations for, 822-823
indirect, 826-827
luting cements as. *See* Luting cements
under full crowns, 844-846, 845*f*
FracFinder, 19-20
Fracture(s)
crown. *See* Crown fractures
crown-root, 759, 773-774
definition of, 793-794
diagnostic challenges for, 793
magnification to detect, 797-798
mechanics of, 793-794
pulpal necrosis induced by, 797*f*
root. *See* Root fractures
symptoms of, 25
transillumination for detection of, 20, 21*f*
treatment of, 25
unobserved trauma as cause of, 793
vertical root. *See* Vertical root fractures
Fractured cusps, 794-795, 797*f*
Full coverage restorations, 85
Full crowns
for endodontically treated teeth, 822-827
foundation restoration underneath, 844-846, 845*f*
Full pulpotomy, 494*t*, 767-768
Full-thickness flap, in periradicular surgery, 410*f*
Functional retention, 479
Fungal cells, 604, 605*f*
Fungi, 619, 622
Furanocoumarins, 555-556
Furcal perforations, 378*f*
Furcation canals, 133, 133*f*
Furcation defects
case example of, 428*f*
causes of, 15
cleaning of, 377
recording of, 15*b*
repair of, 377

G

G protein-coupled receptors, 92
Gap junctions, 533-534
Gates-Glidden burs, 152, 156-157, 225*f*, 235, 236*f*
description of, 152, 156-157, 225*f*, 235, 236*f*
silver point removal using, 360
Gates-Glidden drills
description of, 225, 225*f*
gutta-percha removal using, 347
radicular access using, 365
Gelfoam, 413
Genetic polymorphisms, 651-652
GentleWave system, 264-265, 264*f*
Geristore, 376-377, 377*f*, 421
Gingiva examination, 11
Gingival-derived mesenchymal stem cells, 449
Glass ionomer(s)
core, 826
obturation use of, 292, 292*f*-293*f*
resin-modified, 826
as sealers, 292, 292*f*-293*f*
Glass ionomer cements
luting, 827
pulpal responses to, 586-587
as root-end filling material, 420
Glass ionomer-based endodontic sealer
description of, 504-505
gutta-percha and, 349
Glial cell line-derived neurotrophic factor, 552
Glial cells, 554
Glide path, 243
Glucocorticoids, 395
Glycoproteins, 540
Glycosaminoglycans, 541
Gonon Post Removing System, 340, 342*f*
Granulation tissue, in wound healing, 392-393
Granulocyte-macrophage colony-stimulating factor, 575-576
Granuloma
definition of, 642-643
periapical, 642-646
Granulomatous inflammation, 642-643
Granzymes, 634-635
Greater palatine foramen, 399*f*
Greater Taper nickel-titanium hand files, 372-373, 374*f*
Growth factors
in cementum, 630, 653
in dentin matrix, 453*t*
in odontoblast-like cell differentiation, 454*t*
regenerative endodontics use of, 452-455, 453*t*-454*t*
sequestration of, 453
in wound healing, 653, 653*t*
Growth hormone, 453
GT file, 229, 372-373
GT obturators, 311-312, 311*f*
GTR. *See* Guided tissue regeneration
GTX file, 229
Guided tissue regeneration
case examples of, 427*f*-433*f*
membranes used in, 433*f*-434*f*, 434, 435*t*
in periradicular surgery, 426-435, 427*f*-433*f*
GuttaCore, 312, 355-356
GuttaFlow, 294
GuttaFlow2, 294
Gutta-percha
advantages of, 347
α form of, 298, 311
carrier-based, 311-313
disadvantages of, 297
glass ionomer-based endodontic sealer used with, 349
heat application to, 347
heated, for pulp testing, 17
lateral compaction of, 304
warm, 308-309, 308*f*
modified, 355
nickel-titanium files for removal of, 742
obturation use of, 297-299, 298*f*-299*f*, 304, 355, 681
overextended, removal of, 349, 349*f*, 742
removal of, during nonsurgical retreatment, 347-351, 347*f*-351*f*, 742, 745*f*
Resilon and, comparison between, 300
rotary systems for removal of, 349-350, 350*f*
solvents used to remove, 347-349, 348*f*
thermoplastic injection techniques for, 309-311, 309*f*-310*f*
warm lateral compaction of, 308-309, 308*f*
warm vertical compaction of, 304, 305*f*-306*f*
Gutta-percha cones, 298, 298*f*-299*f*
Gutta-percha points, 299, 299*f*
GyroTip, 341-343, 345*f*

H

Halo radiolucency, 802, 804*f*, 805-806
Halogens, 261
Halothane, for gutta-percha removal, 347-348, 348*f*
Handpieces
for access cavity preparation, 147-148
heat exhaustion from, 411, 411*f*
Hank's Balanced Salt Solution, 785
Hard tissue
apical barrier, 770-773, 770*f*-772*f*
healing of, 393-394
HBPT, 258-259
Headaches, 691-692
Healing, wound. *See* Wound healing
Heart failure, acute, 73
Heart valves, artificial, 73
Heat
bone damage caused by, 411
damage caused by, 370-371
generation of
by ultrasonic instruments, 346, 346*f*, 370-371
for nonsurgical retreatment, 370-371
pulp testing using, 16-17, 17*f*
Heavily restored teeth, access cavity preparation in, 159-162, 161*f*-162*f*
Hedström files
description of, 221*f*, 223-224, 349, 349*f*
for loosening of separated instruments in canal, 365

Hedström files *(Continued)*
separated, 726-727
silver point removal using, 360, 360*f*
Helical angle, 216, 219*f*
Hemostasis
agents for, *e*9
bone wax for, 413
calcium sulfate for, 413
collagen-based materials for, 412, 413*f*
epinephrine pellets for, 413-414
ferric sulfate for, 413
Gelfoam for, 413
in periradicular surgery, 412-414, 437
sodium hypochlorite for, *e*7, *e*21*f*, *e*23*f*-*e*24*f*
Surgicel for, 412-413
Heparin
low-molecular-weight, 397
periradicular surgery in patients receiving, 397
Hermann, B. W., 447
Hermetic seal, 283
HERO 642, 230
Hero Shaper, 230
Herpes zoster, 693-694, 694*f*
Herpesvirus, 619
Hertwig's epithelial root sheath, 132-133, 199-201, 631, 653
Hess, Walter H., 213, 216*f*
Heterotopic pain, 688*b*, 690, 700
Heterotopic symptom, 684
High-resolution magnetic resonance imaging, 48
Histamine, 638
History of present dental problem, 6
HIV. *See* Human immunodeficiency virus
Hodgkin disease, 73
Höehl cell layer, *e*1
Holism, 607
Hormones, in hard-tissue healing, 394
Horner syndrome, 700-702
HotShot delivery systems, 311
Howship lacunae, 660-662, 661*f*, 665
H-type instruments, 223-224, 224*f*
Human cytomegalovirus, 619
Human immunodeficiency virus, 619
CD4+ cell count in, 77
oral manifestations of, 77
Hyaluronan, 541
Hyaluronic acid, 541
Hyaluronidase, 99
Hydrogels, as scaffolds, 455
Hydrophilic resins, *e*5-*e*6
Hyperalgesia, 550-551, 550*t*, 554
secondary, 687, 690-691
Hyperglycemia, 652
Hyperplastic pulpitis, 576, 577*f*
Hypersensitivity, dentin, 549-550, 579, 580*f*

I

Iatrogenic events
cervicofacial subcutaneous emphysema, 748-750, 749*f*-750*f*
definition of, 722
inferior alveolar nerve injury
causes of, 745-748, 745*b*, 746*f*-748*f*
complications of, 748, 749*f*
cone-beam computed tomography of, 748
description of, 745-746
Iatrogenic events *(Continued)*
nonsurgical causes of, 746-747, 746*f*-747*f*
paresthesia caused by, 747-748
prevention of, 747
prognosis for, 748
sealer cement effects on, 745-746
surgical causes of, 747-748, 748*f*
third molar extractions as cause of, 746
instrument separation
causes of, 725-726
chemical removal, 726
description of, 724-725
files, 365, 367*f*-368*f*, 726-727, 728*f*
improper use as cause of, 725-726
management of, 726-727
in mandibular molars, 726
prognosis for, 733
radiograph of, 725*f*-726*f*
removal techniques, 727-733, 728*f*-732*f*
surgical approaches for management of, 731-733
ultrasonic instruments for removal after, 727
ledge formation
bypassing of, 734-737, 735*f*-737*f*
causes of, 733, 734*b*
complications of, 737
description of, 733
hand instruments for bypassing/removal of, 734, 735*f*
incidence of, 733
management of, 733-734
prevention of, 737
prognosis for, 737
rotary instruments for bypassing of, 737
ultrasonic tips for removal of, 736-737, 737*f*-740*f*
nonsurgical perforations, 737-739
root canal filling material radicular extrusion
description of, 739
illustration of, 743*f*
management of, 742-745
obturation as cause of, 740, 742-744
tissue damage caused by, 744-745
sinus perforation, 745
sodium hypochlorite, 722-724, 723*f*-724*f*
tips for reducing, 722
Ibuprofen, 436, 719
I-CAT, 38*f*
Illumination, periapical healing affected by, 497
Images
characteristics and processing of, 35-36
postprocessing of, 35-36
Imaging
cone beam computed tomography. *See* Cone beam computed tomography
conventional radiographs. *See* Radiographs
digital radiography, 22-23, 23*f*
magnetic resonance imaging, 24
three-dimensional, 36-37, 38*f*
Immature teeth
apexification of, 769-773, 770*f*-772*f*
apical closure, *e*1
periradicular periodontitis in, 773*f*
pulp necrosis in, 314-315
pulpectomy contraindications in, *e*5
revascularization of, 780*f*
root canal treatment in, 764
Immediate obturation, 314-315, 315*f*
Immune response
adaptive, 634-635, 636*f*, 642-643
innate, 633-634, 633*t*, *e*1
specific, 634-635, 636*f*
Immunity
adaptive, 633*t*, 634, 636*f*, 642-643
innate, 633*t*, 636*f*
Immunoglobulins, 634
Implants, dental
osseointegrated, 593
periradicular lesions effect on success of, 594
prosthetic, treatment planning affected by, 77-78
pulpal reactions to placement and function of, 593-594
single-tooth, 85-86
site assessments for, using cone beam computed tomography, 60
survival rates for, 85-86
after tooth extraction, 332
treatment planning and, 85-86
Incisal bevel, 152*f*
Incision and drainage procedures
for abscesses, 716
anesthesia for, 113-114
Incisive nerve block, 99
Indirect pulp capping
boundary point for, *e*2
case selection for, *e*2
definition of, *e*2
indications for, *e*2
outcomes of, 480-482, 485*f*, 486*t*, 491*f*
stepwise excavation, 480
Induced pluripotent stem cells, 448-449
Infections
apical, 632
buccal space, 9-10, 9*f*-10*f*
canine space, 9, 9*f*
dentinal tubules, 604, 607*f*
endodontic. *See* Endodontic infections
extraradicular
description of, 612, 622-624
development of, 623-624, 624*f*
intraradicular infection and, 624
posttreatment disease caused by, 326
fascial space, 711-715, 712*f*-714*f*
host–parasite conflict in, 632-633
innate immune response to, 633-634, 633*t*
intraradicular
apical periodontitis caused by, 614*f*, 622-623
bacteria that cause, 614, 615*f*-617*f*
definition of, 612
extraradicular infection and, 624
microbial composition and diversity of, 614, 614*f*
primary, 614-619
persistent, 612
primary, 612
pulpal space, 779-780, 780*f*
secondary, 612
Infective endocarditis, 73
Inferior alveolar nerve, 46-48
Inferior alveolar nerve block
bupivacaine for, 92-93, 98, 98*f*
duration of action, 97

Inferior alveolar nerve block *(Continued)*
enhancement methods for, 101, 101*f*-102*f*
epinephrine concentrations in
2% lidocaine with 1:100,000 epinephrine, 96
4% prilocaine with 1:200,000 epinephrine, 97
articaine with 1:100,000 epinephrine, 97-98
increasing of, 99
failure of
definition of, 96
factors involved in, 100-101
inaccurate injection as cause of, 100
mylohyoid nerve block, 100, 100*f*
needle bevel as cause of, 100-101
needle deflection as cause of, 100
theories on, 101
Gow-Gates technique for, 99
injection sites for, 99
intraligamentary anesthesia with, 101
intraosseous anesthesia with, 101
lidocaine for
2%, with 1:100,000 epinephrine, 96, 101
buffered, 98-99
mannitol and, 99
of mandibular anterior teeth, 112
mannitol for, 99
noncontinuous anesthesia, 96
onset of action, 88, 96-97
preemptive nonsteroidal anti-inflammatory drugs, 113
pulpal anesthesia onset using, 96-97, 97*f*
ropivacaine for, 98
solutions for
2% lidocaine with 1:100,000 epinephrine, 96, 101
2% mepivacaine with 1:20,000 levonordefrin, 97, 103
3% mepivacaine, 97
4% prilocaine, 97
4% prilocaine with 1:200,000 epinephrine, 97
articaine with 1:100,000 epinephrine, 97-98
hyaluronidase added to, 99
increasing the volume of, 99
success of, 96
success of, methods of increasing, 99
in symptomatic irreversible pulpitis, 109
Inferior alveolar nerve injury
causes of, 745-748, 745*b*, 746*f*-748*f*
complications of, 748, 749*f*
cone-beam computed tomography of, 748
description of, 745-746
nonsurgical causes of, 746-747, 746*f*-747*f*
paresthesia caused by, 747-748
prevention of, 747
prognosis for, 748
sealer cement effects on, 745-746
surgical causes of, 747-748, 748*f*
third molar extractions as cause of, 437, 746
Inflammation
acute, 475
apical, 632
chronic, 475
coronal pulp, *e7*
mediators of, 551, 554-555, 647
Inflammation *(Continued)*
neurogenic, 635, 636*f*
percussion testing for, 14-15
periapical, 632
pulpal. *See* Pulpal inflammation
of pulpal interstitium, 541
vascular permeability in, 560
Inflammatory periapical progenitor cells, 449
Inflammatory response
to calcium hydroxide, 587*f*
to caries, 575, 575*f*
mediators of, 639-641, 640*t*
Inflammatory root resorption, 780*f*, 788
Informed consent, for periradicular surgery, 402
Infraorbital nerve block, 103
Infraorbital space, 712*f*-714*f*, 715
Innate immune response, 633-634, 633*t*
Innate immunity, 633*t*
Instrument Removal System, 369-370, 370*f*
Instruments
apical pressure during use of, 362
for crown removal, 336, 337*f*
cutting angle of, 216
cutting edge of, 216
discarding of, 362
engine-driven, 225-235, 226*t*, 228*f*-229*f*
fracture of
cone beam computed tomography of, 48-49, 51*f*
cyclic fatigue as cause of, 726
description of, 226, 227*f*, 266-267, 363
incidence of, 48-49
torsional fatigue as cause of, 726
hand, separation of, 361-362
helical angle of, 216, 219*f*
improper use of, 362
irrigants and, 253
ISO-normed, 217-219, 220*f*
nickel-titanium, 214-216, 218*f*-219*f*, 221
for obturation
continuous wave compaction technique, 305-306, 307*f*
warm vertical compaction technique, 304, 305*f*
periradicular surgery, 405, 405*f*-407*f*
post removal using, 339-341
rake angle of, 216, 219*f*
reciprocal motions with, 726
root canal system cleaning and shaping. *See* Root canal system cleaning and shaping, instruments for
rotary, 226-235, 226*t*, 228*f*-229*f*
separated
causes of, 361-363, 364*f*, 725-726
chemical removal, 726
dental injection needle for removal of, 365-366, 368*f*
description of, 724-725
Endo Extractor kit for removal of, 366-367, 368*f*, 729-731
files, 365, 367*f*-368*f*, 726-727, 728*f*
Hedstrom files for removal of, 365, 367*f*
illustration of, 366*f*
improper use as cause of, 362, 725-726
Instrument Removal System for removal of, 369-370, 370*f*
management of, 726-727
in mandibular molars, 726
Instruments *(Continued)*
Masserann technique for removal of, 367, 726-727
nickel-titanium instruments, 727-728
operating microscope used in removal of, 369, 369*f*
prevention of, 363
prognosis for, 364-365, 733
radiograph of, 725*f*-726*f*
removal techniques for, 365-370, 365*f*-367*f*, 727-733, 728*f*-732*f*
S.I.R. System for retrieval of, 370
surgical approaches for management of, 731-733
ultrasonic instruments for removal of, 365, 366*f*, 727
taper of, 217, 220*f*
tip design of, 214-216, 218*f*
Intentional replantation, 381, 436, 436*f*
Intercellular adhesion molecules, 638-639
Interferon-τ, 641
Interleukin-1, 641, 651
Interleukin-8, 652
Interleukin-12, 641
Internal ankylosis, 566, 566*f*
Internal root resorption
characteristics of, 781
chemicomechanical debridement of root canal for, 679
clinical features of, 677
clinical manifestations of, 782
cone beam computed tomography of, 52-55, 54*f*, 677
definition of, 675
description of, 778
diagnosis of, 677, 784
early, 674*f*
etiology of, 675, 781-782
external cervical resorption versus, 677, 678*f*-679*f*
histologic appearance of, 674*f*, 675-677, 676*f*, 782, 782*f*
historical description of, 660
management of, 677, 679, 681
of maxillary lateral incisor, 676*f*
obturation for, 681
odontoblasts in, 675-676
pathogenesis of, 675, 676*f*
pulp tissue in, 675, 676*f*
radiographic features of, 671, 676*f*, 677, 680*f*, 781*f*-783*f*, 782
in root fractures, 776*f*
root perforations associated with, 681
subepithelial, 784
tissue destruction caused by, 681
treatment of, 782-783
in vital pulp, 784
International Association of Dental Traumatology, 760
International Headache Society, 691
International Standards Organization norms, 217-219, 220*f*
Intersocietal Accreditation Commission, 62
Interstitial fluid, 558
Interstitium, pulpal, 540-541, 540*f*
Intertubular dentin, 819
Interview, 6-9

Intracanal medicaments
  calcium hydroxide, 459-460
  triple antibiotic paste, 459-460, 459*f*
Intracoronal restorations, 798, 838
Intraligamentary anesthesia
  amount of solution delivered, 108
  anesthetic solutions for, 107
  avulsion after, 109
  back-pressure for, 107
  computer-controlled local anesthetic delivery system for, 108, 108*f*, 110
  description of, 101
  discomfort during, 108
  duration of anesthesia with, 108
  endodontic uses of, 110-111
  indications for, 107
  mechanism of action, 107
  onset of anesthesia, 108
  periodontal safety with, 108
  postoperative discomfort caused by, 108
  primary teeth safety with, 109
  pulpal safety with, 109
  success of, 107
  systemic effects of, 108
Intraoral examination
  mobility, 15, 15*b*, 15*f*
  palpation, 13
  percussion, 14*f*, 15, 19
  periodontium, 15
  sinus tracts, 12-13, 14*f*
  soft tissue, 11
  swelling, 11, 12*f*-13*f*
Intraoral radiographs, 20-22, 22*f*
Intraosseous anesthesia
  alveolar mucosa injection of, 106
  attached gingiva injection of, 106
  bupivacaine for, 106
  definition of, 104
  description of, 101
  discomfort associated with, 106
  duration of anesthesia with, 104, 105*f*, 106
  endodontic uses of, 109-110
  failure of, 105
  heart rate increase after, 106-107
  injection pain with, 110
  injection site locations, 106
  lidocaine levels in plasma after, 107
  for mandibular posterior teeth, 111
  medical considerations for, 107
  onset of anesthesia, 106
  perforator breakage with, 105-106
  postoperative discomfort from, 107
  repeating of, 106
  Stabident system for, 104, 105*f*
  success with, 105
  X-Tip delivery system for, 104-106, 105*f*, 110
Intrapulpal injection, for endodontic anesthesia, 110-111, 581
Intraradicular biofilms, 609
Intraradicular infection
  apical periodontitis caused by, 614*f*, 622-623
  bacteria that cause, 614, 615*f*-617*f*
  definition of, 612
  extraradicular infection and, 624
  microbial composition and diversity of, 614, 614*f*
  primary, 614-619
Intrasulcular incision, for periradicular surgery access, 409, 409*f*
Intrusive luxation, 777
Invasive cervical resorption, 54*f*, 667
Iodine potassium iodide, 260-261
Irreversible pulpitis
  anesthesia in
    preemptive nonsteroidal anti-inflammatory drugs, 113
    in symptomatic patients, 111
  asymptomatic
    definition of, 707
    description of, 28-29
    in mandibular posterior teeth, 113
    opioid receptors and, 578-579
    symptomatic irreversible pulpitis versus, 109
  corticosteroids for, 119
  definition of, 28-29
  diagnosis of, 707, *e*5
  emergency management of, 707-708
  maxillary molar infiltration in, 109
  pain reduction in, 112-113
  pulpotomy for, 112
  radiographic findings, 75*f*
  reversible pulpitis and
    differential diagnosis of, *e*5
    sodium hypochlorite for differentiation of, *e*5
  single-visit endodontic treatment for, 709
  sodium hypochlorite for diagnosis of, *e*5
  symptomatic
    anesthesia in, 111
    asymptomatic irreversible pulpitis versus, 109
    definition of, 707
    description of, 28-29
    inferior alveolar nerve block in, 109
    management of, 708
    in mandibular posterior teeth, 113
Irrigants
  benefits of, 251*b*
  biologic function of, 249
  BioPure MTAD, 258-259, 258*f*
  chlorhexidine
    allergic reactions to, 256
    antibacterial efficacy of, 255
    before periradicular surgery, 402-404
    calcium hydroxide with, 256
    cytotoxicity of, 254-255
    as decalcifying agent, 256-257
    dentin bonding and, 256
    as endodontic irrigant, 255
    history of, 254
    indications for, 404
    as intracanal medicine, 255
    mode of action, 254
    molecular structure of, 254, 254*f*
    sodium hypochlorite and, 256*f*
    substantivity of, 254
  dentinal tubule penetration of, 257*f*
  detergents added to, 258
  ethylenediamine tetraacetic acid
    description of, 234, 257
    endodontic applications of, 257-258
    history of, 257
    mode of action, 257
    sodium hypochlorite and, 258
Irrigants *(Continued)*
  HBPT, 258-259
  instrumentation with, 253
  iodine potassium iodide, 260-261
  optimal properties of, 250-251, 251*b*
  QMiX, 259-260
  smear layer effects on action of, 288
  sodium hypochlorite
    allergic reactions to, 252
    calcium hydroxide and, 679
    "champagne bubble" test, 131, 163
    chemicomechanical debridement of root canal using, 679
    chlorhexidine and, 256*f*
    complications of, 722-724, 723*f*-724*f*
    concentrations of, 253
    description of, 770, 818-819
    ethylenediamine tetra-acetic acid and, 258
    extrusion of, 723-724, 723*f*
    heating of syringes for, 252-253, 253*f*
    hemostasis using, *e*7, *e*21*f*, *e*23*f*-*e*24*f*
    history of, 251
    hypersensitivity to, 252
    hypochlorous acid formation, 251-252
    iatrogenic events caused by, 722-724, 723*f*-724*f*
    mode of action, 251-252, 252*f*
    neutralization reaction, 251
    pH of, 252
    pulp tissue dissolution capacity of, 253
    pulpitis diagnosis using, *e*5
    root canal retreatment success affected by, 498-499
    saponification reaction, 251
    temperature effects on, 252-253, 252*f*-253*f*
    time course of, 253
    toxicity of, 253-254, 254*f*
  syringe delivery of, 262
  Tetraclean, 258-259
Irrigation, of root canal
  efficiency of, 249, 250*f*
  GentleWave system, 264-265, 264*f*
  hydrodynamics of, 249-250, 250*f*-251*f*
  irrigants for. *See* Irrigants
  laser-activated, 264*f*, 265
  manually activated, 263
  needles used in
    diameter and bevel of, 249-250, 251*f*
    illustration of, 251*f*
    types of, 262
  negative apical pressure for, 264, 264*f*
  objectives of, 249
  passive ultrasonic, 263-264
  protocol for, 269
  root canal shape effects on, 250*f*
  Safety-Irrigator for, 264, 264*f*
  sonically activated, 263, 263*f*
Irrigation pressure, 249-250
Ischemic heart disease, vasoconstrictors in patients with, 72-73
ISO norms, 217-219, 220*f*
Isolation techniques, 497
Isthmuses, 141, 141*f*

J

Jaw
  metastases to, 75, 75*f*
  osteonecrosis of
    medication-related, 76-77, 76*b*
    radiation therapy as cause of, 655
JS Post Extractor, 341
J-shaped radiolucency, 802, 804*f*, 805-806

K

K3, 229-230
K-files
  for coronal preflaring, 235
  description of, 164*f*, 213-214, 223
  pitch of, 216-217
  for root canal enlargement, 246
  stainless steel, 223, 224*f*
KY Pliers, for crown removal, 336, 337*f*

L

Lamellar bone, 393-394
Lamina limitans, 535-536
Largo Bur, 341-343
Laser(s)
  caries prevention, diagnosis, and treatment using, 588-590, 589*f*
  CO_2, 588
  dentin hypersensitivity treated with, 590
  Er:YAG, 588-589, 590*f*
  GaA1As, 590
  Nd:YAG, 588-589, 589*f*
  pulpal reactions to, 588-590
  pulpotomy applications of, 589-590
  types of, 588
  wavelengths of, 588*f*
Laser Doppler flowmetry, for pulpal blood flow assessments, 19, 759-760, 760*f*
Laser-activated irrigation, 264*f*, 265
Lateral compaction obturation, 302-304, 302*f*-303*f*
Lateral pharyngeal space, 715
Latex allergies, 93-94
Le Fort I osteotomy, 592
Leaving teeth open, 716-717, 717*f*-718*f*
Ledermix, 118, 261-262, 586, 779
Ledges
  bypassing of, 734-737, 735*f*-737*f*
  causes of, 733, 734*b*
  complications of, 737
  description of, 268, 371, 733
  hand instruments for bypassing/removal of, 734, 735*f*
  incidence of, 733
  management of, 733-734
  prevention of, 737
  prognosis for, 737
  rotary instruments for bypassing of, 737
  ultrasonic tips for removal of, 736-737, 737*f*-740*f*
Lentulo spiral
  calcium hydroxide application using, 770, 770*f*
  for sealer placement in obturation, 296, 297*f*
Levonordefrin
  2% mepivacaine with 1:20,000 levonordefrin, for inferior alveolar nerve block, 97, 103
Lidocaine
  2%, with 1:100,000 epinephrine, 96, 101, 111
  buffered, 98-99
  infiltrations of, 99
  meperidine added to, 100
Light-curing luting cements, 827-828
LightSpeed file, 227, 244-245, 246*f*, 266
Lingual shoulder
  in anterior teeth, 152, 152*f*
  in maxillary canine, 169
  in maxillary central incisors, 165
  removal of, 152, 152*f*
Lip lacerations, 787
Lipopolysaccharides
  calcium hydroxide effects on, 260
  description of, 454
  nerve fibers stimulated by, 632
  odontoblast exposure to, 576*f*
  Toll-like receptor 2 expression affected by, 575
  vascular endothelial growth factor affected by, 560
  as virulence factor, 604
Lipoteichoic acid, 633
Local anesthesia/anesthetics
  adverse effects of, 93-94
  allergic reactions to, 93-94
  antagonism of, 96
  articaine, 97-99
  buffered lidocaine for, 98-99
  bupivacaine for, 98, 98*f*
  carbonated anesthetic solutions, 100
  cardiovascular reactions to, 93
  computer-controlled delivery system for, 100
  confirming of, 95, 95*f*
  description of, 388
  diphenhydramine for, 100
  for endodontics, 109-111
  failure to achieve, 96
  G protein-coupled receptor regulation of, 92
  Gow-Gates technique, 99
  incisive nerve block for, 99
  inferior alveolar nerve block for. *See* Inferior alveolar nerve block
  infiltrations, 104
    0.5% bupivacaine with epinephrine, 103
    3% mepivacaine, 102, 102*f*
    4% prilocaine, 102-103
    articaine, 104
    lidocaine, 99, 104
  injection sites for, 99
  intraosseous
    alveolar mucosa injection of, 106
    attached gingiva injection of, 106
    bupivacaine for, 106
    definition of, 104
    description of, 101
    discomfort associated with, 106
    duration of anesthesia with, 104, 105*f*, 106
    endodontic uses of, 109-110
    failure of, 105
    heart rate increase after, 106-107
    injection site locations, 106
    lidocaine levels in plasma after, 107
    medical considerations for, 107
    onset of anesthesia, 106
Local anesthesia/anesthetics *(Continued)*
    perforator breakage with, 105-106
    postoperative discomfort from, 107
    repeating of, 106
    Stabident system for, 104, 105*f*
    success with, 105
    X-Tip delivery system for, 104-106, 105*f*, 110
  latex allergies, 93-94
  lidocaine
    inferior alveolar nerve block use of, 96, 98-99
    infiltrations of, 99
    meperidine added to, 100
  long-acting, 98, 120-121, 408
  mandibular, 101, 101*f*-102*f*
  maxillary, 101-103, 102*f*
  mechanism of action, 91-92
  methemoglobinemia caused by, 93
  nerve blocks for
    anterior middle superior alveolar, 103-104, 104*f*
    incisive, 99
    inferior alveolar. *See* Inferior alveolar nerve block
    infraorbital, 103
    palatal–anterior superior alveolar, 103, 104*f*
    posterior superior alveolar, 103
    second division, 103
  pain origin determination using, 701*f*
  in patients with previous difficulty with anesthesia, 95
  peripheral nerve paresthesias caused by, 93
  periradicular surgery using, 405-408
  phentolamine mesylate for reversal of, 96
  in pregnancy, 74*b*, 94
  pulpal, 95, 95*f*
  pulpal reactions to, 579-581, 580*f*-581*f*
  reversal of action, 96
  ropivacaine for, 98
  routes of administration, 95
  "rule of 25" for, 93
  systemic diseases or conditions that affect, 94-95
  systemic effects of, 93
  topical anesthetics with, 96
  types of, 92-93, 92*t*
  vasoconstrictors
    contraindications for, 94
    description of, 72-73
    drug interactions with, 94*t*
    pulpal blood flow affected by, 579-581, 580*f*
    pulpal health affected by, 579-581
    sulfites included in, 94
  Vazirani-Akinosi technique for, 99
Localized hemostasis, in periradicular surgery, 412-414, 437
Locus ceruleus, 686-687
Long-acting local anesthetics, 98, 120-121, 408
Low-molecular-weight heparin, 397
Lubricants, 262
Ludwig's angina, 716
Luting cements, 827-828

Luxation injuries
apical neurovascular supply damage caused by, 779-781, 779*f*-782*f*
biologic consequences of, 777-784
definition of, 777
description of, 759
external inflammatory resorption secondary to, 662, 664-665
incidence of, 777
intrusive, 777
lateral, 777
osseous replacement for, 778, 778*f*-779*f*
pulpal canal obliteration caused by, 779, 779*f*
pulpal necrosis caused by, 779
pulpal space infection caused by, 779-781, 780*f*
radiograph of, 760*f*
radiographs of, 762, 763*f*
subluxation, 777
treatment of, 777
types of, 777
LY293558, 554
Lymphatic vessel endothelium receptor-1, 558, 559*f*
Lymphocytes, 539, 540*f*, 645
Lymphomas, 5-6

M

Macrophages
antigen-presenting, 538-539
in apical periodontitis, 639, 643, 645
description of, 538, 538*f*
foreign antigens phagocytosis by, 634
micrograph of, 538*f*
in wound healing, 392
Magnetic resonance imaging, 24, 48
Magnetic resonance neurography, 48
Magnification, periapical healing affected by, 497
Major histocompatibility antigen, 538, 539*f*
Malignancy
jaw metastases of, 75, 75*f*
treatment planning affected by, 75-76, 75*f*
Malingering, 696-697
Mandibular anterior teeth
anesthesia for, 112
canines. *See* Mandibular canines
incisors. *See* Mandibular incisors
root canal configurations in, 139*t*
Mandibular artery, 747
Mandibular body, 712
Mandibular buccal vestibule, 712, 712*f*-714*f*
Mandibular canines
access cavity preparation in, 185, 190*f*
apical canal configurations for, 185.*e*1*t*
calcification age for, 188*f*
computed tomography of, 189*f*
eruption time for, 188*f*
morphology of, 135*t*
root canal configurations in, 139*t*, 185, 188*f*-190*f*
Mandibular first molars
access cavity preparation for, 193-199, 197*f*
accessory canals in, 133, 133*f*
accessory foramina in, 195
anesthesia of, 99*f*
apical canal configuration for, 195.*e*1*t*
Mandibular first molars *(Continued)*
calcification age for, 195*f*
caries in, *e*2*f*-*e*3*f*
computed tomography of, 196*f*
deep caries in, *e*8*f*
distal root of, 195.*e*2*t*
eruption time for, 195*f*
external cervical resorption in, 672*f*-673*f*
furcation canal in, 133*f*
lateral canal in, 287*f*
mesial root of, 605*f*, 664*f*, 195.*e*1*t*
morphology of, 135*t*
pulp chamber floor in, 136*f*
radix entomolaris of, 198-199, 198*f*
root canal configurations in, 139*t*, 196*f*-198*f*, 204*f*
Mandibular first premolars
access cavity preparation of, 155*f*, 185-192, 191*f*
apical canal configurations for, 190.*e*1*t*
calcification age for, 191*f*
computed tomography of, 191*f*
eruption time for, 191*f*
extra root of, 133-136, 136*f*
medial mesial canal in, 195
morphology of, 135*t*
root canal configurations in, 139*t*, 185-199, 191*f*-192*f*
Mandibular implants, neuritic complications of, 694
Mandibular incisors
central
access cavity preparation for, 183-185, 188*f*
apical canal configurations for, 185.*e*1*t*
calcification age for, 185*f*
computed tomography of, 186*f*
eruption time for, 185*f*
morphology of, 135*t*
root canal configurations in, 139*t*, 183-185, 188*f*
lateral
access cavity preparation for, 183-185, 188*f*
apical canal configurations for, 185.*e*1*t*
calcification age for, 185*f*
computed tomography of, 187*f*
eruption time for, 185*f*
morphology of, 135*t*
root canal configurations in, 139*t*, 183-185, 188*f*
morphology of, 135*t*
periradicular surgery on, 400
root canal configurations in, 139*t*
Mandibular molars
direct pulp capping of, *e*6*f*
first. *See* Mandibular first molars
furcation openings in, 133
infections of, 11
instrument separation in, 726
mesial roots of, 285, 287*f*, 812*f*
root canal in
calcification of, 163*f*
configurations of, 139*t*
C-shaped, 201, 204*f*
root canal treatment of, 210*f*
second. *See* Mandibular second molars
third. *See* Mandibular third molars
Mandibular posterior teeth
anesthesia for, 111, 113
with asymptomatic irreversible pulpitis, 113
molars. *See* Mandibular molars
premolars. *See* Mandibular first premolars; Mandibular second premolars
with symptomatic irreversible pulpitis, 113
Mandibular premolars
crown of, 154*f*
first. *See* Mandibular first premolars
root canal configuration in, 141*f*
root canal morphology in, 141*f*
second. *See* Mandibular second premolars
Mandibular second molars
access cavity preparation for, 199, 200*f*
apical canal configuration of, 199.*e*1*t*
apical resorption of, 663*f*
in Asians, 137, 140*f*
buccal space swelling associated with, 9-10, 9*f*
computed tomography of, 200*f*
deep caries in, *e*13*f*
eruption time for, 199*f*
external cervical resorption in, 672*f*-673*f*
foramina in, 204*f*
morphology of, 135*t*
in Native Americans, 137, 140*f*
pulp chamber of, 199
pulp floors in, 205
root canal configurations in
C-shaped, 201, 204*f*
description of, 139*t*, 199, 200*f*-201*f*, 204*f*
Mandibular second premolars
access cavity preparation for, 192-193, 194*f*
apical canal configurations for, 193.*e*1*t*
computed tomography of, 193*f*
morphology of, 135*t*
root canal configurations in, 139*t*, 192-193, 192*f*-194*f*
Mandibular teeth, 135*t*. *See also specific teeth*
Mandibular third molars
access cavity preparation for, 199, 201*f*-203*f*
apical canal configuration for, 199.*e*2*t*
buccal space swelling associated with, 9-10, 9*f*
calcification age for, 201*f*
computed tomography of, 202*f*
eruption time for, 201*f*
root canal configuration in
C-shaped, 199, 203*f*
description of, 199, 201*f*-203*f*
Mannitol and lidocaine, in inferior alveolar nerve block, 99
Manually activated irrigation, 263
Marginal periodontitis, 631
Martensite, 219-221, 222*f*
Masserann technique, for separated instrument removal, 367, 726-727
Mast cells, 539, 638
Masticatory muscles, 690
Matrix metalloproteinases, 578, 828
Maxillary anesthesia, 101-103, 102*f*
Maxillary anterior teeth
anesthesia for, 112
apical foramen in, 142-143
canines. *See* Maxillary canines
incisors. *See* Maxillary incisors
labiolingual diameter of, 142-143
root apex in, 142-143

Maxillary arch, 180*f*
Maxillary canines
  access cavity preparation for, 169, 173*f*
  apical canal configurations for, 169.*e*1*t*
  calcification age for, 171*f*
  computed tomography of, 172*f*
  eruption time for, 171*f*
  morphology of, 134*t*
  root canal configurations in, 139*t*, 169, 171*f*
Maxillary first molars
  access cavity preparation for, 171*f*-172*f*, 175-177, 179*f*
  apical canal configuration for, 177.*e*1*t*-177.*e*3*t*
  buccal roots of, 179*f*
  calcification age for, 177*f*
  computed tomography of, 178*f*
  distobuccal root of, 177.*e*2*t*
  eruption time for, 177*f*
  external inflammatory resorption of, 663*f*
  mesiobuccal root of, 178*f*, 415*f*
  mesiopalatal root of, 177, 177.*e*3*t*
  morphology of, 134*t*
  palatal root of, 180*f*, 177.*e*1*t*
  periradicular disease of, 390*f*
  preemptive nonsteroidal anti-inflammatory drugs, 113
  pulp chamber of, 175
  root canal configurations for, 175-177, 177*f*-180*f*
Maxillary first premolars
  access cavity preparation for, 169-173, 175*f*
  apical canal configurations for, 171.*e*1*t*
  in Asians, 169
  bonded root-end filling in, 419*f*
  calcification age for, 173*f*
  computed tomography of, 174*f*
  eruption time for, 173*f*
  morphology of, 134*t*
  pulp chamber of, 169-171
  root canal configurations of, 169-171, 173*f*, 175*f*
Maxillary incisors
  central
    access cavity preparation for, 165, 167*f*-168*f*, 175*f*
    apical canal configurations in, 165.*e*1*t*
    calcification age for, 167*f*
    computed tomography of, 168*f*
    eruption time for, 167*f*
    lingual shoulder of, 165
    morphology of, 134*t*
    periradicular disease associated with, 9
    root canal configurations in, 139*t*, 165, 169*f*
    root fracture in, 46*f*
    trauma to, 51, 52*f*
  lateral
    access cavity preparation, 165-166, 170*f*
    accessory foramina in, 171*f*
    anomalies of, 166, 171*f*
    apical canal configurations for, 166.*e*1*t*
    calcification age for, 169*f*
    computed tomography of, 170*f*
    dens invaginatus of, 166
    eruption time for, 169*f*
    internal root resorption of, 676*f*
    morphology of, 134*t*
Maxillary incisors *(Continued)*
    palatal radicular groove in, 166, 171*f*
    periradicular disease of, 389*f*
    root canal configurations in, 139*t*, 165-166, 169*f*, 171*f*
  morphology of, 134*t*
  root canal configurations in, 139*t*
Maxillary molars
  anesthesia of, 102*f*
  first. *See* Maxillary first molars
  infections of, 11
  palatal roots of, periradicular surgery considerations for, 399
  root canal configurations in, 139*t*, 248
  second. *See* Maxillary second molars
  third. *See* Maxillary third molar
Maxillary posterior teeth
  anesthesia for, 112
  molars. *See* Maxillary molars
  premolars. *See* Maxillary first premolars; Maxillary second premolars
Maxillary premolars. *See* Maxillary first premolars; Maxillary second premolars
Maxillary second molars
  access cavity preparation for, 177-183, 182*f*-183*f*
  apical canal configurations for, 182.*e*1*t*
  calcification age for, 180*f*
  computed tomography of, 181*f*
  distobuccal root of, 182.*e*1*t*
  eruption time for, 180*f*
  heavily restored, access cavity preparation in, 161*f*
  mesiobuccal root of, 177-182, 606*f*, 182.*e*1*t*
  morphology of, 134*t*
  orifices in, 182*f*
  palatal root of, 183*f*, 606*f*, 182.*e*2*t*
  pulp chamber of, 182-183
  root canal configurations in, 139*t*, 177-183, 180*f*-183*f*, 182.*e*1*t*-182.*e*2*t*
Maxillary second premolars
  access cavity preparation for, 173-175, 175*f*-176*f*, 179*f*
  apical canal configurations for, 173.*e*1*t*
  computed tomography of, 176*f*
  morphology of, 134*t*
  root canal configurations in, 139*t*, 173-175, 176*f*-177*f*
Maxillary sinus
  aspergillosis of, 741*f*
  cone-beam computed tomography identification of, 399
  perforation of, 745
  periradicular surgery concerns regarding, 399, 437
Maxillary sinusitis, 5-6
Maxillary teeth. *See also specific teeth*
  morphology of, 134*t*
  root canal configurations in, 139*t*
Maxillary third molar
  access cavity preparation for, 183, 183*f*, 185*f*
  apical canal configurations for, 183.*e*1*t*
  calcification age for, 183*f*
  eruption time for, 183*f*
  root canal configurations in, 183, 183*f*-185*f*
McSpadden Compactor, 313
Mechanical allodynia, 551
Medical conditions
  dental care modification for, 5, 5*b*
  oral manifestations of, 5-6
Medical consultation letter, 71, 72*f*
Medical history, 5-6
  blood pressure measurement, 5
  form for, 4*f*, 5
  medical conditions, 5, 5*b*
Medicaments, 499, 741, 766. *See also* Intracanal medicaments
Medicated sealers, 295-296, 297*f*
Medication-related osteonecrosis of the jaw, 76-77, 76*b*, 394
Medullary dorsal horn, 553-554
Melanocortin-1 receptor gene, 87
Memory cells, 634
Memory T cells, 538
Menstrual cycle, 94-95
Mental foramen
  anatomy of, 747-748
  periradicular surgery localization of, 398, 398*f*
Mental space, 712, 712*f*-714*f*
Meperidine, 100
Mepivacaine (3%)
  for inferior alveolar nerve block, 97
  for mandibular posterior teeth anesthesia, 111
Mesenchymal stem cells
  description of, 448-449, 450*f*
  gingival-derived, 449
  markers of, 450-451, 451*f*
  multipotent, 630-631
  odontoblast differentiation from, 451-452, 454, 454*t*
  in perivascular tissue, 392
  in root canal space, 450-451, 452*f*
  undifferentiated, 562
Mesiobuccal angle, 175
Mesiopalatal canal orifice, 175
Messing gun-type syringe, 407*f*
Metal carrier, 352-353, 352*f*-353*f*
Metal restorations, 149
Metalloceramic crown, access cavity preparation through, 159, 162, 162*f*
MetaSEAL, 293-294
Methacrylate resin sealers
  characteristics of, 292-294
  obturation use of, 292-294
Methemoglobinemia, 93
Methylchloroform, 347-348
Microbiota. *See also* Microorganisms
  definition of, 612
  diversity of, 612-614, 613*f*
  in endodontically treated teeth, 620-622, 621*f*-623*f*
  identification of, 610-612, 610*b*, 612*t*
  in oral cavity, 612-613
Microcomputed tomography, 37
Microcondensers, 406*f*
Microcracks, 810-812, 811*f*-812*f*
Microfilaments, 535
Micromirror, 406*f*
Micro-Openers, 146, 146*f*
Microorganisms. *See also* Bacteria
  anachoresis of, 601
  apical periodontitis caused by, 600
  communities of, 606-607

Microorganisms *(Continued)*
culture of, 610, 610*b*
identification of, 610-612, 610*b*, 612*t*
intraradicular, posttreatment disease caused by, 325-326
molecular methods for studying of, 611*b*, 612, 613*f*
pathogenicity of, 602-604
persistent periradicular disease caused by, 388-389
populations of, 606-607
posttreatment disease caused by, 388
root canal penetration of, during root canal treatment, 602
root canal system cleaning of, 209-210
spatial distribution of, 604-606
studies of, 611-612
virulence factors, 602-604
MicroRNAs, 579
Microseal system, 681
Microsurgical scalpel, 405*f*
Microtubules, 535
Midface swelling, 715
Midroot perforations, 374-375, 379*f*-380*f*
Migraine headaches, 691-692
Miller, Willougby Dayton, 599-600
Mineral trioxide aggregate
apexification using, 462-464, 468, 771*f*
as apical barrier, 314, 314*f*
biocompatibility of, *e*2
characteristics of, 421, *e*2
color of, 766
components of, *e*3
description of, *e*1, *e*6
disadvantages of, 376-377
hard-tissue formation caused by, 563
history of, *e*2
inflammatory cascade, *e*2
MTA-Angelus, *e*3
pulp capping using, 483, 587-588, 766, *e*7, *e*10-*e*16
direct, 587-588, 766, *e*7, *e*8*f*, *e*13*f*
one-step, *e*10-*e*11
two-visit, *e*10-*e*16, *e*26*f*
pulpal repair promotion by, *e*4
pulpotomy with, *e*7, *e*14*f*, *e*16*f*
reparative bridge formation using, *e*4*f*
root perforations repaired using, 26-27, 147, 376-378, 377*f*, 681
as root-end filling material, 421-422
sealers using, 294
for sealing root perforations, 26-27, 147
signaling molecules, *e*2
vital pulp therapy using
biocompatibility of, *e*2
characteristics of, *e*2
components of, *e*3
contralateral tooth development, *e*10
description of, *e*1, *e*6
direct pulp capping with, *e*7, *e*8*f*, *e*13*f*
history of, *e*2
inflammatory cascade, *e*2
MTA-Angelus, *e*3
pulpotomy with, *e*7, *e*14*f*, *e*16*f*
radiographs of, *e*27*f*
signaling molecules, *e*2
Minocycline, 788*f*
Mobility
recording of, 15*b*
testing of, 15, 15*b*, 15*f*
Modified glass ionomer core, 826
Molar triangle, 175
Molars
mandibular. *See* Mandibular molars
maxillary. *See* Maxillary molars
Monoclonal antibodies, 536-537
Monsel's solution, 413
Morphogens, 453-455
Mounce extractor, 369, 369*f*
"Mouse hole" effect, 156, 157*f*
MRONJ. *See* Medication-related osteonecrosis of the jaw
Msx1, 453
Msx2, 453
MTA Fillapex, 295
MTA-Angelus, *e*3
MTwo, 231-232
Mucobuccal fold, 11, 13*f*
Mucoperiosteal flap, 675
Mucous cell metaplasia, 647-649
Mueller bur, 149, 149*f*
Multidetector computed tomography, 37
Multinucleated osteoclasts, 779-780, 780*f*
Multiple myeloma, 5-6
Multiple-visit treatment, 79-82
Multipotent mesenchymal stem cells, 630-631
Multipotent stem cells, 448-449
Musculoskeletal pain, 699
M-Wire, 222
Mylohyoid nerve block, 100, 100*f*
Myofascial pain, 690-691
Myofibroblast, 392
MZ100, 822

N

N-2, 295-296
N-acetyl-*p*-benzoquinone imine, 117-118
Nanoparticles, antimicrobial, 265
Nasal mucosal pain, 691
Nasolabial fold, 9, 9*f*
National Council for Radiation Protection, 35, 35*b*
Native Americans, mandibular second molar root configurations in, 137, 140*f*
Natural killer cells, 639
Nd:YAG laser, 588-589, 589*f*
gutta-percha removal using, 351
Negative apical pressure, 264, 264*f*
Negative rake angle, 216, 219*f*
Negative-pressure irrigation, 264, 264*f*
Neovascularization, 644-645
Nerve blocks
anterior middle superior alveolar, 103-104, 104*f*
incisive, 99
inferior alveolar. *See* Inferior alveolar nerve block
infraorbital, 103
mylohyoid, 100, 100*f*
second division, 103
Nerve fibers, 543*t*
in chronic apical periodontitis, 646*f*
intradental, plasticity of, 552-553
pulpal
description of, 545-546, 545*f*
neuropeptides in, 547
Nerve growth factor, 538, 552
Neural modulating agents, for dentin hypersensitivity, 579
Neuralgia, 692-693
Neuritis, 693-694, 694*f*
Neurogenic inflammation, 635, 636*f*
Neurokinin A, 547
Neuroma, 693
Neuropathic pain, 688*b*, 692-696
Neuropathy, 694-696
Neuropeptides
description of, 547, 635, 641
pulpal immune response modulated by, 577
Y, 542, 543*f*, 578-579
Neurovascular pain, 691-692
Neutrophil extracellular traps, 634
Neutrophils, polymorphonuclear, 392
Niche, 607
Nickel-titanium instruments
coronal preflaring using, 235, 237*f*
deformation of, 223*f*
design of, 228*f*
files, 240
fracture of, 227*f*, 235*b*, 724-725
Greater Taper hand files, 372-373
gutta-percha removal using, 349-350, 350*f*, 742
root canal curvatures maintained using, 737
rotary, 214-216, 218*f*-219*f*, 226, 233*f*, 266, 811*f*
separated, 727-728
success rates with, 497
Nickel-titanium spreaders, 812, 815*f*
Nitinol, 219-220
Nitrous oxide, for conscious sedation, 113
Nitrous oxide–oxygen, in pregnancy, 74
NK1 antagonists, 554
NMDA receptor, 554
Nociceptive pain, 688*b*
Nociceptors
inflammatory mediators on, 550*t*
sensitization of, 550
transmission of information, 546*f*
Nociceptors, pulpal, 689
Nogenol, 292
Noncontinuous anesthesia, 96
Non-narcotic analgesics
acetaminophen, 117
alternatives to, 117
contraindications for, 117
drug interactions with, 117, 117*t*
efficacy of, 116*t*
limitations of, 117
nonsteroidal anti-inflammatory drugs, 114-115
overview of, 114-117
types of, 115*t*
Nonodontogenic lesions, 83
Nonodontogenic toothache. *See also* Pain
cardiac pain as cause of, 697
case studies of, 702-703, 702*f*, 702*t*-703*t*
craniofacial structures that cause, 697-698
description of, 684
examination for, 700-703
frequency of, 698
indicators of, 703
intracranial structures as cause of, 697
musculoskeletal pain as cause of, 690-692

Nonodontogenic toothache *(Continued)*
myofascial pain as cause of, 690-691
neuralgia as cause of, 692-693
neuritis as cause of, 693-694, 694*f*
neuroanatomy of, 684-687
A-beta fibers, 685
A-delta fibers, 685-686
autonomic nervous system, 687
C fibers, 686-687
central nervous system, 686-687
neural structures, 685-687
peripheral nervous system, 685-686
primary afferent neurons, 685-686
second-order neurons, 687
neuroma as cause of, 693
neuropathic pain as cause of, 692-696
neuropathy as cause of, 694-696
neurovascular pain as cause of, 691-692
patient examination for, 700-703
red flags for, 703
salivary gland pain as cause of, 691
sinus pain as cause of, 691
somatic pain as cause of, 690-692
sources of, 690-700
Nonspecific innate immune response, 634
Nonsteroidal anti-inflammatory drugs
acetaminophen and, 121
COX-inhibiting nitric oxide donators and, 122
cyclooxygenase inhibition by, 395
description of, 114-115
drug interactions with, 117, 117*t*
opioid analgesics and, 121
periradicular surgery use of, 402-404, 436
postoperative pain managed with, 436
preemptive uses of, 113, 120
pretreatment uses of, 120
swelling reduction using, 719
wound healing affected by, 395
Nonsurgical perforations, 737-739
Nonsurgical retreatment
access cavity preparation for, 335-336, 335*f*, 337*f*-338*f*
apical area access, 346-361, 347*f*-355*f*, 357*f*-361*f*
apical periodontitis managed with, 389
apical surgery versus, 334
armamentarium for, 325*f*
considerations for, 334
coronal access cavity preparation for, 335-336, 335*f*, 337*f*-338*f*
finishing of, 373-374, 375*f*
gutta-percha removal, 347-351, 347*f*-351*f*, 355
healing rates of, 334, 380
heat generation during, 370-371
irrigant effects on, 498-499
metal carrier removal, 352-353, 352*f*-353*f*
outcome measures for, 477-479
outcome of, 515-517, 518*f*, 521*f*
overview of, 324
paste, 356-358, 357*f*
periapical healing after, 517, 518*f*, 521*f*
persistent periradicular pathosis after, 404*f*
plastic carrier removal, 353-354, 354*f*
post removal in, 337-346, 338*f*-345*f*
posttreatment disease. *See* Posttreatment disease
prognosis of, 380-381, 380*f*-382*f*
Nonsurgical retreatment *(Continued)*
root canal blockage, 373, 374*f*
root canal impediments, 371-373
root perforation repair, 374-378, 375*f*-379*f*
root-filling material removal, 346-361, 347*f*-355*f*, 357*f*-361*f*
separated instrument removal, 361-370
causes of, 361-363
silver point removal, 358-361, 358*f*-363*f*
solid core obturators, 351-356, 352*f*-355*f*
surgical retreatment and, 333
treatment planning for, 331-334, 331*f*-335*f*
Nonsurgical root canal treatment
outcomes evaluation
periapical healing after, 495-509
quality of life, 515, 517*t*
tooth survival, 509-515, 514*f*, 516*f*
quality of life effects of, 515, 517*t*
Nonvital teeth
aesthetic changes in, 821
anterior, 830, 831*f*-832*f*
compositional changes in, 818-819
dentin structure in, 819
discoloration of, 821, 821*f*
fracture resistance of, 819-821
moisture content changes in, 818
posterior, 831*f*-832*f*
tooth stiffness of, 819-821
Novel oral anticoagulants, 397
Nucleus raphe magnus, 686-687

O

Obtura II, 681
Obtura III, 309-310, 309*f*
Obturation
adequate, 282*f*
apical barriers for, 314
apical portion of fill, 290
bacteria during, 620
continuous wave compaction technique of, 305-308, 307*f*-308*f*
core materials used in, 296-301, 812
Active GP, 299
custom cones as, 300-301, 301*f*-302*f*
extrusion of, beyond radicular foramen, 742-745, 743*f*
gutta-percha. *See* Obturation, gutta-percha for
ideal, 289-290, 297*b*
in large root canal systems, 301, 302*f*
Resilon, 299-300, 300*f*, 351, 351*f*
silver cones, 296-297, 298*f*
coronal microleakage after, 315-316
coronal orifice seal after, 315-316
GuttaCore for, 355
gutta-percha for
carrier-based, 311-313
description of, 284, 297-299, 298*f*-299*f*, 304, 681
illustration of, 315*f*
lateral compaction of, 304
overextension of, 742
removal of, 742, 745*f*
solvent techniques, 314
thermoplastic injection techniques for, 309-311, 309*f*-310*f*
vertical compaction of, 312
warm vertical compaction of, 304, 305*f*-306*f*
Obturation *(Continued)*
historical perspectives on, 283-284
immediate, 314-315, 315*f*
inadequate, 282*f*
incomplete, failures caused by, 280
internal root resorption treated with, 681
in large root canal systems, 301, 302*f*
lateral compaction method of, 302-304, 302*f*-303*f*
length of, 285-287, 285*f*-286*f*
paraformaldehyde-containing materials for, 289, 290*f*
pastes for, 314, 356-358, 357*f*
preparation for, 287-289, 288*f*-290*f*
in pulpal necrosis, 284-285
radiographic assessment of, 284
resin-bonded, 351
after root canal cleaning and shaping, 285
sealers used in
biocompatibility of, 290-291
calcium hydroxide, 292, 314
calcium silicate, 294-295, 295*t*-296*t*
extrusion of, 291, 291*f*
glass ionomer, 292, 292*f*-293*f*
ideal properties of, 290*b*
lentulo spiral for placement of, 296, 297*f*
medicated, 295-296, 297*f*
noneugenol, 292
paraformaldehyde-containing, 295-296
periradicular extrusion of, 291, 291*f*
placement of, 296, 297*f*
radiopacity of, 290
resin, 292-294. *See also* Resin sealers
silicone, 294, 294*f*
tricalcium silicate, 294, 295*t*-296*t*
zinc oxide–eugenol, 291-292, 292*t*
smear layer removal before, 288, 289*f*, 311
solid core obturators, 351-356, 352*f*-355*f*
solvent techniques for, 314
thermomechanical compaction technique for, 313
thermoplastic injection techniques for, 309-311, 309*f*-310*f*
timing of, 284-285
in vital pulp tissue, 284
warm lateral compaction technique of, 308-309, 308*f*
warm vertical compaction method of, 304-305, 305*f*-307*f*
Occlusal caries, *e*8*f*
Occlusal cavities, 830-833
Occlusal forces, 829
Occlusal loading, tooth deformation during, 593
Occlusal teeth evaluation, in access cavity preparation, 145-146
Occlusal trauma, 328
Occlusal wear facets, 328
Occlusion-related pain, 706
Ochsenhein-Luebke flap, 410*f*, 424-425
Odontoblast(s)
alkaline phosphatase secretion by, 535
cell body of, 535
cementoblasts versus, 535
characteristics of, 535, *e*1
chemokine secretion by, 575
collagen synthesis by, 535
dentin sialoprotein expression by, 451
in dentinogenesis, 535

Odontoblast(s) *(Continued)*
description of, 451
destruction of, 562-563
differentiation of, 575-576
displacement of, 549, 549*f*
illustration of, 534*f*
inactive, 535
in inflammatory response to caries, 576, 577*f*
in internal root resorption, 675-676
mesenchymal stem cell differentiation into, 451-452, 454, 454*t*
morphologic characteristics of, 535
neuropeptides on, 535, 536*f*
nucleus of, 535
osteoblasts versus, 535
predentin-dentin matrix secreted by, *e*1
progenitors of, *e*1
in reparative dentin formation, *e*1
resting, 535
secretory function of, 537
structure of, 537
Toll-like receptor expression, 575
ultrastructural features of, 535, 536*f*
Odontoblast layer, of pulp, 532-534, 533*f*-534*f*, 577*f*
Odontoblast process, 535-537, 575-576, 590*f*
Odontoblast-like cells
growth factors in differentiation of, 454*t*
regenerative capacity of, 585-586
Odontoclasts, 643-644, 660-662, 661*f*
Odontogenic keratocysts, 647-649
Odontogenic pain
history-taking for, 698-700, 698*f*
pulp-dentin complex as source of, 687-689
Odontogenic toothache
description of, 687-689
sources of, 687-690
One-step pulp capping, *e*10-*e*11
Onlays, 822, 838*f*
Operating microscope, in separated instrument removal, 369, 369*f*
Operative explorer, 150*f*
Opioid analgesics
applications of, 117-118, 118*t*
nonsteroidal anti-inflammatory drugs and, 121
Opioid receptors, in pulp, 578-579
Opportunistic pathogens, 602
Optical coherence tomography, for enamel crack detection, 25
Oral Health Impact Profile, 479, 515, 517*t*
Oral Radiographic Differential Diagnosis, 75
Orofacial pain. *See* Pain
Orthodontic extrusion, 834
Orthodontics
pulpal blood flow affected by, 592
pulpal reactions to, 592
Orthognathic surgery, 592
Osseous defects, 802
Osseous replacement, for luxation injuries, 778, 778*f*-779*f*, 785
Osteoblasts
collagen secretion by, 393-394
in hard-tissue wound healing, 393-394
Osteoclasts
actin cytoskeleton of, 660
in asymptomatic apical periodontitis, 643-644
bone loss caused by, 633
Osteoclasts *(Continued)*
bone resorption caused by, 643, 644*f*
definition of, 660
in hard-tissue wound healing, 393
multinucleated, 779-780, 780*f*
ruffled border of, 660, 661*f*
Osteomyelitis, chronic focal sclerosing, 650
Osteonecrosis of the jaw
medication-related, 76-77, 76*b*
radiation therapy as cause of, 655
Osteopontin, 575-576
Osteoprotegerin, 662
Outcome measures
nonsurgical retreatment procedures, 477-479
nonsurgical root canal treatment procedures, 477-479
periapical surgery, 479-480, 481*f*-483*f*
for regenerative endodontic procedures, 467-468
surrogate, 475
types of, 475
vital pulp therapy procedures, 476-477, 476*t*
vitality responses as, 468
Outcomes evaluation
cone beam computed tomography for, 56-60
context for, 474-475
direct pulp capping, 483, 488*t*-490*t*
effectiveness of procedures determined using, 475
factors that affect, 475-476
full pulpotomy, 494*t*
indirect pulp capping, 480-482, 485*f*, 486*t*, 491*f*
nonsurgical retreatment, 515-517, 518*f*, 521*f*
nonsurgical root canal treatment
periapical healing after, 495-509
quality of life, 515, 517*t*
tooth survival after, 509-515, 514*f*, 516*f*
periodontal incisional wound healing, 522
pooling data, 475-476
prognostication value of, 476
pulpotomy, 483-484
purpose of, 475-476
surgical retreatment, 517-522
Overhangs, 240
Overlays, 822, 830*t*, 838*f*
Oxygen consumption, 539

P

Pain. *See also* Nonodontogenic toothache
aggravating factors of, 699
alleviating factors for, 699
analgesics for, 719
associated factors, 700
cardiac, 697
central sensitization in, 553-555, 688*b*, 695
chronic, 700-702
deep, 699
definition of, 688*b*
description of, 684
descriptors of, 699, 699*t*
detection of, 546-547
facial, 684
gender differences in, 94-95
heterotopic, 688*b*, 690, 700
history-taking, 698-700, 698*f*
induction of, 641
Pain *(Continued)*
intensity of, 699
local anesthetics used to localize, 701*f*
management of, 706-707
flexible analgesic strategy for, 121-122, 121*f*
long-acting local anesthetics for, 98, 120-121
nonsteroidal anti-inflammatory drugs for, 436
after periradicular surgery, 437
pretreatment for, 120
strategies for, 120-122
muscular, 700
musculoskeletal, 690-692, 699
myofascial, 690-691
nasal mucosal, 691
neuroanatomy of, 684-687
A-beta fibers, 685
A-delta fibers, 685-686
autonomic nervous system, 687
C fibers, 686-687
central nervous system, 686-687
neural structures, 685-687
peripheral nervous system, 685-686
primary afferent neurons, 685-686
second-order neurons, 687
neuropathic, 44, 688*b*, 692-696
neurophysiology of, 687
neurovascular, 691-692
nociceptive, 688*b*
occlusion-related, 706
onset of, 699
origins of, determination of, 700, 701*f*
patient examination for, 700-703
peripheral sensitization in, 687, 688*b*, 695
periradicular, 690
after periradicular surgery, 437
persistent dentoalveolar pain disorder, 695-696, 695*f*, 696*b*
progression of, 699
psychogenic, 697
pulpal, 689
pulpitis-related, 551-552
quality of, 699, 699*t*
reduction of, in irreversible pulpitis, 112-113
referred, 30, 684, 688*f*, 699
salivary gland, 691
sinus, 691
somatic, 690-692
subjective nature of, 542
superficial, 699
temporal aspects of, 699
types of, 687, 688*b*
voltage-gated sodium channels and, 92*t*
Pain history, 698-699, 698*f*
Pain modulation, 555*f*
Pain perception
detection and, 546-547
mechanism of, 546
medullary dorsal horn in, 553-554
processing step of, 553-555
schematic diagram of, 555*f*
thalamus to cortex pathway, 555-556
Painless pulpitis, 578
Palatal–anterior superior alveolar nerve block, 103, 104*f*

Palate
anterior, swelling of, 11, 12*f*
posterior, swelling of, 13*f*
Palpation
of cervical lymph nodes, 9
in intraoral examination, 13
of submandibular lymph nodes, 9
Parachloroaniline, 255
Parafunctional habits, 592-593, 829
Parallax radiographs, of internal root resorption, 677
Paranasal sinus aspergillosis, 741
Parapharyngeal space, 712*f*-714*f*, 715
Paresthesia
endodontic-related, 740-741
peripheral nerve, 93
after periradicular surgery, 437
PARRs. *See* Pathogen-associated recognition receptors
Partial pulpotomy
calcium hydroxide, 766*f*
crown fractures treated with, 767, 768*f*-769*f*
Cvek, 767, 768*f*
definition of, *e2*
direct pulp capping versus, *e2*
follow-up after, 767, 769*f*
outcomes evaluation of, 483-484, 491*f*, 492*t*
pulp capping versus, 766-767
root development after, 769*f*
technique for, 767, 768*f*
traumatic pulp exposures treated with, 767, 768*f*-769*f*
Partial restorations, 844
Passive ultrasonic irrigation, 263-264
Paste
nonsurgical retreatment using, 356-358, 357*f*
overextended, 357*f*
Patency file, 236-237
Pathogen-associated recognition receptors, 575, 632-633
Patient
evaluation of, 71
registration of, 2
Pattern-recognition receptors, 575, 632-633
Peeso drills, 225, 341-343
Penicillin V, 787
Percussion testing, 14*f*, 15, 19
Perforations
cone-beam computed tomography of, 50-51
definition of, 50-51
furcal, 378*f*
maxillary sinus, 745
nonsurgical, 737-739
post, 375*f*-376*f*
during post removal, 346
root canal system cleaning and shaping as cause of, 268, 268*f*
Perforin, 634-635
Periapical abscess, 642*f*
Periapical cysts
incidence of, 326
pocket, 326
true, 326
types of, 326
Periapical diseases
classification of, 27-30, 637
inflammatory, 635
after root canal treatment, 477-478, 478*f*
Periapical granuloma, 642-646
actinomyces in, 483*f*
Periapical healing
after apical surgery, 482*t*, 519*t*-520*t*, 521*f*
description of, 479, 482*t*, 484*f*
factors that affect, 495-509
acute exacerbation during treatment, 505
apical preparation, 497-498
bacteria presence, 499-504, 500*t*-503*t*
illumination, 497
irrigants, 498-499
isolation, 497
magnification, 497
mechanical preparation, 497-498
medicaments, 499
number of treatment visits, 505
patient-related, 495-497
root canal bacterial culture, 499, 500*t*-503*t*
root filling, 504-505
summary of, 506-509, 506*f*-509*f*
treatment-related, 497-505
after nonsurgical retreatment, 517, 518*f*, 521*f*
restorations effect on, 515
Periapical health
after periapical surgery, 517-522, 521*f*
after root-end filling, 517-522, 521*f*
Periapical index, 479
Periapical infection
description of, 632
foreign bodies as cause of, 632
with sinus tract, 13
Periapical inflammation, 632
Periapical lesions
nonendodontic, 650
periapical healing affected by, 496-497
therapy-resistant, 481*f*
three-dimensional radiographic appearance of, 44
Periapical osteolytic lesions, 649
Periapical radiolucencies, 282-283
Periapical status, 479, 480*t*
Periapical surgery
anesthesia for, 114
healing after, 479, 482*t*, 484*f*
outcome measures for, 479-480, 481*f*-483*f*
periapical health after, 517-522, 521*f*
quality of life effects of, 522
success criteria for, 482*t*, 484*f*-485*f*
tooth survival after, 522
Periapical wound healing, 653, 653*t*
Peri-implantitis, 332*f*
Periodontal crown lengthening surgery, 834
Periodontal disease
deep pockets associated with, 802
illustration of, 602*f*
pulp vitality in, 82-83
pulpal necrosis caused by, 601
systemic health conditions caused by, 58-60
terminal, 333
Periodontal evaluation, 829
Periodontal examination, 15
Periodontal incisional wound healing, 522
Periodontal ligament
cells of
apoptosis of, 388
description of, 630-631
collagen fibers of, 630-631
contusion injuries to, 665
definition of, 630-631
extracellular matrix of, 630-631
inflammation of, 14-15
mechanoreceptors of, 690
stem cells of, 449-450, 450*f*
substance P in, 577
Periodontal pocket depth, 15
Periodontal probing, 15
Periodontal scaling, 592
Periodontitis
advanced, with periradicular inflammation, 426-428
apical. *See* Apical periodontitis
definition of, 29
marginal, 631
periradicular, 773*f*
pulpal inflammation caused by, 591-592
Periodontium, intraligamentary anesthesia injection effects on, 108
Periorbital space, 712*f*-714*f*, 715
Periosteal derived stem cells, 449, 450*f*
Peripheral inflammatory root resorption, 667
Peripheral nerve paresthesias, 93
Peripheral nervous system, 685-686
Peripheral neuropathy, 696
Peripheral sensitization, 550, 550*b*, 550*t*, 687, 688*b*, 695
Periradicular abscess, 9*f*
Periradicular cholesterol cysts, 388-389
Periradicular disease
of maxillary first molar, 390*f*
of maxillary lateral incisor, 389*f*
Periradicular inflammation
advanced periodontitis with, 426-428
description of, 603-604
Periradicular lesions
implant success affected by, 594
radiograph of, 738*f*-740*f*, 744*f*
Periradicular pain, 690
Periradicular pathosis
etiology of, 281, 388, 414
after nonsurgical retreatment, 404*f*
Periradicular periodontitis, 773*f*
Periradicular surgery
access for
flap designs, 409, 409*f*-410*f*
full-thickness flap, 410*f*
hard-tissue, 411
horizontal incision for, 409, 409*f*
intrasulcular incisions, 409
principles used in, 408
soft tissue, 408
submarginal flap, 410*f*
tissue reflection, 409-411, 410*f*
tissue retraction, 411
triangular flaps, 409*f*
vertical incision for, 408-409, 408*f*-410*f*
anatomic considerations for, 397-400, 398*f*-400*f*
anterior maxilla and mandible, 399-400, 400*f*
mandibular canal, 398

Periradicular surgery *(Continued)*
mental foramen, 398, 398*f*
posterior mandible, 397-398, 398*f*
posterior maxilla, 399, 399*f*
in anterior maxilla and mandible, 399-400, 400*f*
antibiotic prophylaxis before, 396, 402-404
in anticoagulation, 396-397
apical periodontitis treated with, 389
aspirin discontinuation before, 397
bacteremia associated with, 396
in bacterial endocarditis patients, 396
biopsy in, 411-412
bleeding after, 437
bleeding concerns during, 397
bone in
healing of, 393-394
heating effects on, 411
removal of, 411
cautery in, 414
chlorhexidine use before, 402-404
clinical decision making for, 389-390
complications of, 437, 437*f*
cone-beam computed tomography in
computed tomography versus, 400-402
description of, 400-402
mandibular canal identification using, 398
maxillary sinus identification using, 399
mechanism of action, 400-401
scan times with, 401-402
conscious sedation for, 402-404
curettage in, 411-412
electrosurgery in, 414
flap designs used in, 409, 409*f*-410*f*
goals of, 408, 414
guided tissue regeneration in, 426-435, 427*f*-433*f*
hard tissue in
access considerations, 411
healing of, 393-394
removal of, 411
hemostasis during, 412-414, 437
incisions used in
horizontal, 409, 409*f*
intrasulcular, 409, 409*f*
submarginal, 410*f*
vertical-releasing, 408-409, 408*f*-409*f*
indications for, 388-390, 391*f*
informed consent for, 402
instruments used in, 405, 405*f*-407*f*
intentional replantation, 436, 436*f*
local anesthesia for, 405-408
localized hemostasis during, 412-414, 437
in mandible
anterior, 399-400, 400*f*
posterior, 397-398, 398*f*
on mandibular incisors, 400
in maxilla
anterior, 399-400, 400*f*
posterior, 399, 399*f*
maxillary sinus in
cone-beam computed tomography identification of, 399
exposure of, 437
in medically complex patients, 395-397
mental foramen identification, 398, 398*f*
nonsteroidal anti-inflammatory drugs and, 402-404, 436

Periradicular surgery *(Continued)*
objectives of, 414
operatory setup for, 405
pain after, 437
palatal roots of maxillary molars, 399
paresthesia after, 437
patient preparation for, 402-404
persistent periradicular disease treated with, 388-389
in posterior mandible, 397-398, 398*f*
in posterior maxilla, 399, 399*f*
postoperative care and instructions, 426*f*, 436-437
premedication before, 402-404
in prosthetic joint patients, 396
rationale for, 389
regeneration as goal of, 390-391
ridge preservation in, 435
root end
anatomy of, 414
angle of resection, 415, 415*f*
burs for, 416
cavity preparation. *See* Periradicular surgery, root-end cavity preparation
conditioning of, 416
filling materials for. *See* Periradicular surgery, root-end filling materials
goals for, 416
management of, 414-416
perpendicular resection of, 415*f*
resection of, 414-415, 414*f*-415*f*
surface preparation of, 416
surface topography of, 416
root-end cavity preparation, 417-419
errors in, 417*f*
illustration of, 417*f*
root fracture concerns, 417-418, 417*f*
ultrasonic, 417-418, 417*f*
root-end filling materials
bioceramics, 422
bonded, 418-419, 419*f*
composite resins, 420-421
Diaket, 420
Dyract, 421
Geristore, 421
glass ionomer cements, 420
ideal characteristics of, 419
mineral trioxide aggregate, 421-422
overview of, 422
resin-ionomer hybrids, 420-421
zinc oxide–eugenol cement, 419-420
success rates for, 390
surgical site closure
flaps for, 424-425
sutures for, 425-426
technique for, 422-426, 426*f*
sutures for, 425-426, 436
tissue in
reflection of, 409-411, 410*f*
retraction of, 411
transient bacteremia associated with, 396
vertical-releasing incisions for, 408-409, 408*f*-410*f*
wound healing
angiogenesis in, 392-393
biologic principles of, 390-391
bisphosphonates effect on, 394-395
cementoblasts in, 394

Periradicular surgery *(Continued)*
cementogenesis in, 394
clot formation in, 392
collagen in, 392-393
cyclooxygenase-2 inhibitors effect on, 395
endothelial cells in, 392-393
epithelium in, 393
factors that affect, 390
fibroblasts in, 392
glucocorticoids effect on, 395
granulation tissue formation in, 392
hard-tissue, 393-394
hormones involved in, 394
inflammatory phase of, 391-392
macrophages in, 392
maturation phase of, 393
nonsteroidal anti-inflammatory drugs effect on, 395
osteoblasts in, 393-394
osteogenesis in, 393-394
polymorphonuclear neutrophils in, 392
proliferative phase of, 392-393
soft-tissue, 391-393
systemic disease effects on, 389
systemic medications that affect, 394-395
Periradicular tissue
anatomy of, 630
injury response of, 631
sodium hypochlorite effects on, 254*f*
Peritubular dentin, 585, 585*f*
Permanent restoration
traumatic dental injury and, 789
vital pulp therapy and, *e*10-*e*11
Persistent dentoalveolar pain disorder, 695-696, 695*f*, 696*b*
Persistent idiopathic facial pain, 44
Persistent infection, 612
Phagocytes, 639, 647*f*
Phagosomes, 639
Phantom tooth pain, 553, 690, 694-695
Phenol, 261
Phenolic preparations, 261
Phentolamine mesylate, 96
Phospholipase A2, 640
Phosphoryn, 535
Photoacoustic streaming, of irrigants, 265
Photoactivation disinfection, 265
Photostimulable phosphor plates, 33-35
Picture archiving and communication systems, 33
Pink spot, 784, 784*f*
Pit and fissure sealants, 588
Pits and fissures, 588
Plastic carriers, 353-354, 354*f*
Platelet aggregation, 551
Platelet endothelial cell adhesion molecule, 638
Platelet-derived growth factor, 653*t*
Platelet-rich plasma, 455
Platelets, 639
Plexus of Raschkow, 544-545, 544*f*
Pluripotent stem cells, 448-449
PMNs. *See* Polymorphonuclear neutrophils
Pocket cysts, 326, 646-649, 648*f*
Pockets
probes for probing of, 802-803, 806*f*
in vertical root fractures, 802-803, 805*f*-806*f*
Polocaine, 97
Polycarboxylate cements, 827

Polyglactic acid membrane, 435*t*
Polyglycolic acid, 455
Polylactic acid membrane, 435*t*
Polylactic-coglycolic acid, 455
Poly-L-lactic acid, 455
Polymodal receptors, 548-549
Polymorphonuclear leukocytes
  in apical periodontitis, 638-639
  description of, 118
Polymorphonuclear neutrophils
  description of, 634
  in wound healing, 392
Polytetrafluoroethylene membrane, 435*t*
Pooling data, 475-476
Porcelain fused to metal restorations, 336
Positive rake angle, 216, 219*f*
Post
  active, 337, 338*f*
  broken, 339*f*
  cast, 337, 826-827, 833*t*
  cast gold, 844-846, 845*f*
  cementation of, 337-339
  ceramic, 341-343
  characteristics of, 823
  clinical applications of, 337
  clinical procedures for, 838-844, 840*f*-843*f*
  custom cast, 337
  excessive flexing of, 823
  fiber, 825, 834
  fiber-reinforced composite, 341-343
  fractured, 340*f*
  materials used in fabricating, 337, 338*f*
  passive, 337, 338*f*
  perforation, 375*f*-376*f*
  placement of, 841
  prefabricated, 337, 824-825
  removal of
    complications of, 345-346, 346*f*
    exposure of post during, 339
    instruments used in, 339-341
    location considerations, 339
    in nonsurgical retreatment, 337-346, 338*f*-345*f*.
    perforation during, 346
    post separation during, 346
    retention reduction technique, 340
    techniques for, 339-343, 339*f*-345*f*.
    ultrasonic instrument for, 340, 340*f*
  resilience of, 823
  retention of, 824-825, 827
  rigid, 833-834
  shapes of, 337, 338*f*
  stainless steel, 338*f*
  in structurally compromised teeth, 833-834
  threaded, 825
  tooth survival affected by, 515
  tooth weakening caused by, 345-346
  tooth-colored, 341-343
  vented, 337, 338*f*
  zirconium
    composition of, 825
    removal of, 341-343
Post Puller, 341, 344*f*
Post space, 828-829
Posterior mandible, periradicular surgery in, 397-398, 398*f*
Posterior maxilla, periradicular surgery in, 399, 399*f*
Posterior superior alveolar nerve block, 103
Posterior teeth. *See also specific posterior teeth*
  nonvital, restorations for, 830-833, 831*f*-832*f*
  partial restorations in, 844
  structurally compromised, 834
Posttreatment disease
  apical periodontitis, 329, 331*f*, 380-381, 620*f*
  after apical surgery, 334*f*
  clinical decision making for, 326
  clinical presentation of, 326*f*
  comparative testing of, 331
  cone-beam computed tomography of, 328-329, 402
  cysts as cause of, 326, 327*f*
  diagnosis of, 326-331, 328*f*
  etiology of, 324-326, 325*f*-326*f*
  extraction-replantation for, 381, 382*f*
  extraradicular infection as cause of, 326
  foreign body reaction as cause of, 326
  incidence of, 324
  intraradicular microorganisms as cause of, 325-326
  missed canals as cause of, 373-374, 375*f*
  overextended filling materials as cause of, 388
  premolar with, 328*f*
  pulp vitality testing, 331
  radiographic assessment of, 328, 330*f*, 364*f*
  treatment of
    endodontic procedures, 333
    options for, 331-334
    planning for, 331-334, 331*f*-335*f*
    tooth extraction for, 332, 332*f*
Potassium nitrate, for dentin hypersensitivity, 579, 580*f*
Practice management software, 6, 8*f*
Precementum, 662, 665
Predentin, 537, 537*f*, 782
Prefabricated posts, 337, 825
Pregnancy
  acetaminophen use in, 74
  drugs used during, 74, 74*b*
  local anesthetics in, 94
  treatment planning affected by, 74-75, 74*b*
Premedication, before periradicular surgery, 402-404
Premiere, 802-803, 806*f*
Premolars
  mandibular
    crown of, 154*f*
    first. *See* Mandibular first premolars
    root canal configuration in, 141*f*
    root canal morphology in, 141*f*
    second. *See* Mandibular second premolars
  maxillary first. *See* Maxillary first premolars
  maxillary second. *See* Maxillary second premolars
Pressoreceptors, *e*1
Pretracheal space, 715
Pretrigeminal neuralgia, 693
Prevertebral space, 715
Previous endodontic treatment, symptomatic teeth with, 716
Prevotella spp., 718
Prilocaine
  with 1:200,000 epinephrine, 97
  4%, for inferior alveolar nerve block, 97
Primary afferent neurons, 685-686
Primary dentin, 560-561
Primary pathogens, 602
Probes, 802-803, 806*f*
Probing defect, 802
Procollagen, 541-542
Profile GT obturators, 311-312, 311*f*
ProFile instruments, 226, 228-229, 229*f*, 266
Profile Vortex File, 231
Prognostication
  definition of, 476
  outcomes evaluation use for, 476
Projection neurons, 686-687
Pro-Post drills, 312
Proprioceptors, *e*1
ProRoot MTA, 314*f*, 739
Prostacyclin, 551
Prostaglandins
  bone homeostasis regulation by, 395
  E, 395
  F, 395
  as inflammatory mediators, 551
Prosthetic implants, 77-78
Prosthetic joints, 396
ProTaper Universal, 230, 244
ProTaper Universal obturators, 311-312
ProTaper Universal retreatment files, 350
Proteoglycans, 541
Proton pump inhibitors, 117
Provisional crowns, 846
PRRs. *See* Pattern-recognition receptors
Pseudopocket, 646
Psychiatric disorders, 78
Psychogenic pain, 697
Psychogenic toothache, 696-697
Psychosocial evaluation, 78
Pterygomandibular space, 712*f*-714*f*, 713-715
Pulp
  α-adrenergic receptors in, 557
  age-related changes in, 566, *e*1
  amputation of, *e*1. *See also* Pulpotomy
  anatomy of, 451, *e*2
  arterioles of, 556, 556*f*
  biological irritation of, 583-584
  calcific metamorphosis of, 566, 566*f*
  calcifications of, 372*f*, 564-566, 564*f*-566*f*
  capillary blood flow in, 556, 557*f*
  carious exposure of, 578, 578*f*
  cell-poor zone of, 534
  cell-rich zone of, 534-535, *e*1
  central core of, *e*1
  chemical irritation of, 583-584
  collagen fibers in, 541-542, 542*f*
  collagen in, 541, 542*f*
  configurations of, 130
  connective tissue of, 540-542
  coronal
    calcification of, 564, 564*f*
    inflammation of, *e*7
    odontoblast layer of, 533
  defenses of, against caries, 573-576
  degeneration of, inflammatory processes as cause of, 162
  dendritic cells in, 662
  disease of, clinical classification of, 27-30

Pulp *(Continued)*
elastic fibers of, 541
elastin fibers in, 541
extirpation of, 47*f*
fibrosis of, 564*f*
fibrotic, 372*f*
fluid drainage from, 558-559
ground substance of, 540-541
healing potential of, 560
heat-related injury to, 582-583, 582*f*-583*f*
hyaluronan of, 541
hyperalgesic, 551-552
immune response of, 577
innervation of, 542-546, 542*f*-543*f*, 543*t*, 687-689
interstitium of, 540-541, 540*f*
intraligamentary anesthesia injection effects on, 109
metabolism of, 539-540
morphologic zones of, 532-535, 533*f*-534*f*
muscarinic receptors in, 557-558
neuroimmune interactions in, 552
nociceptors of, 689
normal, 28
odontoblast layer of, 532-534, 533*f*-534*f*, 577*f*
opioid receptors in, 578-579
pathosis of, 281
proteoglycans in, 541
radicular
age-related changes of, 566
calcification of, 564, 564*f*
nerve bundles through, 544-545, 544*f*
odontoblast layer of, 533, 534*f*
regenerative potential of, 453, 457, 457*f*
repair of, 560-564, 561*f*-564*f*
restorative materials effect on, 586-587, 587*f*
revitalization of, 772-773
sensory system of, 542
stem cells of, 449, 450*f*, 451, 457
structural proteins in, 541
substance P in, 577
transcapillary fluid exchange, 558-559
vascular supply of, 556-560, 556*f*-558*f*
zones of, 532-535, 533*f*-534*f*, *e*1
Pulp capping
crown fracture treated with, 766-767
definition of, 766
direct
bonding agents for, *e*2
calcium hydroxide for, *e*2
cariogenic bacteria survival after, *e*7
definition of, *e*1
hemostatic agents for, 588
of mandibular molars, *e*6*f*
mineral trioxide aggregate for, 587-588, *e*7, *e*8*f*, *e*13*f*
outcomes of, 483, 488*t*-490*t*
partial pulpotomy versus, *e*2
indications for, 766-767
indirect
boundary point for, *e*2
case selection for, *e*2
definition of, *e*2
indications for, *e*2
outcomes of, 480-482, 485*f*, 486*t*, 491*f*
stepwise excavation, 480
Pulp capping *(Continued)*
mineral trioxide aggregate for, 587-588, 766, *e*7, *e*10-*e*16
one-step, *e*10-*e*11
partial pulpotomy versus, 766-767
repair process after, *e*1
two-visit, *e*10-*e*16, *e*26*f*
Pulp chamber
anatomy of, 132, 136*f*
calcification of
access cavity preparation in, 162-163, 163*f*-164*f*
illustration of, 163*f*
floor of
description of, 145-147
types of, 203*f*, 205
visual inspection of, 157, 159*f*
pulp stones in, 565*f*
roof of, 151, 151*f*
walls of, 147
Pulp horns
anatomy of, 132
blood vessels in, 557*f*
intratubular nerve endings in, 545
Pulp proper, 535
Pulp regeneration
animal models of, 457
potential for, 453, 457, 457*f*
Pulp stones, 564-565, 565*f*
Pulp testing
carbon dioxide for, 16, 16*f*
cold testing, 16, 16*f*-17*f*, 547
electric, 16-19, 18*b*, 18*f*, 92, 547, 578
heat methods for, 16-17, 17*f*
importance of, 15
lack of response to, 578
laser Doppler flowmetry for, 19
in posttreatment disease, 331
for pulpal necrosis, 29
pulse oximeter for, 19, 19*f*
refrigerant spray for, 16, 17*f*
sensitivity of, 18-19
test cavity method for, 20
thermal methods of, 15-19, 16*f*, 759
Pulp vitality
endodontic prognosis and, 78
obturation timing based on, 284
reestablishment of, 780
testing of. *See also* Pulp testing
description of, 17-18, 331, 784
Pulpal anesthesia
articaine for, 99
confirming of, 95, 95*f*
inferior alveolar nerve block for, 96-97, 97*f*
maxillary, 102-104
Pulpal blood flow
age-related decreases in, 585-586
anatomy of, 556*f*-557*f*, 557
electrical stimulation effects on, 557
humoral control of, 558
laser Doppler flowmetry assessments of, 19, 759-760, 760*f*
Le Fort I osteotomy effects on, 592
local control of, 558
orthodontic forces effect on, 592
postural effects on, 560
regulation of, 557-558, 558*f*
Pulpal blood flow *(Continued)*
resting, 557
vasoconstrictors effect on, 579-581, 580*f*
Pulpal canal obliteration, 779, 779*f*
Pulpal cells
calcification effects on, 566
connective tissue replacement of, 563, 564*f*
dendritic cells, 538-539, 538*f*-539*f*, 575-576
fibroblasts, 537-538
lymphocyte, 539, 540*f*
macrophages, 538, 538*f*
mast cell, 539
odontoblast process, 535-537, 575-576
odontoblasts. *See* Odontoblast(s)
Pulpal exposure
caries as cause of
description of, 578
direct pulp capping for, 587-588
disinfectants effect on, 588
hemostatic agents effect on, 588
illustration of, 578*f*, 603*f*
in children, *e*16*f*
endodontic infections caused by, 601
Pulpal inflammation
in apical root canal, 637*f*
caries as cause of, 575, 575*f*
clinical symptoms and, 578-579
description of, 559-560
illustration of, 603*f*
irreversible, 578
mediators of, 578
periodontitis as cause of, 591-592
preoperative, 581-582
reparative dentin associated with, 562
vital pulp therapy in, 765
Pulpal irritation
access cavity preparation as cause of, 585
biological causes of, 583-584
calcium hydroxide as cause of, 586
chemicals as cause of, 583-584
dentin permeability and, 585
description of, 582-585
indirect factors that cause, 587
restorative materials as cause of, 586
Pulpal necrosis. *See also* Intraradicular infection
acute apical abscess with, 708-711
anesthetizing symptomatic teeth with, 112
apical periodontitis after, 637-638
asymptomatic teeth with
anesthetizing of, 112
after avulsion injury, 785
bacterial contamination in, 284
burn lesion with, 583*f*
Calcium hydroxide as cause of, 285, 765-766
canine space infection as cause of, 9
crown fracture as cause of, 764, 764*f*
definition of, 29
diagnosis of, 578
endodontic infections caused by, 602
fracture-induced, 797*f*
illustration of, 603*f*
in immature teeth, 314-315
luxation injuries as cause of, 779
obturation in, 284-285
periodontal disease as cause of, 601
previously initiated therapy category of, 29
previously therapy category of, 29

Pulpal necrosis *(Continued)*
pulp testing for, 29
pulpal inflammation as cause of, 560
revitalization of, 772-773
risks for, 447-448
root fractures as cause of, 777
root resorption and, 286*f*
in sickle cell anemia, 652
symptomatic teeth with, anesthetizing of, 112
Pulpal nerve fibers
description of, 545-546, 545*f*
neuropeptides in, 547
Pulpal pain, 689
Pulpal reactions
age of patient effects on, 585-586
to caries, 573-576, *e*2-*e*6
to glass ionomer materials, 586-587
to implant placement and function, 593-594
to laser procedures, 588-590
to local anesthetics, 579-581, 580*f*-581*f*
neurogenic mediators involved in, 576-578
to orthodontics, 592
to orthognathic surgery, 592
to parafunctional habits, 592-593
to periodontal procedures, 591-592
to restorative materials, 586-587, 587*f*
to restorative procedures
age of patient, 585-586
dentin permeability effects, 585
desiccation, 583, 586
heat, 582-583
odontoblastic layer, 585
overview of, 581
physical irritation, 582-584
preoperative pulpal inflammation, 581-582
proximity of procedures to dental pulp and surface area of dentin exposed, 584-585
to vital bleaching procedures, 590-591
Pulpal space
configurations of, 137
infection of, 779-781, 780*f*
morphology of, 131
Pulp-dentin complex
definition of, *e*1
description of, 532, 600
nerve fibers in, 545, 545*f*
odontoblasts in, 451
odontogenic pain caused by, 687-689
regeneration of, 458
Pulpectomy
definition of, 769
in immature teeth, *e*5
indications for, 769
in mature tooth, 764-765
after pulpotomy, 769*f*
Pulpitis
apical, 637-638
definition of, 28
hyperplastic, 576, 577*f*
irreversible. *See* Irreversible pulpitis
pain associated with, 551-552
painless, 578
reversible, 28
case study of, 703
causes of, 707
description of, 28
vasoactive neuropeptides, *e*1
Pulpotomy
calcific bridge formation after, 477*f*
cervical, 768
definition of, *e*1-*e*2
formocresol, *e*2
full
definition of, 767-768
follow-up, 767-768
indications for, 767
outcomes evaluation of, 494*t*
technique for, 767
indications for, *e*2
intrapulpal anesthesia for, 581
irreversible pulpitis treated with, 112
laser, 589-590
mineral trioxide aggregate for, *e*7, *e*14*f*, *e*16*f*
outcomes evaluation of, 483-484, 491*f*, 492*t*
partial
calcium hydroxide, 766*f*
crown fractures treated with, 767, 768*f*-769*f*
Cvek, 767, 768*f*
definition of, *e*2
follow-up after, 767, 769*f*
outcomes evaluation of, 483-484, 491*f*, 492*t*
pulp capping versus, 766-767
root development after, 769*f*
technique for, 767, 768*f*
traumatic pulp exposures treated with, 767, 768*f*-769*f*
pulpectomy after, 769*f*
Pulse oximeter, for pulp vitality assessments, 19, 19*f*

Q

QMiX, 259-260
Quality of life
nonsurgical root canal treatment effects on, 515, 517*t*
periapical surgery effects on, 522

R

RaCe file, 231
Racellets, 413, 423*f*
Radiation absorbed dose, 401-402
Radiation dose, 35, 35*b*
Radiation therapy
caries caused by, 652
osteonecrosis of the jaw caused by, 655
wound healing affected by, 75-76
Radicular cysts
asymptomatic apical periodontitis and, 646-649, 649*f*, 654
bacteria in, 649*f*
Radicular dentin
description of, 811, 811*f*
luting cement adhesion to, 828
root canal cleaning and shaping objectives for, 211
Radicular pulp
age-related changes of, 566
calcification of, 564, 564*f*
nerve bundles through, 544-545, 544*f*
odontoblast layer of, 533, 534*f*
Radicular space, 282
Radiographs
cone-beam computerized tomography and, 24, 25*f*
deep caries, *e*5*f*, *e*16*f*
digital
advantages of, 33
description of, 22-23, 23*f*, 33
file formats for, 36
external cervical resorption on, 668-672, 668*f*-673*f*
external inflammatory resorption on, 665-666
external root resorption on, 783, 783*f*-784*f*
image characteristics and processing, 35-36
internal root resorption on, 671, 676*f*, 677, 680*f*, 782, 782*f*-783*f*
intraoral, 20-22, 22*f*
luxation injuries, 760*f*, 762, 763*f*
posttreatment disease diagnosis using, 328, 330*f*
principles of, 21
root fractures, 774, 774*f*-775*f*
root resorption, 330*f*, 760-762, 761*f*-762*f*
shortcomings of, 22
traumatic dental injury evaluations, 760-762, 761*f*-763*f*
vertical root fractures, 800*f*, 802, 802*f*, 804*f*, 806, 807*f*-808*f*
Radiography
computed, 34-35
digital modalities for, 33
direct digital, 35
modalities for, 33-35
radiation dose during, 35, 35*b*
Radix entomolaris, 198-199, 198*f*, 731*f*
Rake angle, 216, 219*f*
RANKL, 643, 662
Ratio measurement method, 237-238
RC-Prep, 262
Reactionary dentin, 561, 574-575, 574*f*
RealSeal SE, 294
Reamers, 223, 225
Reciproc, 232
Rectified turpentine, for gutta-percha removal, 347-348, 348*f*
Reductionism, 607
Referred pain, 30, 684, 688*f*, 699
Refrigerant spray
cold testing of pulp using, 16, 17*f*
pulpal anesthesia testing using, 95*f*
Regenerative endodontic procedures
case studies of, 458-459, 461*f*-463*f*
clinical studies on, 460
definition of, 448
goal of, 467
intracanal medicaments used with, 459-460, 459*f*, 462-464
outcome measures for, 467-468
overview of, 460-464
research on, 468-469
revascularization protocol, 464-467, 466*f*
root development after, 468
root length after, 460-462, 461*f*-464*f*, 468
root wall thickening after, 460-462, 461*f*-464*f*, 468
success rates for, 467
summary of, 468-469
terminology associated with, 458

Regenerative endodontic procedures *(Continued)*
treatment procedures, 464-467, 466*f*, 467*b*
triple antibiotic paste, 459-460, 459*f*
Regenerative endodontics/dentistry
clinical studies on, 458-468
contemporary, 448
delivery system, 455-456
goal of, 447
growth factors, 452-455, 453*t*-454*t*
history of, 447
morphogens, 453-455
overview of, 447-448
preclinical studies on, 448-458
principles of, 448
procedures. *See* Regenerative endodontic procedures
research on, 458, 468-469
scaffolds, 455
stem cells in, 448-452
summary of, 468-469
translational studies, 456-457, 457*f*
Remaining dentin thickness, 584, 763
Removable partial dentures, 332
Reparative bridge formation, *e*2-*e*3, *e*4*f*
Reparative dentin, 132, 133*f*, 549-550, 560-561, 561*f*-563*f*, 563, 575*f*, *e*2-*e*3
Reparative dentinogenesis, 574
Replantation, of avulsed teeth, 785
Resilon, 299-300, 300*f*, 351, 351*f*
Resin cements
placement of, pulpal irritation during, 584
self-adhesive, 293
self-etching, 293
Resin composites, 821-822
Resin sealers
epoxy, 292, 293*f*
methacrylate, 292-294
obturation uses of, 292-294, 293*f*
Resin-based luting cements, 827-828
Resin-modified glass ionomer cements, 826, *e*5-*e*7
Resorbable membranes, 434, 435*t*
Restorations
access cavity preparation in, 159-162, 161*f*-162*f*
adhesive systems, 844
aesthetic evaluation and requirements, 829-830
amalgam, *e*10
biomechanical evaluation for, 829
clinical procedures for, 834-846, 835*f*-843*f*, 845*f*
clinical protocols for, 830*t*, 833*t*
coronal leakage of, 159
crowns, 822-827, 846
defective, removal before access cavity preparation, 146-147, 147*f*, 159
direct composite, 821-822, 835*f*-836*f*
endocrown, 822, 830*t*, 839*f*
full crowns, 822-827
guidelines for, 830
heat produced by, 582-583, 582*f*-583*f*
indirect, 822
intracoronal, 798, 838
in nonvital posterior teeth with minimal/reduced tissue loss, 830-833
occlusal force evaluations, 829
onlays and overlays, 822, 830*t*, 838*f*
Restorations *(Continued)*
partial, 844
periapical healing and, 515
periodontal evaluation before, 829
permanent, 789, *e*10-*e*11
physical irritation caused by, 582-584
pretreatment evaluation, 828-830, 829*f*, 830*t*
principles for, 830
removal of, 146-147, 147*f*, 159, 162*f*
in structurally compromised anterior teeth, 834
in structurally compromised posterior teeth, 834
in structurally sound anterior teeth, 830
temporary, 789, 846
tooth position evaluations, 829
tooth preparation for, 838
treatment strategy for, 830, 831*f*-832*f*
veneer, 830*t*, 837*f*
Restorative materials, pulpal reactions to, 586-587, 587*f*
Retractors, 406*f*
Retreatment
case examples of, 79, 80*f*-81*f*
considerations before, 79
illustration of, 79*f*
plan for, 79, 80*f*-81*f*
plastic carriers used in, 312
prognosis for, 79
Retropharyngeal space, 712*f*-714*f*, 715
Retroplast, 420-421
Retrovisceral space, 715
Revascularization protocol, 464-467, 466*f*
Reversible pulpitis
case study of, 703
causes of, 707
description of, 28
diagnosis of, *e*5
irreversible pulpitis and
differential diagnosis of, *e*5
sodium hypochlorite for differentiation of, *e*5
sodium hypochlorite for diagnosis of, *e*5
vital pulp therapy for, *e*2. *See also* Vital pulp therapy
Richwil Crown & Bridge Remover, 336, 338*f*
Ridge preservation, in periradicular surgery, 435
RinsEndo system, 264
Root
apical third of, 143-144, 144*f*, 144*t*
curved, 87*f*
post placement and selection affected by, 823
preparation of, 786-787
radiographic evaluation of, 21
Root apex
apical constriction and, 143*t*
illustration of, 142*f*-143*f*
Root canal(s)
accessory, 132-133
anastomoses, 140
anatomy of, 132-144
apical part
anatomy of, 142
disinfection of, 240
pulpal inflammation in, 637*f*
Root canal(s) *(Continued)*
bacteria in
after chemomechanical procedures, 620
ecosystem of, 616-619, 618*f*
nutrient sources for, 617-618
nutritional interactions, 618-619, 618*f*
blockage of, 268-269, 371, 372*f*, 374*f*
calcification of
access cavity preparation in, 162-163, 163*f*-164*f*
cone-beam computed tomography of, 49-50
removal of calcified material, 163
cone beam computed tomography of, 130-131
configuration of, 136*f*-138*f*
apical considerations, 142-144
clinical determination of, 140-144
coronal considerations, 140, 140*f*
C-shaped, 137*f*, 140*f*, 199-205, 203*f*-204*f*
midroot considerations, 140-141, 141*f*
S-shaped, 132
supplemental, 138*f*
coronal anatomy of, 140, 140*f*
C-shaped
access cavity preparation in teeth with, 199-205, 203*f*-204*f*
illustration of, 137*f*, 140*f*
curvature of, 132
dental operating microscope view of, 131, 132*f*
diameter of
description of, 143, 144*t*
irrigation affected by, 249
embryology of, 132
empty, 806, 807*f*
furcation, 133, 133*f*
impediments of, 371-373
irrigation of. *See* Irrigation
isthmuses, 141, 141*f*
ledged, 372*f*
materials extending beyond, 46-48, 50*f*
midroot anatomy of, 140-141, 141*f*
missed, 373-374, 375*f*
multiple, 87*f*
obstructions of
dental injection needle for removal of, 365-366, 368*f*
ultrasonic instruments for removal of, 365, 366*f*
obturation of. *See* Obturation
orifices of
coronal flaring of, 152, 152*f*, 156-157
identification of, 140, 140*f*, 145-146, 156, 157*f*
instrumentation for, 147, 149
"mouse hole" effect, 156, 157*f*
rotary nickel-titanium openers for, 152
straight-line access determination, 152, 153*f*
overfilling of, 46
pathways of, 136-137
sealers for. *See* Sealers
transportation, 267-268
Root canal infection
bacteria that cause, 616-617
primary, 632
routes of, 600-602

Root canal system
components of, 132-133, 132*f*
cone beam computed tomography of, 46, 49*f*
sealing of, 280-283
Root canal system cleaning and shaping
access cavity preparation. *See* Access cavity preparation
bacteria after, 281
biologic objective of, 211-212, 214*f*
canal enlargement/preparation
apical preparations, 238-240, 239*t*
balanced force technique for, 243, 243*f*, 737
crown-down technique, 241-243, 267
danger zones in, 241
hybrid techniques for, 245-248, 246*b*, 246*f*-249*f*
LightSpeed technique for, 244-245, 246*f*
nickel-titanium instrumentation techniques, 244-248
rationale for, 238-241
rotary instrumentation for, 241, 243, 244*f*
self-adjusting file for, 245
standardized technique for, 241, 242*f*
step-back technique, 241, 242*f*, 737
step-down technique, 241
strategies for, 241
vertical root fracture risks, 811
case studies of, 269-270, 269*f*-270*f*
cervical dentin goals, 211
clinical issues with, 212-213, 215*f*
coronal preflaring, 235-236, 236*f*-237*f*
criteria for evaluating, 265-269
disinfection
antimicrobial nanoparticles for, 265
description of, 373-374
intracanal medications for
bioactive glass, 262
calcium hydroxide. *See* Calcium hydroxide
chlorhexidine. *See* Chlorhexidine
formaldehyde, 261
halogens, 261
lubricants, 262
phenolic preparations, 261
steroids, 261-262
triple-antibiotic paste, 262
irrigants. *See* Irrigants
irrigation. *See* Irrigation
manually activated techniques for, 263
photoactivation, 265
superoxidized water for, 265
syringe delivery for, 262
final apical enlargement, 248
fluid dispersion in, 211-212, 214*f*
instruments for
alloys used in, 219-222, 222*f*
barbed broaches, 224, 224*f*
breakage of, 234*b*
characteristics of, 213-222
cross-sectional design of, 216-217, 219*f*
design elements of, 213-222
Endo-Eze, 232
EndoSequence, 231
engine-driven, 225-235, 226*t*, 228*f*-229*f*
FlexMaster, 230-231
fracture of, 226, 227*f*, 266-267
Gates-Glidden drills, 225, 225*f*
Root canal system cleaning and shaping *(Continued)*
GT file, 229
GTX file, 229
HERO 642, 230
Hero Shaper, 230
H-type, 223-224, 224*f*
irrigation needles, 214*f*
ISO norms for, 217-219, 220*f*
K3, 229-230
K-type, 223
LightSpeed file, 227, 266
longitudinal design of, 216-217, 219*f*
low-speed engine-driven, 224-225
manually operated, 223-224
motion of, 234
motors, 233-235, 233*f*
MTwo, 231-232
Peeso drills, 225
ProFile, 228-229, 229*f*
Profile Vortex File, 231
ProTaper Universal, 230
RaCe file, 231
reamers, 223, 225
Reciproc, 232
rotary, 226-235, 226*t*, 228*f*-229*f*
rotational speed of, 234
SAF, 228*f*
self-adjusting file, 232
taper of, 217
tip design of, 214-216, 218*f*
Twisted File, 231
WaveOne, 232
mechanical objective of, 210-211, 213*f*
microorganisms, 209-210
noninstrumentation technique for, 212, 215*f*
objectives of, 209-212, 210*b*
obturation after, 285. *See also* Obturation
patency file for, 236-237
principles of, 209-213
problems during
blockage, 268-269
canal transportation, 267-268
instrument fracture, 226, 227*f*, 266-267
perforation, 268, 268*f*
protocol for, 269
radicular dentin goals, 211
smear layer removal after, 288-289, 289*f*
surface irregularities created during, 221
technical objective of, 212
well-shaped canals from, 265-266
working length determination, 237-238, 238*f*, 248
Root canal treatment
access cavity preparation for. *See* Access cavity preparation
bacteria effects on success of, 499-504, 500*t*-503*t*
chemomechanical debridement, 666
culture test, 478
description of, 209
diagnostic outcome of, 282-283
hermetic seal as goal of, 283
illustration of, 212*f*
of mandibular molars, 210*f*
microbial penetration during, 602
Root canal treatment *(Continued)*
nonsurgical
outcome measures for, 477-479
periapical wound healing after, 652-654
periapical disease after, 477-478, 478*f*
periapical healing after, 282-283
periapical healing after, factors that affect, 495-509
acute exacerbation during treatment, 505
apical preparation, 497-498
bacteria presence, 499-504, 500*t*-503*t*
illumination, 497
irrigants, 498-499
isolation, 497
magnification, 497
mechanical preparation, 497-498
medicaments, 499
number of treatment visits, 505
patient-related, 495-497
root canal bacterial culture, 499, 500*t*-503*t*
root filling, 504-505
root filling material and technique, 504-505
summary of, 506-509, 506*f*-509*f*
treatment-related, 497-505
periapical radiolucency and, 78
restorative factors that affect, 505-509
success rates for, 324
tooth survival after, factors that affect
description of, 509-515
diabetes mellitus, 509-512
patient factors, 509-512
preoperative conditions of teeth, 513-515
restorative factors, 515
summary of, 515, 516*f*
tooth morphologic type and location, 512-513
treatment factors, 515
Root development
after partial pulpotomy, 769*f*
after regenerative endodontic procedures, 468
Root end
anatomy of, 414
angle of resection, 415, 415*f*
apexification. *See* Apexification
burs for, 416
cavity preparation. *See* Periradicular surgery, root-end cavity preparation
conditioning of, 416
filling materials for. *See* Periradicular surgery, root-end filling materials
goals for, 416
management of, 414-416
perpendicular resection of, 415*f*
resection of, 414-415, 414*f*-415*f*
surface preparation of, 416
surface topography of, 416
Root filling
apical extent of, 504-505
office visit for, 789
quality of, 505
soft technique for, 772*f*
Root filling materials
aesthetic appearance of teeth affected by, 821
heat used to soften, 370

Root filling materials *(Continued)*
neuritis caused by, 694
overextended, 388
removal of, 346-361, 347*f*-355*f*, 357*f*-361*f*
treatment outcomes affected by, 504-505
Root fractures
apical, 774-777
causes of, 823-824
clinical presentation of, 774
complications of, 774-777
cone beam computed tomography diagnosis of, 51-52, 329
coronal, 774, 776*f*
definition of, 774
description of, 759
diagnosis of, 51-52, 329, 391*f*, 774, 775*f*
exploratory surgery to diagnose, 391*f*
facial, 775*f*
fatigue, 798, 823-824
follow-up for, 777
healing of, 52, 774, 776*f*
horizontal (transverse), 762, 763*f*, 774, 776*f*
internal root resorption in, 776*f*
intra-alveolar, 52
midroot, 774-777, 800
prognosis for, 777
pulpal necrosis caused by, 777
radiographs of, 774, 774*f*-775*f*
treatment of, 774
ultrasonic root-end preparation as cause of, 417-418
vertical. *See* Vertical root fractures
Root lesions, apical, 161*f*
Root perforations, 26-27
in apical portion of canal, 378
cervical, 374-375
cone-beam computed tomography of, 50-51, 681
coronal-radicular access to, 377
in deep portion of canal, 377
definition of, 26
furcal, 378*f*
materials used to repair, 376, 376*f*-377*f*
midroot, 374-375, 379*f*-380*f*
mineral trioxide aggregate for, 26-27, 147, 376-378, 377*f*, 681
repair of, 374-378, 375*f*-379*f*
sealing of, 26-27
treatment of, 26-27
Root planing, 592
Root resorption
causes of, 54
cementum involvement in, 653
cone beam computed tomography of, 52-55, 54*f*
definition of, 660
external. *See* External root resorption
histologic features of, 660-662, 661*f*
inflammatory, 780*f*
initiating factors of, 660
internal. *See* Internal root resorption
odontoclasts in, 660-662, 661*f*
osteoclasts in, 660
pulpal necrosis and, 286*f*
radiographs of, 330*f*, 760-762, 761*f*-762*f*, 781*f*
types of, 660
Root wall thickening, after regenerative endodontic procedures, 460-462, 461*f*-464*f*, 468
Root-end filling
periapical health after, 517-522, 521*f*
placement of, 407*f*
tooth survival after, 522
Root-end filling materials
bioceramics, 422
bonded, 418-419, 419*f*
composite resins, 420-421
Diaket, 420
Dyract, 421
Geristore, 421
glass ionomer cements, 420
ideal characteristics of, 419
mineral trioxide aggregate, 421-422
overextension of, 742
overview of, 422
radicular extrusion of
description of, 739
illustration of, 743*f*
management of, 742-745
obturation as cause of, 740, 742-744
tissue damage caused by, 744-745
resin-ionomer hybrids, 420-421
zinc oxide–eugenol cement, 419-420
Ropivacaine, 98
Rotary instruments
description of, 226-235, 226*t*, 228*f*-229*f*
fracture of, 266-267
gutta-percha removal using, 349-350, 350*f*, 355
nickel-titanium, 214-216, 218*f*-219*f*, 226, 233*f*, 266, 811*f*
stress on, 364*f*
Rotary nickel-titanium orifice openers, 152
Rotated teeth, access cavity preparation in, 163-165
Roto-Pro bur, 340, 341*f*
Round carbide burs, 148, 148*f*, 411
Round diamond burs, 148, 149*f*
Roydent Extractor System, 367-369, 369*f*
Rubber dam, periapical healing affected by, 497
Ruddle Post Removal System, 341, 344*f*
Ruffled border, of osteoclasts, 660, 661*f*

S

Saccharolytic bacterial species, 618
SAF instrument, 228*f*
Safety-Irrigator, 264, 264*f*
Salivary gland pain, 691
Salivary gland stem cells, 449, 450*f*
Sargenti paste, 694
Scaffolds, 455
Scheduling of appointments, 88
Schwann cells, 545*f*, 575-576
Scotchbond Multi-Purpose Plus, 766
Sealers
calcium hydroxide, 292
calcium silicate, 294-295, 295*t*-296*t*
epoxy resin, 292, 293*f*
extrusion of, 291, 291*f*
glass ionomer, 292, 292*f*-293*f*
ideal properties of, 290*b*
lentulo spiral for placement of, 296, 297*f*
medicated, 295-296, 297*f*
methacrylate resin, 292-294
Sealers *(Continued)*
noneugenol, 292
paraformaldehyde-containing, 295-296
penetration depth of, 288
periradicular extrusion of, 291, 291*f*
placement of, 296, 297*f*
radiopacity of, 290
resin
epoxy, 292, 293*f*
methacrylate, 292-294
obturation uses of, 292-294, 293*f*
silicone, 294, 294*f*
smear layer effects on action of, 288
tricalcium silicate, 294, 295*t*-296*t*
zinc oxide–eugenol
formula for, 292*t*
obturation uses of, 291-292, 292*t*
Sealing, of root perforations, 26-27
Second division nerve block, 103
Secondary dentin, 560-563, 566
Secondary hyperalgesia, 687, 690-691
Second-order neurons, 686-687
Sedation, conscious
nitrous oxide for, 113
oral, with triazolam and alprazolam, 113
for periradicular surgery, 402-404
Selective anesthesia, 20
Self-adhesive cements, 828
Self-adjusting file, 226, 228*f*, 232, 245
Self-curing luting cements, 827-828
Self-etching adhesive systems, 844, *e*10
Self-etching formulations, 583-584
Sensitization
central, 553-555, 687, 688*b*, 695
peripheral, 550, 550*b*, 550*t*, 687, 688*b*, 695
Sensory fibers, 543*t*, 635, 641
Separated instruments
causes of, 361-363, 364*f*, 725-726
chemical removal, 726
dental injection needle for removal of, 365-366, 368*f*
description of, 724-725
Endo Extractor kit for removal of, 366-367, 368*f*, 729-731
files, 365, 367*f*-368*f*, 726-727, 728*f*
Hedstrom files for removal of, 365, 367*f*
illustration of, 366*f*
improper use as cause of, 362, 725-726
Instrument Removal System for removal of, 369-370, 370*f*
management of, 726-727
in mandibular molars, 726
Masserann technique for removal of, 367, 726-727
nickel-titanium instruments, 727-728
operating microscope used in removal of, 369, 369*f*
prevention of, 363
prognosis for, 364-365, 733
radiograph of, 725*f*-726*f*
removal techniques for, 365-370, 365*f*-367*f*, 727-733, 728*f*-732*f*
S.I.R. System for retrieval of, 370
surgical approaches for management of, 731-733
ultrasonic instruments for removal of, 365, 366*f*, 727

Septocaine. *See* Articaine, with 1:100,000 epinephrine
Serotonin, 392
Sialoproteins
  bone, 452-453
  dentin, 451-453
Sickle cell anemia, 5-6, 652
Sievert, 401-402
Silicone sealers, 294, 294*f*
Silver cones, 296-297, 298*f*
Silver points
  apical point of, 361, 363*f*
  corrosion of, 358-359, 358*f*
  disadvantages of, 358-359
  Gates-Glidden burs for removal of, 360
  Hedstrom files for removal of, 360, 360*f*
  persistent disease with, 358-359, 358*f*-359*f*
  removal of, 358-361, 358*f*-363*f*
  separated, 363*f*
  trephine burs for removal of, 360
SimpliFill, 313, 313*f*
Single amino-acid polymorphisms, 651
Single nucleotide polymorphisms, 651
Single-visit treatment
  description of, 79-82
  for irreversible pulpitis, 709
Sinus pain, 691, 700
Sinus palpation, 700
Sinus tracts
  coronally located, 801*f*, 803-805
  definition of, 12
  extraoral, 10-11, 11*f*-12*f*
  intraoral, 12-13, 14*f*
  localization of, 13, 14*f*
  periapical infection with, 13
  periapical radiograph of, 401*f*
  stoma of, 12-13
S.I.R. System, 370
Site of the pain, 684
16S rRNA, 611
Sjögren syndrome, 652
Small integrin-binding, ligand N-linked glycoproteins, 452-453
Smear layer
  bacterial adhesion affected by, 288
  contamination of, 288
  irrigants affected by, 288
  removal of
    before obturation, 288, 289*f*, 311
    citric acid for, 288-289, 416
    ethylenediamine tetraacetic acid for, 289
    root-end conditioning for, 416
    tetracycline for, 416
Sodium hypochlorite
  allergic reactions to, 252
  calcium hydroxide and, 679
  "champagne bubble" test, 131, 163
  chemicomechanical debridement of root canal using, 679
  chlorhexidine and, 256*f*
  complications of, 722-724, 723*f*-724*f*
  concentrations of, 253
  description of, 770
  ethylenediamine tetra-acetic acid and, 258
  extrusion of, 723-724, 723*f*
  heating of syringes for, 252-253, 253*f*
  hemostasis using, *e*7, *e*21*f*, *e*23*f*-*e*24*f*
  history of, 251
Sodium hypochlorite *(Continued)*
  hypersensitivity to, 252
  hypochlorous acid formation, 251-252
  iatrogenic events caused by, 722-724, 723*f*-724*f*
  mode of action, 251-252, 252*f*
  neutralization reaction, 251
  pH of, 252
  pulp tissue dissolution capacity of, 253
  pulpitis diagnosis using, *e*5
  root canal retreatment success affected by, 498-499
  saponification reaction, 251
  temperature effects on, 252-253, 252*f*-253*f*
  time course of, 253
  toxicity of, 253-254, 254*f*
Soft tissue
  healing of, 391-393
  intraoral, examination of, 11
Software, dental history using, 6, 8*f*
Solid core carriers, 355, 355*f*
Solvents, for gutta-percha removal, 347-349, 348*f*
Somatic pain, 690-692
Sonically activated irrigation, 263, 263*f*
Source of the pain, 684
Space-occupying lesions, 697
Spatial resolution, 35, 42, 43*f*
Spirochetes, 604, 605*f*
Splinting, 787, 787*f*
Split tooth
  definition of, 796-797
  description of, 796
  diagonally, 798, 799*f*
Split tooth/roots, 26
Spoon, endodontic, 150, 150*f*
Spot desmosomes, 533-534
Staining, for crack detection, 20
Stainless steel carriers, 352, 352*f*
Stainless steel files, 223, 224*f*, 237*f*, 246-247
Stainless steel hand spreader, 812
Stainless steel post, 338*f*
Staphylococcus aureus, 643
Steiglitz forceps, 352-353, 352*f*, 359*f*
Stem cells
  apical papilla, 449-451, 450*f*, 460
  bone marrow, 449
  definition of, 448-449, 537
  dental follicle, 449, 450*f*
  dental pulp, 449, 450*f*
  embryonic, 448-449
  epithelial, 449
  gingival-derived mesenchymal, 449
  from human exfoliated deciduous teeth, 449
  mesenchymal. *See* Mesenchymal stem cells
  multipotent, 448-449
  periodontal ligament, 449-450, 450*f*
  periosteal derived, 449, 450*f*
  pluripotent, 448-449
  populations of, 449
  regenerative endodontics use of, 448-452
  salivary gland, 449, 450*f*
Step-back technique, for canal enlargement/preparation, 241, 242*f*, 737
Sterilox, 265
Steroids, for root canal disinfection, 261-262
Stieglitz Pliers, 365
Stoma
  definition of, 12
  sinus tract, 12-13
Straight-line access cavity preparation, 145, 145*f*
Strain measurement, 813*f*
Stromal derived factor 1, 457
Stropko syringe, 422*f*
Subcutaneous emphysema, cervicofacial, 748-750, 749*f*-750*f*
Subepithelial external root resorption, 667
Sublingual space, 712-713, 712*f*-714*f*
Subluxation, 777
Submandibular lymph nodes
  palpation of, 9
  tuberculosis involvement of, 5-6
Submandibular space, 712*f*-714*f*, 713
Submarginal flap, 410*f*, 424-425
Submasseteric space, 712*f*-714*f*, 713
Submental space
  description of, 712, 712*f*-714*f*
  swelling of, 10, 10*f*
Subnucleus caudalis, 686
Subnucleus caudalis neurons, 553-554
Subnucleus interpolaris neurons, 553-554
Subnucleus oralis neurons, 553-554
Substance P, 577, 635, 653*t*
SuccessFil, 313, 313*f*
Sulfite-induced reactions, 94
Super-EBA, 419-420
Superficial pain, 699
Superior cervical ganglion, 542
Superoxidized water, 265
Supraosseous extracanal invasive resorption, 667
Surface irregularities, from root canal system cleaning and shaping, 221
Surgery
  American Society of Anesthesiologists risk system for, 395-396
  periradicular. *See* Periradicular surgery
Surgical debridement, 654
Surgical retreatment
  nonsurgical retreatment and, 333
  outcome of, 517-522
  periapical wound healing after, 654
Surgicel, 412-413
Sutures
  for periradicular surgery, 425-426, 436
  removal of, 436
  resorbable, 425-426
Swelling
  with acute periradicular abscess, 710
  extraoral, 9*f*-12*f*
  intraoral, 11, 12*f*-13*f*
  midface, 715
  mucobuccal fold, 11, 13*f*
Switch recombination, 635
Sympathetically maintained pain, 696
Symptomatic apical periodontitis, 29
Symptomatic irreversible pulpitis
  anesthesia in, 111
  asymptomatic irreversible pulpitis versus, 109
  description of, 28-29
  inferior alveolar nerve block in, 109
  in mandibular posterior teeth, 113
Symptomatic vital teeth, pulp anesthesia determinations in, 95

Syndrome, 26
Synthetic graft, 435*t*
Systemic diseases, 650-652
Systemic medications, wound healing affected by, 394-395

T
Taper, 217, 220*f*, 268*f*
Taper lock, 267
T-cell antigen receptors, 634
Teeth. *See also specific maxillary and mandibular teeth; specific tooth entries*
  abutment. *See* Abutment teeth
  anatomy of, 690
  anterior. *See* Anterior teeth
  avulsed. *See* Avulsed teeth
  crack. *See* Crack(s); Cracked tooth syndrome
  endodontically treated. *See* Endodontically treated teeth
  leaving open, 716-717, 717*f*-718*f*
  nonrestorable, 333, 333*f*
  posterior. *See* Posterior teeth
  sympathetic innervation of, 542
Telopeptide of the procollagen molecule, 541-542
Temporal space, 712*f*-714*f*, 713
Temporary restoration, 789, 846
Tension-type headaches, 691-692
Terminal periodontal disease, 333
Terminal restriction fragment length polymorphism, 611
Tertiary dentin
  description of, 560-562
  formation of, 574-575
Tertiary dentinogenesis, 573-574
Test cavity method, for pulp vitality assessments, 20
Tetraclean, 258-259
Tetracycline
  root-end surface modification using, 416
  smear layer removal using, 416
Tetrodotoxin, 91, 552-553
T_H0 cells, 634
T_H1 cells, 634
T_H2 cells, 634
Thermafil, 311, 311*f*-312*f*, 355*f*
Thermal allodynia, 551
Thermal pulp testing, 15-19, 16*f*, 759
Thomas Screw Post Removal Kit, 340, 343*f*
Threaded posts, 825
Three-dimensional imaging, 36-37, 38*f*, 62
Thromboxane A2, 551
Tissue engineering
  advancements in, 447
  cell homing, 455-456, 456*f*
  scaffolds, 455
Tissue reflection, in periradicular surgery, 409-411, 410*f*
Titanium trauma splint, 787, 787*f*
TLR4/CD14, 633
Toll-like receptors, 575, 632-633, 638-639, *e*1
Tomography
  computed. *See* Computed tomography
  definition of, 36-37
Tooth avulsion
  consequences of, 784-785
  description of, 759
Tooth avulsion *(Continued)*
  emergency management of, 785
  pulpal necrosis caused by, 785
  replantation of, 785
  root preparation in, 786-787
  socket preparation in, 787
  treatment of, 785
Tooth discoloration, 821, 821*f*
Tooth extraction
  indications for, 333
  posttreatment disease treated with, 332, 332*f*
  vertical root fractures treated with, 815
Tooth germ progenitor cells, 449
Tooth pain, acute maxillary sinusitis versus, 5-6. *See also* Toothache
Tooth Slooth, 19-20, 20*f*, 794, 795*f*
Tooth stiffness, 819-821
Toothache
  nonodontogenic. *See* Nonodontogenic toothache
  odontogenic
    description of, 687-689
    sources of, 687-690
  organic source of, 697-698
  psychogenic, 696-697
  referred, 697-698
Topical anesthetics, 96
Torsional fatigue, 726
Torsional fracture, of instruments, 266
Touch 'n Heat, 347, 347*f*
Transcription factors, 453
Transforming growth factor-α, 653*t*
Transforming growth factor-ß, 574, 653*t*
Transient receptor potential, 535
Transient receptor potential vanilloid-1 agonists, 114
Transillumination
  crack detection using, 20, 21*f*, 794, 796*f*
  light source for, 796*f*
Transmetal bur, 335-336
Transmetal burs, 148-149, 149*f*
Transparent dentin, 819
Traumatic dental injury
  adjunctive therapy for, 787
  aspects of, 758-759
  avulsed teeth
    consequences of, 784-785
    description of, 759
    emergency management of, 785
    pulpal necrosis caused by, 785
    replantation of, 785
    treatment of, 785
  cone beam computed tomography of, 51-52, 52*f*-53*f*, 760-761, 763*f*
  crown fractures
    complicated, 763-765, 763*f*-764*f*
    description of, 759
    treatment of, 763-765
    uncomplicated, 763
  crown infraction, 763
  crown-root fractures, 759, 773-774
  description of, 758
  endodontic treatment, 787-788
  external cervical resorption secondary to, 667
  follow-up after, 759-760
Traumatic dental injury *(Continued)*
  luxation injuries
    apical neurovascular supply damage caused by, 779-781, 779*f*-782*f*
    biologic consequences of, 777-784
    definition of, 777
    description of, 759
    incidence of, 777
    intrusive, 777
    lateral, 777
    osseous replacement for, 778, 778*f*-779*f*
    pulpal canal obliteration caused by, 779, 779*f*
    pulpal necrosis caused by, 779
    pulpal space infection caused by, 779-781, 780*f*
    radiographs of, 762, 763*f*
    subluxation, 777
    treatment of, 777
    types of, 777
  periodontal ligament contusion injuries secondary to, 665
  permanent restoration, 789
  prevalence of, 51
  pulpal blood flow assessments, 759-760, 760*f*
  radiographic examination of, 760-762, 761*f*-763*f*
  replantation for, 785, 787
  root fractures
    apical, 774-777
    clinical presentation of, 774
    complications of, 774-777
    coronal, 774, 776*f*
    definition of, 774
    description of, 759
    diagnosis of, 774, 775*f*
    facial, 775*f*
    follow-up for, 777
    healing of, 774, 776*f*
    horizontal (transverse), 762, 763*f*, 774, 776*f*
    midroot, 774-777
    prognosis for, 777
    pulpal necrosis caused by, 777
    radiographs of, 774, 774*f*-775*f*
    treatment of, 774
  root preparation, 786-787
  soft tissue management in, 787
  splinting of, 787, 787*f*
  temporary restoration, 789
  types of, 759
  vital pulp therapy for
    full pulpotomy, 767-768
    partial pulpotomy, 767, 768*f*-769*f*
    pulp capping, 766-767
    pulpectomy, 769
    requirements for, 765-766, 765*f*
Traumatic neuroma, 693
Treatment
  endodontic. *See* Endodontic treatment
  multiple-visit, 79-82
  prognosis of, 78-82
  referral considerations, 79
  root canal. *See* Root canal treatment
  single-visit, 79-82
Treatment failure. *See* Posttreatment disease

Treatment planning
biologic considerations used in, 82
description of, 71
health status considerations, 86
interdisciplinary
dental implants, 85-86
endodontic therapy, 85-86
periodontal considerations, 82-83
prosthodontic considerations, 85
restorative considerations, 85
surgical considerations, 83-85
medical conditions and findings that affect
behavioral disorders, 78
cardiovascular disease, 72-73
diabetes mellitus, 73-74
dialysis, 77
end-stage renal disease, 77
malignancy, 75-76, 75*f*
pregnancy, 74-75, 74*b*
prosthetic implants, 77-78
psychiatric disorders, 78
medical consultation letter, 71, 72*f*
quality of life considerations, 86
risk assessments, 72
treatment plan, 78
Trephination, for pulpal necrosis with acute apical abscess, 708-709
Trephine burs, for silver points removal, 360
T-RFLP. *See* Terminal restriction fragment length polymorphism
Triangular flaps, 409*f*
Triazolam, for oral conscious sedation, 113
Tricalcium silicate, 294, 295*t*-296*t*, *e*3
Trichloracetic acid, 674-675
Triclosan, 259
Tricresol formalin, 261
Triethylene glycol dimethacrylate, *e*2
Trigeminal autonomic cephalalgias, 691-692
Trigeminal ganglion, 685
Trigeminal nerve, 686*f*, 746
Trigeminal neuralgia, 692-693, 702-703
Trigeminal nucleus, 686, 689*f*
Trigeminal system
allodynia, 550-551, 550*t*
deafferentation, 553
dentin sensitivity, 547-550
hyperalgesia, 550-551, 550*t*
inflammatory mediators, 551
innervation, 542-546
neuropeptides, 547
pain perception, 546-547
peripheral sensitization, 550, 550*b*, 550*t*
tissue injury, 553
Trigger points, 690-691, 700
Trigger zones, for trigeminal neuralgia, 692-693
Triple-antibiotic paste, 262, 459, 459*f*, 773
Triptans, 692
Trismegistus, Hermes, 281
Tropocollagen, 541
True cysts, 646-647, 649*f*, 654
Tuberculosis, 5-6
Tumor necrosis factor-α, 551
Twisted File, 231
Two-visit pulp capping, *e*10-*e*16, *e*26*f*
Type I collagen, 541
Type II collagen, 541
Type III collagen, 541

U

Ultimate tensile strength, 819
Ultrafil 3D, 310, 310*f*
Ultrasonic instruments
complications of, 346, 346*f*
drawbacks of, 727
heat generated by, 346, 346*f*, 370-371, 418
post removal using, 340, 340*f*
root canal obstructions removed using, 365, 366*f*, 371
root-end cavity preparation using, 417-418, 417*f*
separated instruments removed using, 365, 366*f*, 727
Ultrasonic tips
design of, 418, 418*f*
ledge removal using, 736-737, 737*f*-740*f*
small diameter, 736
Ultrasonic unit and tips, 150, 150*f*, 232*f*
Ultrasonically activated files, 356, 357*f*
Uncomplicated crown fractures, 763
Universal Post Remover, 341
Unmyelinated axons, 544

V

van Leeuwenhoek, Antony, 599-600
Vascular cell adhesion molecules, 638
Vascular endothelial growth factor, 455-456, 644-645, 653*t*
Vascular endothelial growth factor receptor 3, 558
Vasoactive intestinal polypeptide, 542, 558, 578-579, 635
Vasoactive neuropeptides, *e*1
Vasoconstrictors
antihypertensive medications and, 73
in cardiovascular disease patients, 72-73
local anesthesia uses of, 72-73, 94*t*, 579-581
pulpal blood flow affected by, 579-581, 580*f*
pulpal health affected by, 579-581
Vazirani-Akinosi technique, 99
V(D)J gene, 635
Veneer, 830*t*, 837*f*
Vertical root fractures
apical, 809*f*
bone defect associated with, 803*f*, 809*f*
bone loss associated with, 800*f*-801*f*
bone resorption associated with, 809
clinical manifestations of, 800-809, 801*f*-803*f*
complete, 794*f*
cone-beam computed tomography of, 45, 46*f*, 389, 808-809
definition of, 798-799
description of, 26, 27*f*-28*f*, 335*f*
diagnosis of, 799-809
diagonal, 799*f*
early diagnosis of, 802
in endodontically treated teeth, 800, 809-810
etiology of, 809-815, 810*f*-815*f*
exploratory surgery of, 809
illustration of, 147*f*, 333*f*, 425*f*
J-shaped radiolucency associated with, 802, 804*f*, 805-806
long-standing, 804*f*
misdiagnosis of, 802
occlusal factors associated with, 810
Vertical root fractures *(Continued)*
patient history-taking, 799-800
periradicular radiolucency associated with, 806, 808*f*
pockets associated with, 802-803, 805*f*-806*f*
predisposing factors, 809-815, 810*f*-815*f*
prevention of, 815
radiographs of, 800*f*, 802, 802*f*, 804*f*, 805-806, 807*f*-808*f*
root cross section in, 809-810
tooth extraction for, 815
treatment failure caused by, 389
treatment planning for, 815
types of, 800*f*
warm vertical compaction as cause of, 304*f*, 305
Vertical-releasing incisions
periradicular surgery access using, 408-409, 408*f*-410*f*
Virulence, 602
Virulence factors, 602-604
Viruses, 619
Vital bleaching
carbamide peroxide for, 590-591
pulpal reactions to, 590-591
Vital pulp
anatomy of, *e*2
cells of, *e*1
emergencies in, 707-711
obturation in, 284
Vital pulp therapy
bacteria-tight seal in, 765
direct pulp capping. *See* Direct pulp capping
dressing used in, 765-766, 765*f*
indications for, *e*5-*e*6
materials for
calcium hydroxide, 765-766, 765*f*, *e*5
calcium silicate cements, *e*1, *e*6-*e*10
description of, *e*5
hydrophilic resins, *e*5-*e*6
mineral trioxide aggregate. *See* Vital pulp therapy, mineral trioxide aggregate
resin-modified glass ionomer cements, *e*5-*e*6
medicaments for, 766
mineral trioxide aggregate
biocompatibility of, *e*2
characteristics of, *e*2
components of, *e*3
contralateral tooth development, *e*10
description of, *e*1, *e*6
direct pulp capping with, *e*7, *e*8*f*, *e*13*f*
history of, *e*2
inflammatory cascade, *e*2
MTA-Angelus, *e*3
pulpotomy with, *e*7, *e*14*f*, *e*16*f*
radiographs of, *e*27*f*
signaling molecules, *e*2
in noninflamed pulp, 765
objective of, *e*1, *e*10
outcome measures for, 476-477, 476*t*
partial pulpotomy. *See* Partial pulpotomy
permanent restoration, *e*10-*e*11
postoperative follow-up and recall, *e*11
prognostic factors for, 495
pulpotomy. *See* Pulpotomy
purpose of, *e*1
reparative bridge formation, *e*2-*e*3, *e*4*f*

Vital pulp therapy *(Continued)*
requirements for, 765-766, 765*f*
techniques for
caries removal, *e*9
diagnosis, *e*7-*e*9
hemostatic agents, *e*9
treatment considerations, *e*9-*e*10
techniques used in, 476-477
Vital teeth
asymptomatic, pulp anesthesia determinations in, 95, 95*f*
obturation timing in, 284
partially, anesthetic success in, 110
symptomatic, pulp anesthesia determinations in, 95
Vitronectin, 643
Voltage-gated sodium channels
description of, 91
inflammation-related changes in distribution of, 552-553
pain and, 92*t*
subtypes of, 92*t*
subunits of, 92
Voxel sizes, 38-39, 42
Voxels, 38-39, 38*f*-40*f*, 329

W

Warfarin, 396-397
Warm vertical compaction obturation method, 304-305, 306*f*-307*f*
WaveOne, 232
Whitlockite crystals, 574, 574*f*
Working length determination
description of, 237-238, 238*f*
digital radiographs for, 36, 37*f*
Wound
decontamination of, polymorphonuclear neutrophils role in, 392
maturation of, 393
Wound healing
angiogenesis in, 392-393
biologic principles of, 390-391
bisphosphonates effect on, 394-395
cementoblasts in, 394
cementogenesis in, 394
chemotherapy effects on, 75-76
clot formation in, 392
collagen in, 392-393
cone beam computed tomography assessment of, 58-60
cyclooxygenase-2 inhibitors effect on, 395
cytokines in, 653, 653*t*
description of, 652-653
diabetes mellitus effects on, 654-655
digital radiography evaluation of, 36
endothelial cells in, 392-393
epithelium in, 393
factors that affect, 390, 654-655
fibroblasts in, 392
glucocorticoids effect on, 395
granulation tissue formation in, 392
growth factors in, 653, 653*t*
hard-tissue, 393-394
hormones involved in, 394
inflammatory phase of, 391-392
macrophages in, 392
maturation phase of, 393
nonsteroidal anti-inflammatory drugs effect on, 395
after nonsurgical root canal therapy, 653, 653*t*
osteoblasts in, 393-394
osteogenesis in, 393-394
periapical, 652-654, 653*t*
periodontal incisional, 522
polymorphonuclear neutrophils in, 392
Wound healing *(Continued)*
proliferative phase of, 392-393
radiation therapy effects on, 75-76
root fractures, 774, 776*f*
soft-tissue, 391-393
after surgical endodontic therapy, 654
systemic disease effects on, 389
systemic medications that affect, 394-395
Woven bone, 393

X

Xenograft, 435*t*
Xylene, for gutta-percha removal, 347-348, 348*f*

Y

Yeast cells, 605*f*

Z

Zinc oxide eugenol, 587, *e*24*f*
Zinc oxide–eugenol cement
intermediate restorative material, 419
as root-end filling material, 419-420
Super-EBA, 419-420
Zinc oxide–eugenol sealers
formula for, 292*t*
obturation uses of, 291-292, 292*t*
Zinc phosphate cements, 827
Zirconia
burs for, 149
characteristics of, 149
Zirconium posts
composition of, 825
removal of, 341-343
Zorcaine. *See* Articaine, with 1:100,000 epinephrine

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